





E06210



# THE ESSENTIALS OF MODERN SURGERY

EDITED BY

**R. M. HANDFIELD-JONES**

**M.C., M.S., F.R.C.S.**

SURGEON TO OUT-PATIENTS AND E.M.S. COMMANDANT, ST MARY'S HOSPITAL; CONSULTING SURGEON, ST GILES' AND PADDINGTON HOSPITALS (L.C.C.); LECTURER IN OPERATIVE SURGERY AND SURGICAL TUTOR FOR UNIVERSITY DEGREES, ST MARY'S HOSPITAL MEDICAL SCHOOL; EXAMINER IN SURGERY TO THE UNIVERSITIES OF CAMBRIDGE AND LONDON, AND TO THE SOCIETY OF APOTHECARIES; EXAMINER IN PATHOLOGY TO THE EXAMINING BOARD IN ENGLAND; LATE HUNTERIAN PROFESSOR, R.C.S

AND

**A. E. PORRITT**

**M.A., M.Ch., F.R.C.S.**

**Brigadier R.A.M.C.**

SURGEON TO HIS MAJESTY'S HOUSEHOLD; SURGEON TO OUT-PATIENTS AND ASSISTANT DIRECTOR OF THE SURGICAL UNIT, ST MARY'S HOSPITAL; SURGEON, PRINCESS LOUISE KENSINGTON HOSPITAL FOR CHILDREN, AND CONSULTING SURGEON, ACTON, NORTH HERTS AND SOUTH BEDS, TEDDINGTON, HAMPTON WICK AND DISTRICT AND SAVERNAKE HOSPITALS

**Second Edition**

*(Reprint)*

**WITH 624 ILLUSTRATIONS**  
*of which many are in Colour*

EDINBURGH

**E. & S. LIVINGSTONE LTD.**

**16 AND 17 TEVIOT PLACE**

**1945**

*First Edition* - - *October 1938*  
*Reprint* - - - *January 1940*  
*Second Edition* - - *September 1943*  
*Reprinted* - - - *August 1945*

## PREFACE TO THE SECOND EDITION

**T**HE call for a second edition has come while we are still engaged in fighting for our lives and liberties. My friend and co-editor, Lt.-Col. A. E. Porritt, is on Active Service, and with infinite regret has felt compelled to take no part in this work. I trust that we shall resume our co-operation on still further improvements to this book when peace shall have returned to the world. Until then I must take sole responsibility for any mistakes and omissions that may occur.

The war has rendered inevitable many changes in those sections dealing with wounds, burns, hæmorrhage and shock ; these have been largely rewritten and brought up to date, and the chapter which originally covered all this matter has been divided into two. The following sections have been rewritten and rearranged either wholly or in part : Inflammation and Infection, Cleft Palate, Thyroid Surgery and Hernia ; Mr Paton has completely revised the whole subject of Fractures. Mr Mercer has slightly rearranged his contribution on Deformities and has added a section on Low Back Pain.

In dealing with the so-called sulphonamide group of drugs I have adhered to their scientific names, thereby answering the recent appeal of a distinguished chemist that the multitude of proprietary names should give place to those bearing some resemblance to their chemical formula. Sulphanilamide, sulphapyridine and sulphathiazole are mentioned in many places. In every instance the particular drug is carefully chosen and its use specifically selected for our purpose. Sulphadiazine is unquestionably less toxic than the others, but I had not had sufficient personal experience of its therapeutic value to include it.

Over 125 new illustrations have been added, amongst them six oil paintings by Miss Anna Zinkeisen, who has lived and worked with us in St Mary's Hospital the whole of the war in her capacity as a member of the Order of St John of Jerusalem. This distinguished artist has given much of her spare time to helping members of our staff by illustrating their work. I am quite unable adequately to express my gratitude for all she has done for me. A number of illustrations depicting the treatment of fractures have been taken from Watson-Jones' magnificent work. Others illustrating the treatment of wounds and methods of transfusion have come from Hamilton Bailey's "Surgery of Modern Warfare" and Farquharson's "Illustrations of Surgical Treatment." To all these authors I tender my grateful thanks. A certain number of clinical photographs have been taken at St Giles' and Paddington Hospitals, and I am most happy to acknowledge my indebtedness to Dr Alan Daley, Principal Medical Officer

to the London County Council, for his kind permission to reproduce them. Two schematic drawings are taken from E.M.S. Memorandum No. 8 upon the Scottish Treatment of Burns, published by H.M. Stationery Office in Scotland, to whom I am greatly obliged for their permission to reproduce these figures.

In an attempt to bring this book right up to date I have drawn upon certain well-known works, and I desire to acknowledge how greatly they have eased my task. They are Joll's "Surgery of the Thyroid Gland," Dible and Davie's "Pathology," Raven's "Handbook on Shock," and Michael Oldfield's article on Cleft Palate, from which last I have had the kind permission to adapt in colour certain of his drawings. Again I owe more than I can say to Mr V. Z. Cope and Professor W. D. Newcomb, who have been constant in their inspiration and untiring in their assistance. A number of correspondents, some students and many quite unknown to me, have written with corrections of typographical errors and suggestions for the future for which I offer them my profound thanks. My wife has shared all the heavy work of typing new work, arranging fresh illustration material, reading the proofs and compiling the index, and to her I render my gratitude.

Lastly, a radical change has been made in the arrangement of the work and the type used for headings, etc., by which means we believe the subject-matter will be more clearly and attractively presented. In spite of many additions and alterations the size of the book has not been materially increased: this has been an important matter in these days of paper shortage, and for this reason I have refrained from including some additional material upon the commoner operations. This must wait until happier days return. Mr McDonald Walker and Mr Macmillan of Messrs E. & S. Livingstone have given me never-failing help and encouragement, and have met my wishes with unfailing courtesy and co-operation. They have appreciably lightened a heavy burden, and I would assure them how greatly I value their help.

R. M. HANDFIELD-JONES.

*September 1943.*

## Editor's Note for Reprint of Second Edition

During the summer of 1944 it became apparent that either a third edition or a reprint of the second would be urgently needed. For many reasons I had not time to do justice to a new edition, and in deciding to reprint, I was influenced by two important factors. My friend and co-editor, Brigadier PORRITT, is still on active service and unable to take part. We hope to make the third edition even more attractive by certain additional material and, as the end of the war is in sight, I preferred to wait until Brigadier PORRITT could rejoin me. In this reprint, therefore, I have confined myself to the correction of typographical errors and have made a few changes to render some points more clear.

## PREFACE TO THE FIRST EDITION

**T**HE preparation of this book was undertaken only after careful consideration as to the real need for yet another textbook of surgery. During the past fifteen years it appears to us that new works have tended to fall into two groups. The first includes comprehensive products of two or more volumes, while the second comprises "short" textbooks in one volume of comparatively small size. The latter seem frankly to aim at providing the less enthusiastic student with sufficient easily assimilable material with which to satisfy the examiners. The student is not inspired to inquire into the reasons why certain symptoms occur or some methods of treatment are superior to others.

We believe that there is room for a single volume textbook in which surgical teaching is based on the fundamental principles of anatomy, physiology and pathology. It is only by building on sound foundations that men can be taught to think for themselves and to avoid falling into the deplorable habit of subjecting every patient to countless laboratory investigations without making even some small effort to arrive at a diagnosis on clinical grounds.

Our object has been to provide students and practitioners with the essentials of modern surgery in as concise a form as possible, and also to produce a book which will be of practical use to students studying for the higher examinations. We have attempted to show that in many diseases the clinical picture follows logically on the pathological changes, and without entering into detail controversy have indicated those lines along which still unsolved problems should be pursued.

We do not consider that the details of operative treatment should be included in a textbook of clinical surgery. The nature of the treatment has been clearly stated, but no details of technique are included except in a few instances, *e.g.*, tracheotomy, the urgent necessity for performing which may fall to the lot of any medical man with little or no warning. We have acted on the advice of Mr F. A. Williamson-Noble, Ophthalmic Surgeon to St Mary's Hospital and to the National Hospital for Paralysis and Epilepsy, and have omitted a description of the diseases of the eye which usually appears in textbooks of surgery. While it is true that certain lesions in the eye are treated surgically, nevertheless the bulk of ophthalmology is more intimately connected with medicine and neurology. Its scope is such that it cannot usefully be included in a book of this size, more especially when there are so many admirable short books on this subject available.

Certain aspects of surgery have become so highly specialised that we have enlisted the co-operation of thirteen of our colleagues, whose

names appear in the list of contributors. To them we desire to express our heartfelt gratitude and our appreciation of their enthusiastic loyalty in helping us. In the very important section on fractures we have had the advice and co-operation of both Mr W. H. Ogilvie, Surgeon to Guy's Hospital, and of Mr V. H. Ellis, Orthopædic Surgeon to St Mary's Hospital, to whom we tender our sincere thanks.

We are greatly indebted to Dr Courtney Gage and Dr Rohan Williams for many beautiful X-ray negatives, from which Messrs Kodak Ltd. have produced splendid negative prints. To all those concerned in this aspect of the work we tender our admiration and hearty thanks.

We have to acknowledge with great pleasure our indebtedness to Messrs Allen & Hanburys, John Weiss & Son, Meyer & Phelps and Charles King for the loan of blocks. Messrs H. K. Lewis & Co. most kindly allowed us to reproduce thirteen drawings from the second edition of E. H. Kettle's "Pathology of Tumours"; Messrs Butterworth likewise provided us with two blocks used by Mr Colledge in the "Encyclopædia of Medicine." We ask these firms to accept our grateful thanks and appreciation of their ready kindness.

A number of drawings were executed by Miss Margaret Paton, and we are sure that our readers will join with us in congratulating her on the high standard of excellence of her work. Mr W. E. Baker has taken many of the clinical photographs and to him we offer our sincere thanks. Mrs Handfield-Jones and Miss Tudor Jones have been of inestimable service in the production of the typescript, in the taking and arranging of a large number of photographs of pathological specimens and in the correction of the proofs. We have to thank Professor W. D. Newcomb and Mr V. Z. Cope for their constant encouragement and advice, the latter reading a section of the proofs for us.

Lastly, but by no means least, we must place on record our great appreciation of the work of the publishers. Apart from the high technical excellence of the production, we have to thank Mr McDonald Walker for his unfailing kindness and patience during the time this book has been in preparation. He has never failed to meet our wishes and has helped us in every difficulty.

R. M. HANDFIELD-JONES.  
A. E. PORRITT.

*October 1938.*



## CONTRIBUTORS

**E. P. BROCKMAN, M.Ch., F.R.C.S.,**

Orthopædic Surgeon, Westminster Hospital; Surgeon to the Royal National Orthopædic Hospital and Vincent's Orthopædic Hospital, Pinner; Consulting Orthopædic Surgeon, Gerrard's Cross Hospital.

**DISEASES OF BONES AND JOINTS.**

**LIONEL COLLEDGE, M.B., B.Ch., F.R.C.S.,**

Late Acting Surgeon to the Ear, Nose and Throat Department, St Mary's Hospital; Surgeon to the Ear and Throat Department, St George's Hospital; Surgeon to the Throat Hospital, Golden Square; Aural Surgeon to the Royal Masonic Hospital and the West-End Hospital for Nervous Diseases; late Examiner in Laryngology and Otology to the Royal College of Physicians; Semon Lecturer in Laryngology to the University of London.

**DISEASES OF THE EAR.**

**DISEASES OF THE PHARYNX AND ŒSOPHAGUS.**

**DISEASES OF THE LARYNX AND TRACHEA.**

**A. TUDOR EDWARDS, M.A., M.D., M.Ch., F.R.C.S.,**

Surgeon to the Brompton Hospital for Diseases of the Chest; Surgeon in charge of the Department of Thoracic Surgery in the London Hospital; Honorary Surgeon-in-Chief, Thoracic Surgical Clinic, St Mary Abbott's Hospital (L.C.C.); Consulting Surgeon, National Hospital for Consumption, King Edward VII.'s Sanatorium and Queen Alexandra's Hospital; late Hunterian Professor, R.C.S.; Examiner in Surgery, University of Cambridge; late Surgeon and Lecturer, Westminster Hospital.

**DISEASES OF THE CHEST, LUNGS AND PLEURA.**

**GORDON GORDON-TAYLOR, O.B.E., M.A., M.S., F.R.C.S.,  
Hon.F.R.A.C.S.,**

Surgeon Rear-Admiral, R.N.; Senior Surgeon, Lecturer in Surgery and Surgical Anatomy, Middlesex Hospital; Surgeon, St Saviour's Hospital; Consulting Surgeon, West Herts, Kettering, St Columba's, Potter's Bar, Victoria Memorial Hospital, Welwyn and Teddington Hospitals; Member of the Council, R.C.S.; late Hunterian Professor, R.C.S.; Examiner in Surgery to the Universities of London, Durham, Leeds and Belfast, and in Anatomy in the Primary F.R.C.S. Examination.

**THE GENERAL SURGERY OF THE ABDOMEN  
PERITONEUM.**

**R. M. HANDFIELD-JONES, M.C., M.S., F.R.C.S.,**

Surgeon to Out-Patients and E.M.S. Commandant, St Mary's Hospital; Consulting Surgeon, St Giles' and Paddington Hospitals (L.C.C.), Lecturer in Operative Surgery and Surgical Tutor for University Degrees, St Mary's Hospital Medical School; Examiner in Surgery, Universities of Cambridge and London and to the Society of Apothecaries; Examiner in Pathology to the Examining Board in England; late Hunterian Professor, R.C.S.

**VARIOUS CHAPTERS.**

**WALTER HOWARTH, M.A., M.B., B.Ch., F.R.C.S.,**

Surgeon, Ear, Nose and Throat Department, and Lecturer in Diseases of the Ear, Nose and Throat, St Thomas' Hospital; Consulting Aural Surgeon, Haslemere Cottage Hospital; Examiner in Laryngology and Otology, R.C.S. and the University of London (M.S.); Specialist in Otology and Laryngology, War Office Headquarter Medical Board; late Hunterian Professor, R.C.S.

**AFFECTIONS OF THE NOSE AND ACCESSORY SINUSES.**

**R. VAUGHAN HUDSON, F.R.C.S.,**

Surgeon to Out-Patients and Surgical Tutor, Middlesex Hospital; Consulting Surgeon, Connaught Hospital, Walthamstow, and the Royal Infant Orphanage, Wanstead; Surgeon, St Saviour's Hospital.

**HERNIA.**

**G. L. M. McELLIGOTT, M.A., M.R.C.S., L.R.C.P.,**

Wing-Commander, R.A.F.V.R.; Director of Venereal Diseases Department and Lecturer in Venereal Diseases, St Mary's Hospital; late V.D. Officer, City of Stoke-on-Trent, and Assistant Medical Officer, V.D. Department, St Thomas' Hospital.

**VENEREAL DISEASES.**

**A. W. MATTHEW, M.R.C.S., L.R.C.P.,**

Anæsthetist and Lecturer on Anæsthetics, St Mary's Hospital; Senior Anæsthetist to the Throat Hospital, Golden Square, and to the Marylebone Dispensary; Honorary Consulting Anæsthetist, King Edward's Memorial Hospital, Ealing.

**ANÆSTHETICS.**

**WALTER MERCER, M.B., Ch.B.(Ed.), F.R.S.(Ed.), F.R.C.S.(Ed.),**

Assistant Surgeon, The Royal Infirmary, Edinburgh; Surgeon to Chalmers' Hospital and Ministry of Pensions Hospital, Edinburgh; Consultant in Orthopædies, Berwickshire Educational Authority; Consulting Surgeon in Surgical Tuberculosis, South-East Scotland Joint Sanatorium Board Hospital; Lecturer in Clinical Surgery, University of Edinburgh.

**DEFORMITIES.**

**R. Y. PATON, M.A.(St Andrews), B.A., M.B., B.Ch.(Camb.), F.R.C.S.,**  
 Surgeon, the Royal National Orthopædic Hospital and the Princess  
 Louise Kensington Hospital for Children ; Orthopædic Surgeon, Nelson  
 Hospital, Wimbledon ; Consulting Surgeon, Chiswick General Hospital ;  
 Consulting Orthopædic Surgeon (L.C.C.).

**THE DISEASES OF NERVES.**

**INJURIES OF BONES AND JOINTS.**

**A. E. PORRITT, M.A., M.Ch., F.R.C.S.,**

Lieut.-Colonel, R.A.M.C. ; Surgeon to His Majesty's Household ; Surgeon  
 to Out-Patients and Assistant Director of the Surgical Unit, St Mary's  
 Hospital ; Surgeon, Princess Louise Kensington Hospital for Children,  
 and Consulting Surgeon, Acton, North Herts and South Beds and  
 Teddington, Hampton Wick and District and Savernake Hospitals.

**VARIOUS CHAPTERS.**

**JULIAN TAYLOR, O.B.E., M.S., F.R.C.S.,** Fellow of University College,  
 London,

Surgeon to University College Hospital, the National Hospital, Queen's  
 Square, and the Harrow Hospital ; Examiner in Surgery to the  
 Universities of Cambridge, London and Belfast.

**THE DISEASES OF THE APPENDIX.**

**LESLIE H. W. WILLIAMS, M.D., M.S., F.R.C.S., F.C.O.G.,**

Obstetric Surgeon, St Mary's Hospital ; Surgeon, Queen Charlotte's  
 Hospital and the Samaritan Hospital for Women ; Examiner in  
 Obstetrics, University of Cambridge, of the Conjoint Board and Central  
 Midwives Board.

**DISEASES OF THE FEMALE GENITAL ORGANS.**

**A. DICKSON WRIGHT, M.S., F.R.C.S., D.T.M. & H.,**

Surgeon to Out-Patients, St Mary's Hospital, and Assistant Surgeon,  
 Prince of Wales' Hospital, Tottenham ; late Assistant Director, Surgical  
 Unit, St Mary's Hospital ; Professor of Clinical Surgery, College of  
 Medicine, Singapore ; Surgeon, Tan Tock and St Andrew's Hospital,  
 Singapore.

**THE DISEASES OF THE BRAIN AND ITS COVERINGS.**

**DISEASES OF THE SCALP AND SKULL.**

# CONTENTS

CHAPTER	PAGE
<b>I. INFLAMMATION AND REPAIR</b> . . . . .	1
Definition. Pathology. End Results. Acute and Chronic Inflammation, their Clinical Signs and Treatment. Scars.	
<b>II. INFECTION AND IMMUNITY</b> . . . . .	15
Definition. Immunity. Anaphylaxis	
<b>III. NON-SPECIFIC INFECTIONS</b> . . . . .	22
<i>Local, Diffuse and Generalised.</i>	
<b>IV. SPECIFIC INFECTIONS</b> . . . . .	32
Bacilli, Cocci, Spirochaetes, Fungi, Worms, Amoebae and Insects.	
<b>V. VENEREAL DISEASES</b> . . . . .	54
Gonorrhoea in the Male, the Female and in Young Girls. Syphilis. Chancroid. Lymphogranuloma Inguinale.	
<b>VI. TUMOURS AND CYSTS</b> . . . . .	83
General Definition and Etiology. Innocent Connective Tissue Tumours. Malignant Connective Tissue Tumours. Innocent Epithelial Tumours. Malignant Epithelial Tumours. Teratomata Cysts.	
<b>VII. WOUNDS AND BURNS</b> . . . . .	118
Wounds. Types. General Principles of Treatment. Thermal Injuries, Burns, etc. Electrical Injuries.	
<b>VIII. HÆMORRHAGE AND SHOCK</b> . . . . .	142
Pathology. Clinical Picture and Treatment.	
<b>IX. ULCERATION AND GANGRENE</b> . . . . .	163
Simple Non-specific Ulceration. Acute and Chronic Ulcers. Skin-grafting. Gangrene, Threatened, Dry and Moist. Vascular, Infective, Traumatic and Toxic Gangrene.	
<b>X. GENERAL SURGICAL TECHNIQUE</b> . . . . .	180
Antiseptic and Aseptic Surgery. Theatre Arrangements and Technique. Ligature Materials. Pre-operative Preparation and Post-operative Treatment.	
<b>XI. ANÆSTHESIA</b> . . . . .	188
Examination of Patients before Anæsthesia. Pre-operative Medication. General Considerations. Signs of Anæsthesia. Choice of Anæsthetic. Ether, Chloroform. Ethyl Chloride. Nitrous Oxide. Analgesia.	

CHAPTER	PAGE
<b>XII. PHYSIOTHERAPY AND RADIOTHERAPY . . .</b>	<b>213</b>
Prevention of Deformity and Preservation of Function. Massage. Electrical Methods. X-rays in Diagnosis and Treatment. Radium.	
<b>XIII. DISEASES OF THE SKIN . . . . .</b>	<b>231</b>
Boils. Carbuncles. Impetigo. Tuberculous Affections. Growths and Cysts. Sebaceous Glands. The Nails.	
<b>XIV. INFECTIONS OF THE FINGERS AND HAND . . .</b>	<b>245</b>
Anatomy. Prophylaxis. The Distal Segment of the Fingers. Tenosynovitis. Abscess in the Palm. Lymphangitis.	
<b>XV THE SURGERY OF THE BLOOD VESSELS . . . . .</b>	<b>259</b>
Arteries : Injury, Disease, Atheroma, Arteriosclerosis, Aneurysm. Thrombosis and Embolism. Veins : Injury, Phlebitis, Varicose Veins. Growths of the Blood Vessels.	
<b>XVI. THE DISEASES OF THE LYMPHATIC SYSTEM . . .</b>	<b>289</b>
Injury and Growths of Lymph Vessels. Elephantiasis. Infection of the Lymph Glands. Lymphadenoma. Lympho- sarcoma.	
<b>XVII. THE FACE, LIPS AND JAWS . . . . .</b>	<b>300</b>
Face : Development, Congenital Anomalies, Growths. Lips : Ulceration and Growths. Jaws : Injuries, Infections and Growths. Gums : Infections and Growths. Teeth : Infection, Cysts, Odontomes.	
<b>XVIII. THE MOUTH, PALATE, TONGUE AND SALIVARY GLANDS . . . . .</b>	<b>327</b>
Development. Stomatitis. Cleft Palate. The Tongue : Inflammation, Ulcers and Growths. Salivary Glands : Injuries, Inflammation, Calculi and Growths.	
<b>XIX. THE SURGERY OF THE NECK . . . . .</b>	<b>352</b>
Congenital Anomalies. Inflammation. Injury. Cysts. Growths. The Thyroid Gland : its Anomalies of Development and Function. Inflammation. Varieties of Goitre. Growths.	
<b>XX. THE EAR . . . . .</b>	<b>378</b>
Anatomy. Examination. The External Ear : Cerumen, Otitis, Osteoma, Deafness. Diseases of Middle Ear : Acute Otitis Media, Chronic Suppuration, Complications. Mastoid Disease.	
<b>XXI. AFFECTIONS OF THE NOSE AND ACCESSORY SINUSES . . . . .</b>	<b>399</b>
Nasal Obstruction. Inflammation of the Nasal Sinuses. Growths. Adenoids.	

CHAPTER	PAGE
<b>XXII. THE PHARYNX AND ŒSOPHAGUS . . . . .</b>	<b>423</b>
Examination. Foreign Bodies. Pharyngitis. Vincent's Angina. Tonsillitis. Quinsy. Removal of Tonsils. Keratosis. Tuberculosis, Syphilis and Nervous Affections of the Pharynx. Tumours. Cardiospasm. Diverticulum. Rupture. Stenosis. Growths.	
<b>XXIII. THE LARYNX . . . . .</b>	<b>444</b>
Examination. Foreign Bodies. Inflammation. Paralysis of Larynx. Growths.	
<b>XXIV. THE CHEST . . . . .</b>	<b>462</b>
Acute and Chronic Empyema. Infection of the Lungs; Abscess, Gangrene. Bronchiectasis. Growths of Bronchi and Lungs. Pulmonary Tuberculosis.	
<b>XXV. THE BREAST . . . . .</b>	<b>504</b>
The Nipple: Eczema. Paget's Disease. Breast Abscess. Chronic Mastitis. Cysts. Papilloma. Adenoma. Fibroadenoma. Carcinoma and Sarcoma. The Male Breast.	
<b>XXVI. THE GENERAL SURGERY OF THE ABDOMEN AND PERITONEUM . . . . .</b>	<b>528</b>
Penetrating and Non-penetrating Injuries of Hollow and Solid Viscera. Surgical Affections of the Abdominal Wall, Umbilicus, Omentum and Mesentery. Peritonitis: Acute, Localised and Generalised. Local Intraperitoneal Abscess. Chronic Peritonitis. Tuberculous Peritonitis.	
<b>XXVII. HERNIA . . . . .</b>	<b>559</b>
Anatomy of Hernia. Sites of Hernia Formation. Causation. Contents of Sac: Inguinal, Femoral, Umbilical, Diaphragmatic. Other Special Varieties. Treatment.	
<b>XXVIII. THE STOMACH AND DUODENUM . . . . .</b>	<b>584</b>
Injuries. Hypertrophic Stenosis. Acute and Chronic Dilatation. Carcinoma. Peptic Ulceration. Gastric and Duodenal Ulcers and their Complications.	
<b>XXIX. THE SMALL AND LARGE INTESTINE . . . . .</b>	<b>613</b>
Congenital Anomalies. Errors in Function. Enteritis. Colitis. Tuberculous and other Infections. Diverticulitis. Growths.	
<b>XXX. INTESTINAL OBSTRUCTION . . . . .</b>	<b>631</b>
Mechanical Obstruction. Acute Intestinal Obstruction: Strangulated Hernia. Strangulation of Bands. Intussusception. Volvulus. Gall-stone Obstruction. Paralytic Obstruction. Paralytic Ileus. Mesenteric Vascular Occlusion. Chronic Intestinal Obstruction.	

CHAPTER	PAGE
<b>XXXI. THE RECTUM AND ANAL CANAL . . .</b>	<b>645</b>
Imperforate Anus. Injuries. Prolapse. Inflammation and Ulceration. Stricture. Ischiorectal Abscess. Fissure. Fistula-in-Ano. Hæmorrhoids. Growths.	
<b>XXXII. THE DISEASES OF THE APPENDIX . . .</b>	<b>666</b>
Pathology of Appendicitis. Acute Appendicitis. Symptoms. Clinical Varieties and Treatment. Chronic Appendicitis. Pseudomyxoma Peritonei. Carcinoid Tumours.	
<b>XXXIII. THE LIVER AND BILIARY SYSTEM . . .</b>	<b>685</b>
Injuries, Infections and Growths of the Liver. Acute and Chronic Cholecystitis. Gall-stones. Growths.	
<b>XXXIV. THE PANCREAS AND THE SPLEEN . . .</b>	<b>706</b>
Injuries. Acute Pancreatitis. Pancreatic Cysts, Calculi and Growths. Spleen : Injury, Torsion, Infection, Cysts, Growths. Surgical Splenomegalies.	
<b>XXXV. THE KIDNEY AND URETER . . .</b>	<b>714</b>
Examination. Renal Function. Anomalies. Injuries. Hydronephrosis. Pyelitis. Pyonephrosis. Renal Tuberculosis. Calculi. Cysts. Growths. Ureteric Calculi. Calculus. Anuria.	
<b>XXXVI. THE BLADDER, PROSTATE AND VESICLES . . .</b>	<b>754</b>
Ectopia. Diverticula. Incontinence. Retention. Injuries. Cystitis. Fistula. Calculi. Growths. Senile Enlargement of Prostate. Growths.	
<b>XXXVII. THE PENIS AND URETHRA . . .</b>	<b>778</b>
Hypospadias. Epispadias. Phimosis. Infection and Injuries. Stricture. Growths.	
<b>XXXVIII. THE TESTIS AND SPERMATIC CORD . . .</b>	<b>791</b>
Imperfect Descent. Infections : Gonococcal, Tuberculous, Syphilitic. Growths. Hydrocele. Spermatocele. Hæmatocele. Varicocele. Diseases of Scrotum.	
<b>XXXIX. DISEASES OF THE FEMALE GENITAL ORGANS . . .</b>	<b>809</b>
Diseases of the Vulva. Caruncle. Dysmenorrhœa. Sterility. Salpingitis. Extra-uterine Gestation. Ovarian Cysts and Tumours. Neoplasms of the Uterus.	
<b>XL. DISEASES OF THE SCALP AND SKULL . . .</b>	<b>824</b>
Injuries and Diseases of the Scalp. Injuries of the Skull. Fractures of the Vault and Base. Inflammatory Disease.	
<b>XLI. THE BRAIN AND ITS COVERINGS . . .</b>	<b>838</b>
Head Injuries, their Manifestations and Sequelæ. Meningitis. Extradural Abscess. Brain Abscess. Hydrocephalus. Intracranial Tumours.	

CHAPTER	PAGE
<b>XLII. DISEASES OF THE SPINE AND SPINAL CORD</b>	<b>871</b>
Spina Bifida. Sacrococcygeal Tumours. Injuries to the Spinal Cord: Concussion, Compression, Hæmorrhage, Complete Transverse Lesions, Incomplete Lesions. Diseases of the Cord, Myelitis and Meningitis. Tumours of the Cord. Diseases of the Spinal Column. Infections, Arthritis, Growths.	
<b>XLIII. INJURIES AND DISEASES OF THE NERVES</b>	<b>889</b>
Injuries, Affections of Individual Nerves. Spinal Nerves. Brachial Plexus. Nerves of the Lower Extremity. Cranial Nerves. The Sympathetic Nervous System.	
<b>XLIV. INJURIES OF BONES AND JOINTS</b>	<b>915</b>
Fractures. Classification. Repair. Diagnosis. Treatment. Splints. Delayed Union and Non-union. Wounds. Dislocation of Joints.	
<b>XLV. INJURIES OF THE UPPER LIMB</b>	<b>938</b>
Fractures of all Individual Bones of the Upper Limb. Dislocation of all Individual Joints of the Upper Limb.	
<b>XLVI. INJURIES OF THE LOWER LIMB AND OF THE SPINE</b>	<b>973</b>
Fractured Pelvis. Fractures of all Individual Bones of Lower Limb. Dislocation of all Individual Joints of Lower Limb. Fractures and Dislocation of the Spine.	
<b>XLVII. DISEASES OF BONE</b>	<b>1014</b>
Inflammations: Acute and Chronic. Deficiency Diseases of Bone. Achondroplasia. Acromegaly. Paget's Disease. Osteitis Fibrosa. Osteochondritis of Various Bones. Tumours of Bone, Innocent and Malignant. Cysts of Bone.	
<b>XLVIII. DISEASES OF JOINTS</b>	<b>1059</b>
Inflammation: Acute and Chronic. Tuberculous Disease of Individual Joints and of the Spine. Arthritis Deformans. Loose Bodies.	
<b>XLIX. DEFORMITIES</b>	<b>1113</b>
Deformities of the Neck, of the Spine, of the Upper and Lower Extremities. Chronic Low Back Pain.	
<b>L. DISEASES OF THE MUSCLES, TENDON SHEATHS AND BURSAE</b>	<b>1161</b>
Injuries. Inflammation. Tenosynovitis. Diseases of Bursæ.	
<b>INDEX</b>	<b>1177</b>



# THE ESSENTIALS OF MODERN SURGERY

## CHAPTER 1

### INFLAMMATION AND REPAIR

**I**NFLAMMATION is the active reaction of a living tissue to injury, irritation or invasion, providing its structure and vitality are not immediately destroyed. It consists of a series of changes designed to limit and eliminate the attacking agent, remove all dead tissues and finally to restore the area as nearly as possible to normal. Repair therefore is an integral part of the inflammatory process.

The causes of Inflammation may be classified as (1) bacterial, (2) traumatic, *e.g.*, wounds, blows and crushes, (3) thermal, *e.g.*, burns and frost-bite, (4) electrical, *e.g.*, all forms of electrical current, X-rays, radium and ultra-violet light, (5) chemical, *e.g.*, irritant liquids and gases and (6) neoplastic.

The body's response varies with the potency and virulence of the pathological stimulus, but in general it may be said that the more intense the irritant, the more rapidly and actively do the tissues react, and the result is described as Acute Inflammation. Less noxious stimuli applied more gradually produce a slower response, which is known as Chronic Inflammation. In spite of the widely divergent features presented for example by acute staphylococcal osteomyelitis and chronic tuberculous osteitis, these share in reality the same fundamental pathological processes, though in different proportions. These processes and the changes they produce must now be considered in detail.

### THE INFLAMMATORY REACTION

**Vascular Changes.** -These may be studied in the thin web of a frog's foot observed under the microscope after application of an irritant. At first there is a generalised dilatation of arterioles, capillaries and venules, and the stream within them flows more rapidly. This is the stage of *active hyperæmia*, when capillaries not ordinarily in use are taken into service and pulsation is often to be seen in them. Within two hours the circulation becomes slower (*retardation*), the vessels being still widely dilated. Finally, *stagnation* or stasis occurs, and this may or may not end in thrombosis. At this time it will be

noticed that the white corpuscles are drifting out of the central axial stream and collecting in the peripheral stream in close contact with the capillary walls. This *margination of the leucocytes* leads to their adherence to the vessel wall and soon a most striking phenomenon can be observed. Polymorphonuclear leucocytes begin to pass through the vessel walls by a process called *diapedesis*, which is seen chiefly in capillaries. Large numbers of these white cells collect in the perivascular tissues, in which they exhibit amoeboid movement. This migration of leucocytes is followed by that of red cells to a degree which varies greatly in different types of infection, being most marked in certain very severe examples.

These vascular changes are brought about by the action of certain "H" substances (Krogh and Lewis), of which histamine is the best known, and which are set free when tissues are damaged. These substances also exert a chemiotactic effect upon leucocytes, thereby encouraging diapedesis.

**The Inflammatory Exudate.**—It will be realised that changes in the walls of small vessels, which allow solid corpuscles to pass, must inevitably permit the fluid constituents of the blood to filter through. This *increased permeability* affects the lymphatics as well as the blood vessels: it leads to an outpouring of fluid and accounts for that swelling of the tissues which is so characteristic of all inflamed areas. This exudate varies in composition according to the nature and severity of the stimulus. In slight injury it is thin, of low specific gravity and low protein content, being rapidly removed by the lymphatics: when the injury is severe the exudate closely resembles blood plasma and contains a marked proportion of fibrinogen, which is converted into fine fibrils of fibrin by the action of thrombokinase liberated by the damaged tissue cells. The exudate is also rich in the normal antibodies of the blood, such as agglutinins, precipitins and bacteriolysins.

**Abscess Formation.**—An acute abscess is frequently the result of invasion by *Staphylococcus aureus*, whose presence in the tissues rapidly calls forth the defence mechanism described above. Cells in the immediate vicinity of the cocci are killed and a small nidus of bacteria and dead cells is formed. Soon this is surrounded by a zone of fibrin containing polymorphs and the fluid exudate rich in antibodies. The nearest leucocytes gain contact with the cocci and engulf them in their protoplasm, a process known as phagocytosis. In a mild infection such as a "blind boil" the digested bacteria are removed and the signs of inflammation slowly disappear.

Usually, however, the defence fails to achieve so rapid and complete a victory. Polymorphs of the advance guard are themselves destroyed, the cocci continue to multiply and the central area of dead cells is thereby increased in size, being added to by death of tissue cells by the toxins of the invading bacteria. The leucocytic barrier is now reinforced, more polymorphs go into action and eventually the infection is controlled and walled in. Progressive liquefaction of the necrotic debris is brought about chiefly by digestive enzymes set free by disintegrating leucocytes and to a less extent by a specific factor secreted by staphylococci which digests protein. The resulting liquid

is called pus, whilst the lining of the cavity is known as a pyogenic membrane.

Untreated an abscess may continue to spread until the skin or mucous lining of a cavity or tube gives way and the pus is evacuated, after which the abscess cavity heals. The object of surgery in these cases is to provide an outlet for the pus as soon as possible.

**End Results of Inflammation.**—I. RESOLUTION is the restoration of the affected part to its normal condition; it can occur only when the attack is weak and the patient's resistance high. In it the stages of inflammation are simply reversed. Slowly the blood stream quickens, white cells cease to adhere to the vessel walls, the fluid exudate is absorbed by the lymphatics and the cells either return to the blood vessels or disintegrate and are absorbed. Finally, vasodilatation passes off, the vessels regain their tone and all signs of inflammation disappear.

II. ABSCESS FORMATION, which is described above.

III. ULCERATION, in which the changes are similar to those in an abscess, except that they occur upon a surface.

IV. NECROSIS AND GANGRENE.—When an extensive area of tissue is devitalised by severe injury or virulent infection, it dies and remains as an inert mass. An inflammatory reaction occurs at the junction of living and dead tissues, and the latter is separated from the former. When the dead area is small, it is extruded as a *slough*, but if a large area of a functioning unit of the body dies, we speak of *necrosis* of bone and *gangrene* of soft parts.

V. REPAIR is an integral part of an inflammatory process. It is not necessarily the same in every pathological condition; for example, the edges of an incised wound may be either accurately drawn together by sutures, unstably united by blood clot or widely separated, and further they may be sterile or infected. Again a considerable mass of tissue may have died and a gaping cavity remain. Although the processes of healing may vary in such widely different conditions, nevertheless the fundamental pathological principles are essentially the same.

### HEALING AND REPAIR

The foregoing description has dealt chiefly with those reactions of the circulatory system which are designed to surround and control the invader, but we have not yet discussed the mechanism by which the body repairs the damage.

**Tissue Changes.**—If an inflamed area is examined microscopically, its surface will be seen to consist of cellular debris, organisms and polymorphs. Immediately beneath is the zone of dilated vessels and exuded fluid, which is separating and bathing the cells of the part. In this oedematous area a great number of *large mononuclear cells* (histiocytes) can be observed exhibiting amœboid movement and active phagocytosis. Their origin remains a subject for debate and, although in the early stages of inflammation they all appear alike, it is probable that they arise from diverse sources and eventually have different functions to fulfil. Suffice it to say here that they are derived either

from the large lymphocytes of the blood, wandering cells from tissue spaces, fixed connective tissue cells of the part, or the reserves in the reticulo-endothelial system. Whatever their origin, their primary function is phagocytic and within forty-eight hours they will contain polymorphs in varying stages of disintegration, red blood cells and organisms. For this reason Metchnikoff named them "macrophages" in contradistinction to polymorphs, the "microphages."

As the acute phase of the inflammation subsides other cells make their appearance, chief amongst them being *small lymphocytes*. Another is intermediate in size between them and their large brothers, the large mononuclear, namely *plasma cells*. These are oval in shape and have an eccentric nucleus, in which the chromatin is arranged in nodules around the periphery—the "cart-wheel" effect. These two cells are associated more commonly with the chronic forms of inflammation, so that under such conditions we speak of "a small round-celled reaction." Eosinophil and basophil leucocytes may also be seen, but they usually denote certain specific types of infection.

We shall frequently speak of *giant cells* in connection with foreign bodies, chronic inflammation such as tuberculosis and syphilis and certain new growths. A giant cell is formed by fusion of a number of cells of similar type, *e.g.*, large mononuclear, endothelioid or tumour cells. They are therefore characteristically multinucleated, and the arrangement of their nuclei in each disease is a matter of some slight significance.

**Granulation Tissue.**—As the phase of active phagocytosis begins to die down, the large mononuclears continue to divide but now show signs of differentiation. In their early stages they are embryonic in character but their offspring start to reproduce cells of a more mature type. Some of these become oval, their nuclei assume a spindle shape and long protoplasmic processes are developed. These latter form an interlacing trellis-work with those of neighbouring cells and, as they grow older, adult fibrous or collagen fibres are laid down around them. These are *fibroblasts* beginning to lay the foundation of all healing processes, *viz.* that of fibrosis.

While these embryonic cells are preparing to form adult fibrous tissue, endothelial cells are growing out from dilated capillaries and forming strands of cells advancing towards the healing surface. At first they appear as parallel uncanalised columns, but soon a lumen develops, blood corpuscles enter and a new capillary is formed. These unite with their neighbours with the result that a series of vascular arcades is formed with their convexity towards the healing surface. As this formation of granulation tissue (*i.e.*, capillary arcades plus fibroblastic support) continues, the arcades increase in depth and number, until finally the gap is bridged and the healing surfaces are united. Meanwhile in the deeper, *i.e.*, older, layers deposition of collagen fibre increases, the capillary network is reduced to a small number of better developed vessels, and eventually firm fibrous tissue alone remains to mark the place where the body had triumphed over the invading forces. Such an area of fibrous tissue is called a *scar*.

**Variation in the Healing Process.**—I. HEALING BY FIRST INTENTION.—

In a clean incised wound, as in a surgical operation, very few cells are destroyed and infection is absent. Its edges are accurately apposed by sutures and the cleft is sealed by a fibrinous exudate. The typical stages of inflammation are so slight as to escape notice, polymorphs digest the fibrin and a thin zone of granulation tissue paves the way to the formation of fibrous tissue.

II. ORGANISATION OF BLOOD CLOT.—Less perfectly opposed wounds and small cavities will contain a varying amount of blood clot. Here again no organisms are present and no pus produced. Clot takes the place of necrotic debris described above, fibroblasts and polymorphs digest the fibrin, large mononuclears follow to remove all disintegrated matter and the whole is replaced by granulation tissue. Finally the area is permeated by fibrous tissue and healing is complete. This process is known as organisation of a hæmatoma, and is one of great importance in pathology.

III. HEALING AFTER SUPPURATION.—The healing of an abscess has been described in detail.

IV. HEALING BENEATH A SCAB.—Ulceration or suppuration upon a surface differs from the general picture only in one respect. The granulation tissue forms a raw exposed area which demands protection. This is afforded by coagulation of the exudate which forms a thick secure shield, beneath which the tender healing tissues can work without interruption. This scab is shed when delicate surface epithelium has grown in and covered the granulating area.

V. HEALING OF AN ULCER.—In some cases a scab does not form, a raw area remains exposed and pus is discharged freely. Epithelium grows in from the edges and under favourable conditions eventually covers in the whole area. Frequently, however, the formation of granulation tissue is too exuberant and needs controlling by surface applications of silver nitrate. There is a limit to the size of a raw area which can be epithelialised in this manner. In such cases healing is brought about by skin grafting.

## TYPES OF INFLAMMATION

Various types of inflammation are described according to the degree of severity and the site of attack. The primary division of inflammation is into acute and chronic. In acute inflammation the changes described above take place rapidly and in maximal degree in response to a sudden noxious irritant. Chronic inflammation may arise as a later stage of the acute or *de novo* in response to a less noxious stimulus applied over a longer period. Both acute and chronic types have their own particular histological response—polymorphonuclear in acute and lymphocytic in chronic.

A *catarrhal* inflammation is one affecting mucous membranes and is essentially mild in degree. It is characterised by an outpouring of mucus from the affected cells. Of such a type is the well-named "streaming cold."

If the process should increase in intensity cellular destruction takes place and pus is formed. This is acute *suppurative* inflammation. If

the virulence of the attack is excessive, cellular death may occur and *gangrenous* inflammation results.

Should inflammation occur in a serous cavity, *e.g.* pleura or peritoneum, the milder stages are called *serous* inflammation, an outpouring of intracavitary serum being the typical feature. This in later or more severe cases tends to clot and the term *fibrinous* is applied.

In some very acute inflammations the cellular destruction is so marked as to cause actual bleeding into the tissues concerned—*hæmorrhagic* inflammations. Such a very acute process occurring in a mucous surface tends to produce the so-called “false-membrane” (*e.g.* of diphtheria) from a mass of necrotic cells on the surface welded together by fibrin, thus the terms *membranous*, *croupous* or *plastic* inflammation.

In discussing inflammation in any particular organ the description *interstitial* or *parenchymatous* is used according to whether the supporting or essential cellular tissue of that organ is chiefly involved.

## ACUTE INFLAMMATION

### CLINICAL PICTURE

**Local Condition.**—Since Celsus about A.D. 50 described the four cardinal signs of inflammation as calor, rubor, tumor and dolor (heat, redness, swelling and pain) only one other has been added, namely, loss of function.

**Heat** is due to hyperæmia. The increased temperature can be appreciated by the hand and measured by a surface thermometer.

**Redness** is also due to hyperæmia. In the earliest stages of inflammation the colour is bright red, fading and returning with equal rapidity on pressure. During the stage of retardation the colour is more dusky, sometimes even bluish and pressure evokes a slower response. Later still when stasis has occurred the purple colour is more obvious and does not fade on pressure. Further, the dissolution of red blood corpuscles frees hæmoglobin, which adds to the colour and indeed may lead to a lasting brownish-red discoloration.

**Swelling** is due partly to vascular congestion and partly to the exudate. Its extent varies considerably according to the tissue affected. In lax, distensible structures such as eyelid and scrotum it is very marked, in bone it is absent.

**Pain** is the result of pressure of the exudate upon sensory nerve endings. It is therefore due to raised tension, a cause of pain we shall meet with in many surgical diseases. If the affected structures are rigid and inelastic, tension rises rapidly and steeply, and pain is severe; where the swelling is great, pain is likely to be slight. Any factor which increases this tension will aggravate the pain, for example, by allowing an infected hand to hang down. The pain of acute inflammation is described as throbbing, since the vasodilatation allows pulsation in the smaller vessels, and each beat raises the tension and so increases the pain.

Pain is not always limited to the inflamed area, but may be referred by a sensory nerve either to its peripheral distribution or by its central connections.

**Tenderness** is a special type of pain produced by pressure and is one of the most important clinical signs in the diagnosis of acute inflammation.

**Loss of Function** is often due to a reflex immobility of muscles designed to prevent pain, but mechanical reasons, such as swelling of a joint, may account for it. Another important factor is the local toxic damage to the cells of the part caused by the invading organisms.

**Constitutional Involvement.**—Inflammation always causes some degree of general constitutional response. This is due to the absorption of toxins from the site of local reaction into the blood stream. In non-bacterial cases this response is slight and transient; in bacterial cases it is always more pronounced though varying considerably in intensity. In very virulent inflammations the toxæmia may be sufficiently potent to cause death.

This general reaction is described clinically as the febrile state. Fever or pyrexia implies a raised body temperature. This is accompanied by an increased pulse rate and very often respiration is also more rapid. The pulse is more full and bounding than in the normal person. Headaches are frequent and a general feeling of malaise makes the patient restless and irritable.

Anorexia (distaste for food) is usually present, and thirst is excessive. The skin is hot and dry, the face flushed. The mouth is dry and the tongue covered by a white fur. In the more prolonged acute fevers the gums and lips become coated with masses of dried mucus called *sordes*. This frequently leads to the breath being very foul. Constipation is the rule and the motions when passed are very offensive. The urine is scanty and highly coloured with a high specific gravity and contains excessive quantities of uric acid, urates and quite commonly a trace of albumen.

In the later stages of acute fever the toxæmia leads to emaciation, anæmia (the complexion now becoming pasty and sallow) and general muscular weakness and exhaustion. Vomiting may be a marked feature. In the most acute cases semi-consciousness passes into true delirium, collapse occurs and the patient dies.

Fever may be continuous at a certain level, it may fluctuate but never return to normal (remittent), or occur at definite periodic intervals (intermittent). Some particular diseases have characteristically a sudden excessive pyrexial response, the temperature returning either to normal or to lower levels just as rapidly as it rose originally. Such an exacerbation accompanied by a shivering attack is termed a rigor.

The heat-regulating mechanism of children is far from stable, and high temperatures in young patients have not the same significance as in later life, unless continued over a considerable period of time. Fever is the outward clinical manifestation of the body's general response to noxious attack (usually bacterial) and hence, again provided it falls within a reasonable time, a high temperature is indicative of a good resistance. When suppuration is present a small amount of pus, if



under tension, will produce a very marked febrile reaction quite out of proportion to the local condition.

### TREATMENT

In order to avoid unnecessary repetition we shall describe general principles rather than details of treatment, which latter will be dealt with in relation to different infections and regions of the body. No reference will be made here to the treatment of open wounds which follows in Chap. VII.

**Local Treatment.**—(a) PREVENTION.—Many causes of inflammation are avoidable but this covers so vast a field of preventive medicine that its range can only be indicated here. In industry prevention of injury and prophylaxis of infection are of immense importance, as is also that of endemic and epidemic infective diseases.

(b) REMOVAL OF THE CAUSE.—This is a relatively simple matter when the cause is evident *e.g.*, a foreign body, a buried suture or ligature, a carious tooth, etc. More active measures may be needed, such as excision of an infected focus (an acutely inflamed appendix), of a sinus or fistulous track or curettage of unhealthy tissue.

(c) GIVE REST TO THE INFLAMED PART.—The lesson taught by Hilton in “Rest and Pain,” so long forgotten, has again become the guiding principle of treatment. Wherever possible complete immobilisation should be assured by plaster of Paris bandages, firm fixation in splints, bandaging, etc. The function of inflamed internal organs, glands, gastro-intestinal tract, etc., must also be reduced to a minimum. Enforced rest can easily be overdone and the resumption of activity is to be encouraged as soon as the acute stage of inflammation has definitely subsided, otherwise loss of function may lead to prolonged incapacity.

(d) RELIEVE TENSION.—Tension is due to hyperæmia and accumulation of inflammatory exudate. If under pressure the fluid causes great pain and it is then a source of danger, since it may embarrass the circulation and lead to an increased area of necrosis. It is relieved by several means.

1. *Elevation of a limb* assists the venous and lymphatic drainage and diminishes pain. Its importance is not sufficiently realised. The limb should be supported in a special bed-rest fitted with an adjustment to vary the angle of elevation. Different types are needed for the upper and lower limbs.

2. *Surgical Measures.*—(a) Incision and drainage of an abscess should be provided as soon as its presence is diagnosed; (b) multiple small incisions are useful in certain types of inflammation *e.g.*, cellulitis, in which tension is high, but little actual pus has collected; (c) local relief may be obtained by the use of leeches and by scarification followed by dry and wet cupping (methods now obsolete in this country but worthy of a limited return to favour).

3. *Surface Applications.*—(a) Heat either moist or dry is valuable in reducing tension and relieving pain. Its many methods of application are described in Chap. XII; (b) cold in the form of ice bags,



evaporating lotions and a flow of cold water is of limited use, but care must be exercised in its employment, for it may do more harm than good by depressing the local circulation.

(e) **INCREASE THE BLOOD SUPPLY.**—This is done by inducing hyperæmia which is brought about in two ways. Active hyperæmia encourages an increase in arterial flow by producing vasodilatation by various forms of radio-therapy, such as radiant heat, infra-red rays and short wave diathermy. Passive hyperæmia is venous in type and is best obtained by the use of the pneumatic limb compressor of a sphygmomanometer, by which the venous and lymph flow can be impeded without any restraint upon the arterial supply. Bier's or Klapp's suction glasses of varying sizes are used to produce local hyperæmia (Fig. 1).

(f) **PREVENT MIXED INFECTIONS** gaining access to an open wound (see p. 128).

**General Treatment.**—(a) **REST** in bed is essential in all but the most trivial inflammation. Neglect of this principle accounts for a vast loss of man-hours work in every class of the community. Refusal to give up work may be admirable in theory but is uneconomic in practice.

(b) **DILUTION AND ELIMINATION OF TOXINS.**—The organs of excretion must be assisted to get rid of circulating toxins as rapidly as possible. A daily action of the bowel is essential but too active purging should be avoided. Kidney action is increased both by the quantity of fluid introduced into the body and by the use of diuretic drugs. Elimination by the skin is aided by heat and diaphoretics.

(c) **PRESERVATION OF THE WATER BALANCE.**—In many inflammatory diseases there is a great loss of fluid by profuse discharges and it is remarkable how rapidly signs of dehydration appear. This loss must be made good and in addition to plentiful drinks, saline should be given rectally, subcutaneously and intravenously. These methods and their indications are described in Chap. VIII.

(d) **DIET** should be reduced to its most nutritious as well as easily assimilable form, *e.g.* milk, meat-juices and extracts, chicken and calf's-foot jelly, etc.

(e) **RELIEF OF PAIN.**—Pain, severe and prolonged is most debilitating and wears down the patient's powers of resistance. Its relief is obtained partly by local measures and by analgesics or hypnotics (aspirin, bromides, nembutal, morphia, etc.). With this is associated the control of sleeplessness, a matter of the greatest importance.

(f) **SUPPORT THE HEART.**—In certain conditions and in strong, healthy patients, the blood pressure may be sufficiently high to demand relief; this may be done either by drugs or by a carefully controlled venesection. Stimulants will be called for if strength is failing, brandy, strychnine and digitalis being the most valuable. A moderate degree of rise in temperature is a favourable sign of the body's efficient resistance and only hyperpyrexia (105° F and over) needs treatment. Tepid sponging of the whole body or even ice packs may be required, while aspirin, phenacetin and quinine will temporarily lower the temperature.



FIG. 1

A Bier's cupping glass with rubber suction bulb.  
(Allen & Hanbury's.)

(g) **SPECIFIC REMEDIES.**—Certain diseases are relieved by specific antisera, others, for example hæmolytic streptococci by drugs of the sulphanilamide group. In very active general reactions these methods can be reinforced by the transfusion of blood or plasma.

(h) **RESTORATION OF FUNCTION.**—Treatment does not cease with the control and resolution of the inflammation, but suitable methods must be applied to restore the affected area to full use and power (Chap. XII).

## CHRONIC INFLAMMATION

The pathological processes underlying all inflammations are essentially the same, but in chronic inflammation some changes are more and others less prominent than in the acute condition. There is a wide range of virulence and there are therefore many intermediate stages between the extremes of acute and chronic reactions. In chronic inflammation the stage of active hyperæmia is slight but prolonged, diapedesis is on a small scale, while the fluid exudate contains little protein and fibrin. The greatest difference concerns the tissue response and the cells with which the area is infiltrated, chronic inflammation being characterised by "a small round-celled" reaction. These cells are probably lymphocytes and occur in large numbers, while others are derived from the endothelium of blood vessels, lymph vessels and spaces. Plasma cells are commonly seen and eosinophils are present in certain diseases. The end results vary considerably according to the nature of the inflammation and the part of the body affected. Tuberculosis and syphilis have their own individual reactions, but in general it may be said that in most instances chronic inflammation ends in the formation of fibrous tissue.

**Clinical Picture.**—A mild degree of pyrexia often noticeable only at certain times of the day (especially evening) is the usual feature, but an increase in temperature is frequently absent. Patients are steadily absorbing small doses of toxins and this leads to a state of chronic poisoning of the whole system known as *toxæmia*. The patient becomes pale, has no appetite and loses weight rapidly and, if relief is not forthcoming, literally seems to fade away.

Local signs are different to those of acute lesions. Heat and redness are absent, but swelling may be marked. Pain is less acute and is not throbbing in character, while tenderness is correspondingly diminished. Loss of function is often a prominent feature, as for example in a tuberculous joint.

One complication of prolonged inflammation, especially if associated with long-continued suppuration, is *amyloid disease*. This change affects chiefly the smaller vessels of the kidney, spleen, liver and small intestine, in which it leads to extensive pathological degenerations and eventually the death of the patient. Reference should be made to textbooks of Pathology and Medicine for a full picture of the pathological changes and the clinical picture.

**Treatment.**—LOCAL (a) *Remove the Cause*, if possible. Such poison factories as the teeth, tonsils, gall-bladder and appendix can be removed,

antra and chronic abscesses can be drained and foreign bodies or diseased particles of bone extracted.

(b) *Rest the Affected Part.*—Whether its function be mechanical or physiological, continued activity will often cause prolongation of a chronic inflammatory process. The part concerned must be temporarily given a rest from its normal function to allow its cells to concentrate on the process of repair. This applies equally to part of a limb or to an organ. But, in that the process is a protracted one, common sense and judgment must be used in preventing disuse atrophy. At a certain stage of proceedings there is no doubt that reasonable and controlled activity will assist recuperation.

(c) *Physical Methods.*—In long-standing inflammation heat is of considerable value and is usually employed as dry heat (hot air or radiant heat baths) or can be engendered by massage. Pressure also finds a greater field of usefulness in chronic than in acute inflammation, and the same applies to passive hyperæmia. One line of therapy peculiar to chronic inflammation is the use of counter-irritants, although this again is essentially a means of producing localised hyperæmia (active). The substances in commonest use are tincture of iodine, mustard plasters, cantharides ("Spanish fly") and Ung. Hydrarg. Co. ("Scott's dressing"). The cautery—actual or diathermy—can also be employed in this connection.

GENERAL.—This may be further subdivided into non-specific and specific. *Non-specific* general treatment follows the same lines as enunciated for acute inflammation above. As chronic inflammation will usually automatically imply a prolonged absorption of small doses of toxin into the general blood stream, the stimulation of the various excretory functions forms a very important part of treatment.

*Specific Treatment* involves the use of sera and drugs proved to be of therapeutic value in the particular disease concerned.

## SCARS

A scar is a mass of devascularised fibrous tissue which in a superficial wound is covered by a single layer of epithelium. This epithelium has no papillæ, no hair follicles, no sebaceous glands, no lymphatics, and usually no nerves.

A superficial scar involving skin only may become almost completely obliterated. Scars involving deeper tissues and internal organs never disappear. In their early stages, before all the capillary loops have been squeezed out of existence by fibrosis, they may be obviously red in comparison to surrounding tissue, but when fully established they are dead white and have a glazed appearance.

Various pathological conditions can occur in scars.

1. **Weak Scars** usually occur in places subjected to considerable mechanical strain, e.g., the neck, amputation stumps, distended abdomen, etc. Very often there is an added element of mild sepsis, and this may lead to actual *ulceration* of the scar. Chronic irritation of the cicatrix, as, for instance, in the continued rubbing of an artificial limb, is again an important factor in weakening of scars.

Such scars are typically stretched out and broadened, thin and easily irritated. Treatment is symptomatic, consisting in local support and protection, local stimulating applications or, in the absence of actual infection, excision and resection to obtain healing by first intention.

**2. Contracted Scars.**—Excessive contraction of scars is not uncommon and may occur equally well in superficial structures and in deeper tissues. A new scar contracts to about two-thirds of its original length. In the skin over-contraction is particularly prevalent after extensive burns (Fig. 2), and is most marked, even sometimes producing severe deformity, if occurring on the face, neck or flexures



FIG. 2

Severe scarring of the region of the elbow following a burn showing limitation of full extension as a result.

of the limbs. Excessive fibrosis in deeper structures may lead to muscular contractures as in torticollis (*q.v.*), or to partial obliteration of some part of the intestinal canal or urinary ducts, *e.g.*, urethral stricture (*q.v.*).

Treatment of the cutaneous type of excessive contracture consists in either gradual stretching, division or excision. It should be remembered that important structures are liable to become attached to the under surface of such scars, and due care is required either in stretching or cutting them. Any bare area caused by division is treated by suitable skin grafts (*q.v.*).

**3. Adherent Scars**, one form of which is the *Depressed Scar*, occur when two or more tissue planes, one of which at least is mobile, are bound together by fibrosis. Such scars occur either superficially (skin adherent to tibia), in muscular planes (quadriceps adherent to femur) or in the viscera (post-operative adhesion of intestine to

peritoneum). Depressed scars are usually the result of healing in an old-standing sinus.

Adherent scars may lead to considerable deformity ; in the limbs they may be responsible for false ankylosis of a joint, and are usually painful. Treatment consists in gradual stretching by massage and movements or by operative freeing of adhesions.

**4. Painful Scars.**—These are due to involvement of a nerve trunk in the contracting fibrous tissue of the scar. This leads to persistent and severe pains which may be felt not only at the site of pressure but radiating to the distribution of the nerve concerned. It is typically encountered in the severed nerve trunks of an amputation scar, where the terminal "neuroma" becomes involved in the fibrosis. Injection of alcohol locally may serve to paralyse the nerve, but if this is not practicable it must either be freed by operation from the scar tissue or excised.

**5. Pigmented Scars.**—These are usually due to extraneous particles introduced at the time of injury, e.g., gunpowder and coal dust. But the scar of a healed chronic ulcer is often brownish-red in colour from staining with hæmatoidin. Syphilitic scars are dead white in colour. Tattooing is deliberate pigmentation. Apart from æsthetic reasons treatment is not required. On the face and hands such scars may demand excision.

**6. Hypertrophied Scars.**—A true hyperplasia of scar tissue in its early stages is common, particularly in the presence of mild infection. Such is easily treated by the application of a caustic (silver nitrate stick). A more serious condition is known as *Keloid* (Fig. 3), when the scar becomes raised above the surface, sends ex-



FIG. 3

An extensive keloid forming in the scar of an operation for left inguinal hernia.



FIG. 4

Marjolin's ulcer. A squamous-celled carcinoma which has arisen on an old chronic varicose ulcer.

especially via stitch holes, and is dusky red in colour. This change occurs particularly in tuberculous patients and in negroes, most commonly in the neck (especially vertical scars) and after burns.

The most widely accepted theory of etiology is that it is a condition of fibromatosis of the walls of the occluded capillaries in the original scar tissue. A keloid is often intensely itchy and always disfiguring. Treatment is most disappointing. Excision with resuture nearly always leads to recurrence, but skin grafting the excised area may avoid this. Both X-rays and radium have in isolated cases produced cures. A certain proportion disappear spontaneously.

7. **Neoplastic Scars.**—Malignant changes are rare, scars particularly affected being those subjected to chronic irritation, such as X-ray burns. The growth is a true carcinoma, very slow-growing and not painful until it spreads to normal tissue. Ulceration is common, the condition then being known as *Marjolin's disease* (Fig. 4). Treatment consists in early and wide excision.

A. E. PORRITT.

R. M. HANDFIELD-JONES.

## CHAPTER II

### INFECTION AND IMMUNITY

#### INFECTION

**I**NFECTION implies the penetration of living bacteria into the tissues of the human body. This invasion may occur either through a break, often microscopical, in the continuity of the body's surface, whether it be of skin or mucous membrane, or by direct inoculation into the deeper tissues as happens in the case of bites from animals and insects or of wounds by sharp foreign bodies. Surface infection takes place not only by contact of bacteria with the skin, but also by inhaling them into the respiratory tract or ingesting them with food or fluids into the alimentary canal. Moreover it is known that bacteria can enter the body through an intact skin or mucous membrane.

**Bacteria** are low forms of plant life. They are unicellular and multiply by fission (*schizomycetes*), contain no chlorophyll, and require for their existence moisture, a suitable temperature, certain salts, combined nitrogen and in some cases oxygen. To avoid the ill-effects of lack of moisture, many of them are capable of forming spores, which can resist high degrees of desiccation over long periods. The optimum temperature for most bacteria is somewhere in the neighbourhood of body temperature. Their nitrogen supply is derived either from living tissues direct (*parasites*) or from dead organic material (*saprophytes*). Both types are found in human pathology and under certain conditions one can be transformed into the other, facultative parasites. Those bacteria that require oxygen are known as aerobic, those that do not anaerobic. Again some transmutation of forms is possible, thus the existence of facultative aerobes. The morphology and characteristics of the many and varied forms of micro-organisms is dealt with to some extent in a later chapter, but for a full description textbooks of bacteriology should be consulted.

**The Distribution** of bacteria is widespread. They exist in large numbers in the air, especially that of cities, in water and soil. The bacterial content of air varies with many factors ; around the mountain summits and above mid-ocean it is negligible, in enclosed spaces devoid of air currents particles of dust and bacteria sink to the ground and the air, previously heavy-laden, becomes almost sterile. Many natural water supplies teem with dangerous organisms, so that domestic water has to be purified before delivery. The soil especially in heavily manured areas contains at least two organisms very lethal to human beings, namely those of tetanus and gas gangrene. Human skin, mouth,

intestinal tract, external ear, anterior nares, vulva and anterior urethra are all heavily contaminated, but very few of these bacteria are capable of producing disease, whilst others require specially favourable conditions in which to do so.

**Pathogenicity and Virulence.**—Bacteria which can produce disease are defined as *pathogenic*, and only a small number of this vast family of organisms can be classed as such. Furthermore not only must bacteria be pathogenic to man, they must also obtain access to tissues in which they can thrive and produce their noxious results. For example, if tetanus bacilli are swallowed no harm will ensue but if they are placed in a cut in the skin a typical lesion develops; conversely, little ill-effects follow if cholera bacilli are introduced beneath the skin, whereas their presence in the intestine has grievous effects.

The severity of an illness depends upon the strength of the attacking organism and the defensive powers of the body. Individual bacteria, *e.g.* streptococci may produce a mild local lesion or endanger the patient's life. This variability is referred to as the *virulence* of organisms and we speak of them as of "high" or "low" virulence. Moreover some bacteria normally non-pathogenic or of low virulence are capable of becoming highly virulent if their environment is suddenly and profoundly altered. Conditions lowering the general health and powers of resistance of an individual, such as previous illness, exhaustion, exposure, starvation, poor hygienic surroundings, hæmorrhage or concomitant infection all adversely affect the body's response.

Under certain conditions bacteria may be shut off in a part of the body, still alive but temporarily inactive. In due time they will die out, but occasionally they survive and a return to favourable conditions permits a recrudescence of their activity. This is known as a *latent infection*.

Certain infections are disseminated by people known as *carriers* who, although harbouring pathogenic organisms, are not affected by them owing either to an acquired immunity from a previous attack (*e.g.* typhoid) or to natural powers of resistance (*e.g.* diphtheria).

**Products of Bacterial Activity.**—These are many and varied, including gases, pigments, enzymes, alcohol, acids and alkalis. Their importance is bacteriological rather than surgical in that they are used chiefly in the typing of various strains of bacteria. Some organisms produce a proteolytic enzyme (*leucocidin*) which digests leucocytes, whilst others destroy red blood cells (*hæmolysins*) and set free their hæmoglobin, and reference will continually be made to the gravity of hæmolytic streptococcal invasions. But the most important chemical result of all bacterial metabolism is the production of toxins.

**Toxins** are non-crystalline, non-dialysable substances which have never been isolated in pure form. They are always associated with proteins and proteoses and are probably adsorbed upon the surface of protein particles. Every toxin is specific, which is to say it produces one disease and that one only. They are divided into two groups.

1. **EXOTOXINS** are secreted by bacteria both in the body and in culture media, from which they can be separated by infiltration. Their introduction into the body produces the clinical picture of the disease.



The best examples of exotoxins are those of *C. diphtheriæ*, *Cl. tetani* and *Cl. botulinum*, but an increasing number of bacteria are coming to be recognised as exotoxin producers. They are extremely potent and their minimum lethal dose is far smaller than that of an alkaloid poison, but unlike the latter they do not act immediately after injection, but only after a certain latent period. Finally one of their most important properties is that of stimulating the body to produce an antitoxin, a substance capable of neutralising their poisonous effects. Just as its parent toxin is specific, so antitoxin will counteract only its own disease. Certain animal venoms and vegetable poisons have the same property of developing an antivenom.

2. **ENDOTOXINS** are more intimately associated with the bacterial body and little or no diffusible toxin is found in culture media. Our knowledge of endotoxins is scanty.

**The Infective Reaction.**—The sequence of events in infection now becomes apparent. At the site of bacterial invasion a primary focus develops and gives rise to a typical inflammatory reaction, spread to the regional lymph glands follows and, if the defence mechanism breaks down, bacteria enter the blood stream and infection is disseminated all over the body. Spread to remote parts occurs in another way, for example the exotoxins of tetanus spread along peripheral nerves to reach the central nervous system, while others circulating in the blood stream produce their effects at a distance, for example peripheral nerve palsies in diphtheria. Bacteria are rarely found in the blood in any numbers or for any length of time, although mild bacteriæmia is probably more common than is usually thought. Their presence together with their toxins in the blood stream may constitute a very dangerous condition known as **septicæmia** (p. 28). Should bacteria or minute particles of infected cellular debris lodge in a radicle of the peripheral circulation, a local inflammatory lesion will result—a condition named **pyæmia**.

## IMMUNITY

We have seen that our whole environment is laden with potentially pathogenic organisms and yet a relatively small proportion of the population succumb to diseases produced by bacteria. Obviously there must exist a lack of susceptibility to or protection against these ubiquitous micro-organisms and their toxic products. This is called immunity, which may be either natural or acquired.

**Natural Immunity** is a part of our natural inheritance enabling us to repel bacterial invasion. It varies not only with the species concerned but also with the individual and to a certain extent with the tissue attacked. Thus certain diseases are peculiar to man, such as gonorrhœa, syphilis, scarlet fever and typhoid, which are unknown among the lower animals. Similarly a certain number of individuals will escape infection in a severe epidemic (*e.g.* of scarlet fever in a school) although constantly exposed to infection. In addition to certain inherited powers of resistance there are other factors which influence an individual's susceptibility.

Certain conditions are said to predispose to infection such as cold, wet and exhaustion either separately or even more effectively when combined. Age is an important factor, either extreme particularly infancy being less able to ward off infection. Starvation, insanitary conditions, lack of sunlight play their part. Severe hæmorrhage, certain chronic poisons and long-standing debilitating diseases all tend to a lowering of resistance to infection.

Locally certain tissues seem ill able to defend themselves, for example fat, but this is probably a question of a poor blood supply. Injury to a part depresses its vitality and favours infection, whilst the presence of foreign bodies such as metal fragments, pieces of dead bone and surgical ligature materials render tissues more liable to attack. Again powerful antiseptic chemicals when applied to open wounds may do more harm than good by destroying living tissue cells.

**Acquired Immunity** is of an entirely different type and is of two distinct varieties, active and passive.

**ACTIVE IMMUNITY** develops as a result of a series of vital processes within the patient's tissues, whereby certain specific substances are formed and retained in the blood stream. These are referred to as "antibodies" which are able to neutralise or destroy the toxin which has produced them, so that a patient cannot contract that particular disease. Active immunity can be acquired in two ways either naturally or by artificial means. The victim of scarlet fever, typhoid, smallpox and many other less serious infectious fevers rarely suffers from a second attack. During his illness and subsequent convalescence he has built up a supply of "antibodies" and these afford almost complete protection in the future.

The artificial production of active immunity is based upon the observation that repeated sub-clinical attacks of infection will result in an immunity similar to that conferred by an active attack. There are several methods available : (a) the inoculation of attenuated strains of live bacteria, *e.g.* vaccination against smallpox ; (b) the injection of prophylactic vaccines, such as Wright's against typhoid and paratyphoid—the well-known T.A.B. vaccine, Haffkine's against plague and others ; (c) the injection of poisonous toxins, as in the production of therapeutic antisera in horses ; and (d) by using specially prepared toxins, called "toxoids" such as toxin-antitoxin mixtures, formol-toxoid or alum precipitated toxoid, which achieve their end without giving severe toxic reactions.

Such acquired immunity takes a little time to develop, usually about a week, but it persists for a long time varying with the disease ; for example in diphtheria it is permanent, with T.A.B. vaccine it lasts for about two years, in cholera about a year while in some diseases it lasts but a few weeks.

**PASSIVE IMMUNITY** is a temporary protection conferred upon man or animals by introducing into the circulation a supply of "antibodies" manufactured by a process of active immunisation in a horse, or by a human patient while convalescent from disease. The familiar anti-tetanic, anti-diphtheritic and anti-gas gangrene sera are examples of the former, while antisera for measles, whooping-cough and scarlet

fever illustrate the latter. Their effect is immediate but transient in duration.

**Mechanism of Immunity.**—We refer to “antibodies,” but we are ignorant of many of the processes concerned in their production. Certain types of antibodies are described :—

1. Antitoxins, whose action is essentially a neutralising one ;
2. Lysins, which break up not only bacteria but cells of the body, *e.g.* red blood cells ;
3. Agglutinins, which cause bacteria to clump, thereby reducing their potency and exposing them to a more intensive attack ;
4. Precipitins with a similar action.

**Clinical Applications.**—A. **DIAGNOSTIC.**—The complement fixation tests for syphilis (Wassermann reaction), gonorrhoea and other diseases is based upon antibody reactions. The agglutination properties of the enteric group of organisms is made use of as a test, while the intradermal injection of vaccine or toxin has given us tests for scarlet fever (Dick), diphtheria (Schick), for tubercle (Mantoux) and many others.

B. **THERAPEUTIC.**—Antitoxic sera are used in the treatment of diphtheria, tetanus, scarlet fever, bacillary dysentery and snake-bites. Anti-bacterial sera are used to combat pneumococci, meningococci and anthrax bacilli.

A vaccine is made by suspending living organisms in saline, estimating their number per cubic centimetre and diluting to a convenient dosage. The bacteria are then killed either by heat, formalin or other antiseptic. Prophylactic vaccines are of the very greatest importance in producing immunity but their value as therapeutic agents in the presence of active disease is almost negligible.

## ANAPHYLAXIS

This curious phenomenon is closely related to, although superficially so completely at variance with, immunity and was discovered accidentally by Richet during researches into certain toxic substances. If a solution containing a foreign protein, for example horse-serum, is injected into an animal, a change occurs whereby the subject is rendered highly sensitive to any further injection of this particular protein. Should a second dose be given fourteen or more days later, the animal rapidly becomes gravely ill and may die. Several factors influence both the occurrence of anaphylactic shock and its severity. The second or activating dose need be quite small, but the larger it is the more severe will the symptoms be. The route by which it is administered is important, ingestion by mouth is harmless, subcutaneous injection gives a moderate reaction, intravenously its effects are very severe, and the intrathecal method gives the gravest of all. The time interval between the two injections is important ; just as immunity takes some days to develop, so does the anaphylactic state, indeed they go hand in hand. The hypersensitivity persists for many months and slowly disappears, as do the antibodies of an immune serum. Finally the nature of the protein does not matter, egg albumen, vegetable proteins,

grass pollens etc. will produce the result. The reaction however is specific. Anaphylaxis therefore is a condition of hypersensitiveness produced either naturally or by the injection of a foreign protein.

### ANAPHYLAXIS IN MAN

In human pathology this rarely occurs except in association with the use of antitoxic sera either in prophylaxis or treatment, and most of the reported cases are concerned with tetanus. If a patient receives an injection of antitetanic serum some months after a previous dose, violent symptoms and even death may follow. Acute anaphylactic shock is characterised by mental distress, dyspnoea, cyanosis and collapse; in man death is fortunately a rare event. It can readily be appreciated how immensely important the subject is in time of war and how essential it is that every injection should be entered upon a patient's case paper. There is one type of individual subject to anaphylaxis without a previous injection, viz., the person who is sensitive to horse-protein and suffers from so-called "horse-asthma."

**Desensitisation.**—At any time, but especially in war, the indications for an injection of an antitoxin in a patient known to be or suspected of being sensitive may override all other considerations. In such cases the patient is first tested by giving 0.1 c.c. of serum intradermally and, if no erythema or urticaria develops within forty minutes, the intravenous injection can be given. If a reaction occurs, the patient must be desensitised and two methods are available.

1. **NON-URGENT CASES.**—A dose of 0.025 c.c. of serum diluted in saline is given subcutaneously and then is doubled every half-hour. When 1 c.c. has been reached, 0.1 c.c. of serum diluted with saline is given intravenously and again this is doubled every half-hour until 25 c.c. of serum has been given; a tedious but safe method.

2. **URGENT CASES, e.g. tetanus.**—5 c.c. of serum are diluted with 50 c.c. of saline and intravenous injections of this mixture are given as follows:—first 1 c.c. followed four minutes later by 3 c.c., then three minutes later by 10 c.c., two minutes later by 25 c.c., after which the full dose of undiluted serum may be given. All injections must be given slowly and stopped immediately any untoward symptoms occur.

**Serum Sickness.**—An anaphylactoid phenomenon follows in a considerable number of non-sensitised people upon a single injection of antitoxic serum. Although not dangerous, this serum sickness may be exceedingly alarming to the patient. After about one week to ten days itching and urticaria of the skin suddenly appear, and in more severe cases the urticaria may assume the "giant" form and there may be swellings of internal mucous membranes with dyspnoea, hæmorrhages, pain and effusion into joints, etc.

**Allergy.**—Some individuals have a natural susceptibility to certain proteins or they may suddenly acquire it. The most familiar examples are foodstuffs, oysters, crabs and lobsters, certain fruits such as strawberries and certain vegetable proteins amongst which the grass pollens, which produce asthma, are prominent. Allergy is more related to

medicine but in different sections of this book reference will be made to this condition.

**Treatment of Anaphylactic States.**—Severe cases are treated by intramuscular injection of 5 to 8 minims of a 1 in 1000 solution of adrenalin. Atrophin gr.  $\frac{1}{50}$  subcutaneously and solutions of calcium chloride intravenously are also used.

A. E. PORRITT.

R. M. HANDFIELD-JONES.

## CHAPTER III

### NON-SPECIFIC INFECTIONS

**T**HE differentiation between specific and non-specific infection is admittedly based upon somewhat slender grounds, but it does serve a useful purpose in clinical teaching.

Non-specific infections are those produced by many organisms which show no particular predilection for any special tissue and produce different results under varying conditions. The organisms chiefly concerned in human pathology are the staphylococci and streptococci.

**Staphylococci** are gram-positive, aerobic but facultative anaerobic and non-motile cocci, which divide in any plane to form clusters which are likened to bunches of grapes or groups of small shot. They give rise to a hæmolysin, a leucocidin and an exotoxin; they liquefy gelatin on culture and ferment mannite. They are classified according to the colour of their colonies on culture media, *S. aureus* (golden), *S. albus* (white) and *S. citreus* (yellow). The aureus strain is the most pathogenic and citreus is purely saprophytic.

**Streptococci** are gram-positive, aerobic but facultative anaerobic and non-motile cocci which grow in chains, the length of which varies but remains constant for each strain. Their classification into groups, subgroups and strains is of purely bacteriological interest, but one primary classification is of surgical importance. This is based upon the reactions of streptococci when grown upon a blood-agar culture. One group produces colonies surrounded by a clear halo due to clearing of the medium by hæmolysis of the red cells, a process called  $\beta$  hæmolysis. These are hæmolytic streptococci. A second group has colonies surrounded by a zone in which the colour is changed to green ( $\alpha$  hæmolysis) and these organisms are known as *S. viridans*. A third class produces no change—a non-hæmolytic streptococcus.

In surgical practice we regard the hæmolytic streptococci as being among our most sinister opponents and many of the most severe and fulminating infections are due to them.

The lesions produced by staphylococci and streptococci are many and varied and we shall classify them as localised, diffuse and generalised.

### LOCALISED INFECTION

#### ACUTE ABSCESS

The pathology of suppuration has been described in detail in Chap. I, p. 2.

The clinical picture of an acute abscess may be considered under the headings local and general.

1. *Locally*, an area showing all the classical signs of inflammation starts to soften. This stage is preceded by an increasingly hard and brawny swelling with marked surrounding oedema. In deep-seated abscesses this condition may be all that can be detected, but as pus forms and approaches the surface the presence of fluid in the swelling is indicated by eliciting *fluctuation*. By this is implied the feeling of a fluid wave when the swelling is compressed by two fingers of one hand and lightly palpated with the index of the other. It should be remembered that this sign of fluctuation is given by some soft solids such as fat and muscle, particularly when the latter is palpated across the direction of its fibres. On the other hand, fluid may be present in a cavity under such tension or the walls may be so thick that it feels hard and it is impossible to elicit fluctuation.

The pain of an abscess is throbbing in character and varies in intensity according to the site. Two factors are of importance, the sensory nerve supply to the part concerned and the degree of tension under which the pus is held. Occasionally an abscess may produce specific signs from pressure on surrounding structures, but the classical picture is throbbing pain in an area of inflammation with definite brawny oedema ultimately showing fluctuation.

2. *Generally* the patient is feverish with a moderately high fluctuating temperature. The absorption of toxins, particularly in the early stages, may be so marked as to affect the heat centres in the brain and produce a rigor, or shivering attack. At the same time a typical change will be found in the blood. A leucocytic count well above 12,000 cells per c.mm. is normally found. The increase chiefly affects the polymorphs.

**Treatment of Acute Abscess.**—This may be most simply summarised as “incision and drainage.” Once the presence of pus has been diagnosed, the correct treatment is to evacuate it. In superficial abscesses the incision should be sufficiently large to ensure adequate drainage, and where necessary gentle exploration of the cavity with a finger prevents secondary loculi being left undrained. It should be made if possible at the most dependent part of the abscess to allow satisfactory gravitational drainage, and placed in such a direction that muscular movements in the neighbourhood do not tend to close it. If dependent drainage is not possible, counter drainage through unaffected tissue must be instituted. Having opened the abscess and explored the cavity, all obvious sloughs should be removed and either a rubber tube, rubber tissue or occasionally a gauze wick placed in the evacuated space to ensure adequate drainage. The whole area is then covered with a sterile dressing to prevent contamination from the outside with secondary organisms. The dressing often requires to be applied with some pressure as it is usual for an abscess cavity to ooze a considerable amount of blood in the early stage after opening, owing to the relief of pressure on the walls when these contain many congested thin-walled vessels.

If an abscess is deep-seated or in a particularly dangerous position, *e.g.*, axilla or neck, where there are many vital or important structures in the neighbourhood, it should be opened by what is known as

*Hilton's method.* This consists of a simple skin incision through which is thrust into the abscess cavity a pair of sinus forceps or closed artery forceps. On opening the blades a sufficient aperture can be made to evacuate the pus and insert a drainage tube.

The remaining essential in the treatment of an abscess is rest and this should be obtained by bandaging, splints, etc. The general treatment of inflammation (p. 9) applies in all details to that of abscess.

### CHRONIC ABSCESS

A chronic abscess is most commonly due to tuberculous infection ("cold abscess") and is considered in a later section (p. 39). A certain number are the result of pyogenic organisms, in which case these have been either of low virulence in the first place or left behind after the treatment, either natural or surgical, of a pre-existing acute abscess. This is very commonly the case in bone infections. Pathologically there is the formation of pus with the minimum of inflammation; clinically fluctuation and perhaps slight pain, without the signs of inflammation.

In a chronic pyogenic abscess it is unusual to find any but a very few attenuated atypical organisms, either in the pus or in the walls of the cavity. In contradistinction to this the wall of a tuberculous abscess cavity is the most usual site in which to discover the offending organism. The treatment of the specific type is considered later. A chronic pyogenic abscess requires incision, removal of slough and granulation tissue and drainage, just as does the acute variety.

### SINUS AND FISTULA

Many non-specific chronic abscesses are, however, clinical entities owing to their persistent connection with the surface, either the skin or some internal cavity. Such connections, which are really tubular ulcers, are either blind at one end, or complete via the abscess cavity from one surface to another. The former is termed a **sinus**, the latter a **fistula**. Such tracks are lined with granulation tissue and later even with actual epithelium and surrounded by a zone of fibrosis. The persistent non-closure of a sinus or fistula may be due to many causes, amongst which may be mentioned (1) inadequate preliminary drainage of an abscess (Fig. 5), (2) foreign bodies, infected cellular debris, especially bone fragments, or catgut ligatures at the bottom of the track, (3) continual secondary infection from neighbouring structures, as is the case with rectal and urinary fistulæ, (4) want of sufficient rest to the part, (5) epithelialisation of the track and excessive fibrosis around it, (6) specific infection, especially by tubercle or syphilis, (7) the development of a neoplasm at the affected site and (8) general constitutional causes.

**The treatment** of a sinus or fistula varies according to its position, but in general it may be said that where possible, *e.g.*, with a foreign body, the cause should be dealt with, and a residual chronic abscess adequately and efficiently drained. The track itself may be scraped and cauterised or preferably excised complete, the resultant wound



being allowed to granulate up from the bottom, either with or without light gauze packing. Radiant heat and infra-red light greatly assist this process. As much rest as possible must be secured and general treatment instituted. In this latter respect vaccines have often proved of considerable benefit.

In cases in which such long-continued suppuration persists, certain general changes take place in the patient, due to the constant absorption of small doses of toxins into the blood stream. The so-called "hectic fever" is the outward and visible sign of this chronic toxæmia. The patient has a constant rise of temperature in the evenings and at this time feels well. As the temperature falls towards morning, often to below normal, sweating is profuse and exhaustion is felt. Gradually anæmia and wasting become evident and the condition, if untreated, may lead to a fatal issue.

It is in such patients that **amyloid disease** occurs. Its appearance is an indication for radical treatment as soon as possible. If the suppurating focus is in a limb, amputation may be the only means of saving life, and elsewhere as radical an excision as possible should be performed.



FIG. 5

A chronic sinus injected with lipiodol to show its extent.

**BOILS (FURUNCLES) AND CARBUNCLES.**—A boil is an acute abscess originating in the infection of a hair follicle or submucous gland usually by the staphylococcus. A carbuncle is a more diffuse infection of the subcutaneous tissues, leading to the formation of a series of intercommunicating abscesses.

Both these conditions are considered in detail in Chap. XIII.

**ULCERATION, NECROSIS AND GANGRENE (INFECTIVE).**—These conditions, in their many and varied manifestations, show a wide range of naked-eye appearances and clinical features. Nevertheless, they all afford examples of the basic pathological principles underlying all inflammatory processes. In some these may be acute in type, while in others more chronic forms are seen. A full description of each will be given in the different sections of Chapter IX.

## DIFFUSE INFECTION

### CELLULITIS

This is a spreading inflammation of the subcutaneous or loose fascial connective tissue, due usually to infection from the streptococcus pyogenes. It is particularly prone to occur in lax tissues, *e.g.*, scalp, orbit, pelvis, scrotum and neck, and is really an expression of the breakdown of natural defensive barriers, due to either excessive virulence of the attacking organism or a lowered general resistance on the part of the patient. The infection is usually derived from some wound which, although more commonly deep and poorly drained, may in some cases be microscopical. Sometimes infection may be carried by the blood and lymph streams from a distant focus.

**Clinical Picture.**—There is often an incubation period of two to three days, after which the onset is heralded by a rigor and general malaise with headache.

Fever is marked, the temperature rising to about 103° to 104° F. and fluctuating slightly at that level. Rigors may be repeated, anorexia is complete, the tongue dirty and furred, bowels constipated and urine scanty and high-coloured. Vomiting is often a feature in children. Delirium and sleeplessness occur in most cases.

Locally, the infective focus may at first show only mild signs of inflammation, but extending from it there develops a rapidly spreading diffuse area of brawny infiltration. If superficial this is often slightly raised, red, intensely tender and covered by shiny skin. The pain is at first aching in character, but may later become throbbing. The edges of the inflamed area are indefinite and fade off into surrounding normal tissue. Slowly the brawny area will soften until it feels boggy to the touch, and if untreated the overlying skin will develop vesicles which discharge a thin, often sanious, sero-pus. Below the skin necrosis, sloughing and gangrene may take place over a very considerable area. The lymphatic glands draining the affected tissues are enlarged and tender.

**Treatment.**—Careful attention to wounds will prevent many cases of cellulitis. In the early stages complete rest to the affected part, where possible, is the first essential, and this should be combined with some means of increasing the local blood supply. Fomentations or radiant heat baths will promote active hyperæmia. Bier's method of producing passive hyperæmia can also be used for periods of ten to twenty minutes two or three times a day. Spread of the inflammation or any tendency to soften calls for more active intervention, and multiple incisions should be made through the skin into the subcutaneous tissue, but if possible stopping at the deep fascia level. For obvious reasons these incisions are made parallel to the main structures (nerves, vessels and tendons) of the limb. They should be lightly packed with sterile gauze, and warm baths in which the whole limb can be immersed give much relief.

General treatment consists in the administration of sulphapyridine and in providing a plentiful nourishing diet and ample fluids, keeping

the bowels freely open, and giving such drugs as may be required to ensure relief of pain and adequate sleep. Alcohol (brandy) undoubtedly does good in these cases. A blood transfusion may on occasions be a life-saving measure.

### SPECIAL VARIETIES OF CELLULITIS

**A. Scalp.**—This usually results from a puncture wound piercing the occipito-frontalis aponeurosis, when the loose areolar tissue at this level allows of easy diffusion. In bad cases the whole scalp is lifted and floats on a bed of pus. Subsequent infection of cranial bones, meninges and brain may occur. The less common variety is a subcutaneous cellulitis. In neither case does sloughing of the skin occur owing to the good blood supply of the scalp. Treatment is by multiple incisions in line with the main vessels.

**B. Orbit.**—This follows either penetrating wounds or spread of infection from within the skull. The eyeballs become protruded, the eyelids oedematous and vision is markedly affected, often permanently. Necrosis of the bony walls of the orbit, damage to the optic nerve, the ocular muscles and the eyeball, and cavernous sinus thrombosis are all more than likely sequelæ. Treatment consists in adequate exposure and drainage in any case of orbital wounds.

**C. Neck.**—This is caused by some focus of infection in the mouth or throat and, starting as it does in deep lymphatic glands, usually is at first confined beneath the deep cervical fascia. The brawny, painful swelling is very characteristic (a well-known form being that limited to the submental region, *Ludwig's angina*), and it may be some days before softening occurs and pus tracks to the surface. In the meantime extensive spread in the neck and to the mediastinum may have occurred, and pressure on important vessels and nerves and venous thrombosis have produced serious symptoms. The greatest danger is oedema of the glottis, with its concomitant acute dyspnoea, which may demand a tracheotomy. Again incisions into the deep cervical fascia to relieve tension, even before suppuration is evident, are the correct treatment. The constitutional reactions, which in this type are most severe, will need adequate symptomatic treatment.

**D. Scrotum.**—This results in most cases from extravasation of urine after rupture of the urethra. The process is very acute, sloughing of the skin extends over a considerable area, and the general reaction is severe.

**E. Pelvis.**—Pelvic cellulitis is due either to wounds which penetrate to the loose areolar tissue of this region and are imperfectly drained, or to infection which spreads either directly or indirectly from the various pelvic organs. Being a deep-seated inflammation local signs are at first absent, but the constitutional reaction is marked. In due course a tender mass will be felt per rectum or per vaginam and ultimately pus may track to the surface (either above Poupart's ligament or into the perineum) or burst into one of the internal organs or peritoneum. Secondary venous thrombosis with subsequent pyæmia is by no means uncommon. Adequate drainage, once suppuration has occurred, is the essential treatment.

## GENERALISED INFECTION

### SEPTICÆMIA

SEPTICÆMIA is the condition which results when the circulation becomes flooded with bacteria, due either to the failure of local natural defensive reactions at the site of infection or to delayed or inadequate treatment. Once in the blood stream the organisms proliferate therein and, in a true case of septicæmia, can always be found in blood cultures. A minor degree of this state of affairs in which organisms can only be sporadically recovered from the blood is known as "bacteræmia," and is rather the expression of an overflow from the local focus of infection than a true blood infection. Clinically the two conditions may often be indistinguishable.

Streptococci, especially the hæmolytic types, are by far the commonest cause, though staphylococci may also be found as well as many of the so-called specific organisms, such as *Pneumococcus*, *B. coli*, *B. pyocyaneus*, *Gonococcus*, *B. welchii*, *B. pestis*, *B. anthracis*, etc.

The actual site of infection may be very obvious as in the case of gangrene of part of a limb or a large area of cellulitis, but may on the other hand quite frequently be almost indistinguishable. It is well known that seemingly harmless small wounds, scratches or pricks may give rise to a fatal septicæmia. The wounds from post-mortem examinations and septic operations are notable examples.

**Clinically** septicæmia is usually initiated by a severe rigor, which is seldom repeated. The temperature, except in the worst cases when it is subnormal, rises to a considerable height (104° to 105° F.) and remains about this level. The pulse is fast and weak and the heart becomes dilated. Hæmic murmurs are often present. The skin is dry and hot and may show multiple petechial patches (from hæmolysis) or even an erythematous desquamating rash. The tongue is parched and furred. Thirst is insatiable, but the appetite disappears. Diarrhœa is more common than constipation, the stools often being streaked with blood and mucus. The urine also contains blood and albumen and is scanty and highly coloured. Leucocytosis is well marked, except in the worst cases. Restlessness and sleeplessness soon pass into active delirium and, unless treatment is successful, coma will ensue and death occur in less than a week.

Post-mortem changes are quite distinct. Rigor mortis is late and slight, but decomposition and post-mortem lividity rapid and marked. The blood is very dark and coagulates slowly. Serous cavities contain small quantities of blood-stained fluid, and subendothelial petechial hæmorrhages of the pleura, peritoneum, pericardium and lining of major vessels are very typical. The spleen is enlarged, soft and friable and, like the liver and kidneys, shows definite cloudy swelling. The lungs are congested and œdematous.

**Treatment.**—The local focus should be either excised or thoroughly cleaned and adequately drained. Amputation may be indicated.

Rest in bed is, of course, essential and ample fresh air, a good

nourishing diet, stimulants (especially alcohol) and soporifics must be provided. To ensure an adequate intake of fluids it should be a routine to give 7 to 8 pints of 10 per cent. glucose saline daily either

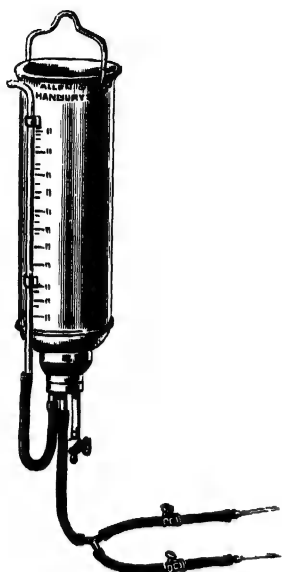


FIG. 6

Apparatus suitable for either subcutaneous or intravenous saline infusion; for the latter only one needle would be required.

by the intravenous (continuous drip method) or rectal routes (Figs. 6 and 7). Sulphapyridine is a specific remedy for many cases of septicæmia as the majority of responsible organisms are susceptible to the drug; sulphathiazole will be used for staphylococcal cases. In the remainder a specific antitoxin will often be available.

A chronic form of septicæmia is described, although this is probably rather a bacteræmia and often due to a less virulent strain of streptococcus (*e.g. S. viridans*). The clinical picture is milder in degree, but the ultimate result in the absence of discovery and adequate treatment of the source of infection is equally fatal.

### SAPRÆMIA

Sapræmia is synonymous with "toxæmia" and implies a condition in which the blood is infiltrated either with poisons actually manu-

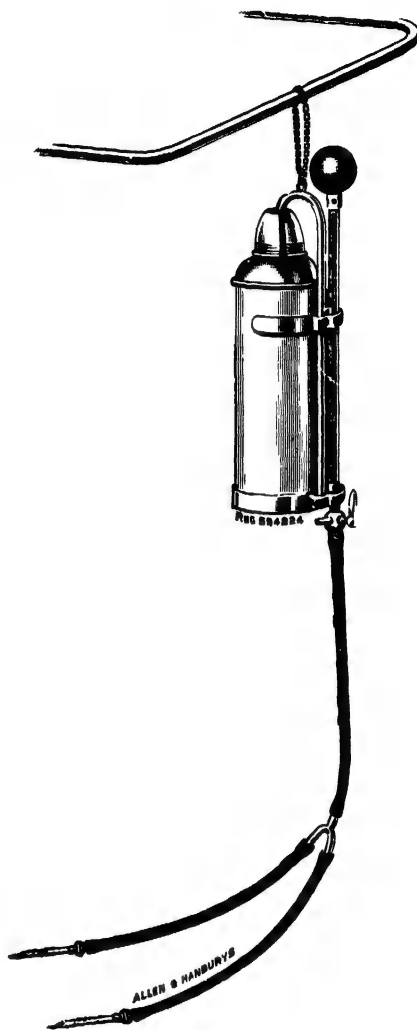


FIG. 7

A modified thermos flask adapted for use with subcutaneous, intravenous and rectal saline infusions. For rectal use a soft rectal catheter would be substituted for the needles.

factured by organisms at the site of infection (*e.g.*, tetanus and diphtheria) or by the toxic protein by-products produced in the infected focus by the proteolytic action of saprophytic bacteria. The organisms themselves remain localised by natural protective barriers to the site of infection. Blood cultures are therefore negative.

The individual powers of resistance of the patient as well as the virulence of the attacking organism will cause considerable variation in the clinical picture. Naturally the more diffuse forms of inflammation and the infection of large organs or cavities will produce more marked symptoms. It should be remembered, however, that quite small collections of pus, if retained under tension, will lead to severe toxæmic reactions.

**The clinical picture** is practically identical with that of septicæmia, although there may be no rigor at the onset. High temperature, rapid pulse, dry skin and tongue, anorexia and thirst, constipation, headache and delirium are the chief manifestations.

**Treatment** is directed to the primary focus, the general resistance being maintained to the greatest possible extent by such measures as have been described in connection with septicæmia. The administration of copious fluids is of the utmost importance.

### PYÆMIA

This is a condition in which multiple abscesses develop in various parts of the body, due to the presence in the blood of septic emboli. These emboli may consist either of masses of bacteria or of small detached pieces of infected blood clot from the thrombus in a vein at the site of primary infection. The first type is seen principally in cases of infective endocarditis, the second in any focus of infection, frequent sites being bones (osteomyelitis and otitis media), the puerperal uterus and abdominal inflammations.

The streptococcus is the commonest organism to be disseminated in this manner, but the staphylococcus, pneumococcus, typhoid bacillus and many others may be at the root of the trouble.

These emboli are washed off free into the blood stream and travel until they reach an arteriole or capillary, the diameter of which will not permit them to pass. As soon as they are held up bacterial activity increases and an area of inflammation is set up, in which in most cases pus is formed with a resultant secondary abscess. If the original septic clot lies in some part of the systemic venous system, the emboli get carried to the right heart and so come to rest in the peripheral pulmonary vessels; if the emboli start from the heart valves they may be held up in any peripheral systemic radicle; and if primarily in the portal system, the secondary abscesses are in the liver (the condition of *pylephlebitis*).

If the primary site of infection is accessible it will clinically be found to present an area or wound, inflamed but without much reaction, in which it may be possible to feel the firm, tender thrombosed veins.

It is, however, the general picture which is so distinctive. The onset is accompanied by a sudden and severe rigor and this is repeated

afterwards at approximately periodic intervals. The temperature swings in a similar manner, rising to 104° F. or more immediately after the rigor, only to fall in twenty-four to forty-eight hours towards normal. The repeated rigors and hectic intermittent temperature chart are practically pathognomonic of the condition. At the same time the pulse is rapid and of poor volume, the skin dry, hot, and often erythematous, the tongue parched and brown, and the complexion sallow. The breath, owing to the presence of acetone, often presents a typical mawkish, hay-like smell. Weakness develops rapidly and delirium at night is usual. By the end of a week, if the patient survives the toxæmia accompanying the first flood of septic emboli, the secondary abscesses begin to make their appearance. These are commonest in the lungs, and their appearance is accompanied by local pain and dyspnoea. Serous cavities and joints are frequently involved and, if the main brunt of the attack falls on such vital organs as brain, kidney or heart, the result is likely to be rapidly fatal.

The secondary abscesses may contain frank pus, but more often a thin watery sero-pus forms their content. In both cases the fluid teems with the causal organism. Tender patches in the skin, without actual abscess formation, show where emboli have lodged in areas, the natural reaction of which is strong enough to meet the new attack. The abscesses are often surprisingly painless and are characterised by an almost complete absence of surrounding inflammatory reaction.

**Treatment.**—At the primary site of infection every effort must be made to prevent the spread of further septic emboli. The method of choice is to ligature the offending vein proximal to the clot and clear out all debris. In some cases actual amputation may be indicated. At least it is possible to open up the causal wound or area, clear out clot, etc., and ensure good drainage.

As a prophylactic measure some surgeons advocate a preliminary ligature of the main venous drainage from a septic focus that is being dealt with surgically, *e.g.*, the appendicular vein in acute purulent appendicitis.

The secondary abscesses can be treated either by aspiration or by opening and drainage. In the early stages of their formation this latter procedure is inclined to lead to further spread.

General treatment, which is of vital importance, follows similar lines to those advocated in cases of septicæmia.

A. E. PORRITT.

R. M. HANDFIELD-JONES.

## CHAPTER IV

### SPECIFIC INFECTIONS

#### TETANUS

**T**HE *Clostridium tetani* is a rod-shaped bacillus, 5  $\mu$  in length ; it is gram-positive, strictly anaerobic and possesses numerous flagellæ, in spite of which it is only feebly motile. It is not easy to cultivate, giving a scanty surface growth, in which after forty-eight hours the bacilli exhibit their characteristic terminal spores, from which the name "drum-stick" is derived.

**Tetanus bacilli** do not invade the body but, being implanted in a wound, they multiply *in situ* and develop a powerful exotoxin. This toxin is absorbed by motor end-plates in the recesses of the wound and passes up the axis cylinders to the anterior horn cells in the spinal cord. If produced in great quantities it may also be absorbed by the lymphatics and reach the blood stream, from which it is taken up by motor end-plates all over the body. Once in the cord toxin spreads up the anterior horns until it reaches the vital centres in the medulla. In fatal cases the post-mortem findings are practically negative ; degenerative changes may be present in the anterior horn cells and in those of the pons and medulla.

**The Spores** are resistant to heat, desiccation and chemical antiseptics and the organism therefore has the power of lying latent for many years. Both spores and bacilli, washed free of toxin, can be injected without producing any evil result, but any factor which damages tissue, be it a concomitant infection, injury or necrosis of tissue, will provide the necessary pabulum for activation of both spores and bacilli and tetanus develops. This latency is of great surgical importance in that a secondary operation upon a wound or scar in a patient showing no signs of tetanus may precipitate an attack.

The **type of wound** which favours tetanus is of two kinds, either a lacerated wound with much tissue damage or a puncture with little or no drainage. The bacilli are found in large numbers in cultivated soil and in the intestinal canal of horses, so that people coming in contact with arable land and horse excreta are prone to tetanus if they sustain either of these types of injury ; in connection with the latter street accidents in towns must always be regarded with suspicion. In war fighting is so often over highly manured country that the incidence is high, and for this reason every wounded man receives a prophylactic injection of antitoxin.

The **incubation period** varies from four to twenty-one days and has an important bearing upon prognosis. The longer the interval between infection and the onset of symptoms the more favourable the outlook.



## CLINICAL TYPES

Several varieties are described :—

1. **Acute Tetanus** with a very short incubation period, a rapid onset of severe symptoms and a fatal result.
2. **Delayed Tetanus** with an unusually long delay in the onset of symptoms, which are mild and the prognosis is good.
3. **Chronic Tetanus**, when an old wound is reopened and symptoms develop.
4. **Local Tetanus**, in which only those muscles around the wound are involved. This is due either to a slight production of toxin or to the effect of antitoxin.
5. **Cephalic Tetanus**.—Infection enters a wound in the head and neck and affects cranial nerves. Incubation period is short, spasms of the muscles of the head and neck predominate and the prognosis is very grave.
6. **Tetanus Neonatorum** is due to infection of the umbilical cord at birth. It is accompanied by jaundice and is inevitably fatal.
7. **Operation Tetanus**, in which infection comes from spores embedded in catgut ligatures and sutures.
8. **"Fourth of July" Tetanus**, so named in America, where on this date fireworks are in great demand. Bacilli are present in the wads and padding (often made from hoofs or hair of animals). In this country percussion caps for toy pistols have a similar effect.
9. **Tetanus "Hydrophobius"** in which symptoms are largely pharyngeal and the clinical picture somewhat resembles that of rabies.

## CLINICAL PICTURE

An attack is ushered in with vague indefinite prodromal symptoms. Stiffness of the muscles around the wound with occasional twitchings may be the first warning or the stiffness may be in the posterior muscles of the neck. Soon this is followed by similar signs in the masseter muscles. Shortly these are affected by violent spasms, first the masseter and temporal groups giving the characteristic trismus (*lockjaw*). Muscles of the neck, of the anterior aspects of thorax and abdomen, of the back and of the limbs follow in that order. The hands are the last parts to feel the effects of the toxin. The condition is essentially a primary tonic contraction of the affected muscles and the spasms may last for a few minutes to several hours. In severe or progressive cases there may be superimposed a secondary clonic spasm, which gives rise to certain well recognised positions ; *opisthotonus*, when the back is arched off the bed (and the rectus abdominis may be ruptured) ; *emprosthotonus*, in which the reverse position is taken up ; *pleurosthotonus*, when the body is bent to one side or other, typically to that of the original injury.

A further characteristic spasm is that of the facial muscles producing the horrible fixed grin, known as "*risus sardonius*." All these contractions are agonisingly painful and their repetition leads to great exhaustion. The temperature in the early stages has probably shown

a moderate degree of pyrexia due to sepsis in the wound, but towards the end it may rise to phenomenal heights ( $110^{\circ}$  or  $112^{\circ}$  F.). There will be excessive sweating and a scanty urine loaded with albumen. The mind remains distressingly clear throughout.

The differential diagnosis includes strychnine poisoning, hydrophobia, tetany, meningitis, hæmorrhage into the cerebral ventricles, catalepsy and hysteria.

### TREATMENT

**A. Prophylactic.**—In war every wounded person, and in peace every patient with a wound in any way likely to be contaminated with tetanus, must be given a prophylactic dose of antitetanic serum (A.T.S.). In this country the Ministry of Health instructions are that 3000 units (U.S.A. 1500 units) are to be injected intramuscularly immediately on arrival at a casualty receiving hospital, and the dose and time of its administration are to be entered on the casualty card. This dose is unnecessarily generous and 1500 units are amply sufficient.

**B. Therapeutic** treatment has four objects : (1) to neutralise toxin ; (2) to prevent its further absorption ; (3) to limit spasms and (4) to maintain the patient's strength. These are attained by the following procedures.

1. The use of antitetanic serum at the earliest possible moment after diagnosis is imperative. The route by which it is administered has been a matter of controversy in the past, but to-day it is generally agreed that the intravenous method is superior to all others. It is accepted that intrathecal injection is no more efficacious and it has the disadvantage of producing in many patients a considerable degree of shock. Local intramuscular injection is of little value.

The technique is as follows : an initial dose of 100,000 units A.T.S. is given intravenously ; it should be warmed and run in slowly. Twelve hours later 50,000 units are given, after which 25,000 units are injected every twenty-four hours for four days (see also p. 20).

2. Treatment of the wound provides a problem requiring a nice judgment. Unnecessary interference and rough handling may liberate an additional amount of toxin, so that more harm than good results. Treatment will depend upon the extent, site and number of wounds, whether one or all are infected with tetanus, and whether thorough excision is possible. Extensive lacerated wounds of the extremities with compound fracture may demand amputation as the surest way of saving life, whilst others may be ideal for excision. In any event operation should be delayed for three hours after the first dose of antitoxin ; the wound is then explored, all dead and devitalised tissue removed together with every particle of foreign bodies and every recess laid open. It is then treated with a powerful oxidising dressing, of which zinc peroxide is the most efficient. A cream is made of 40 per cent. free oxygen powder (supplied by Laporte & Co., of Luton, Beds.) and this is gently syringed into every part of the wound, the remainder of which is loosely packed with gauze impregnated with the cream.

3. All possible stimuli likely to excite spasms should be avoided and for this reason a perfectly quiet darkened room with careful and

silent nursing and the minimum of interference is essential. Cole has shown that avertin given per rectum in doses of 1 c.c. per kilogram of body-weight controls spasms better than anything else. Other drugs used are nembutal, chloral and paraldehyde.

4. As much nourishment as possible should be given. As a rule only fluids are acceptable and trismus may make it impossible to give it in any other way than rectally. If feasible a Ryle's tube should be passed into the stomach and left *in situ* for regular feeding.

Attention must be paid to the bladder as retention occurs in a number of patients.

## GAS GANGRENE

Gas gangrene results from a group of organisms and not from any single bacterium as does tetanus. They fall into two classes, those that ferment sugars and others that break down proteins. All belong to the type—**Clostridia**.

The sugar-splitting group (saccharolytic) include *Cl. welchii*, *Cl. septicum* (vibrio septicum of the French) and *Cl. oedematiens*, while the proteolytic representative is *Cl. sporogenes*.

*Clostridium welchii* is the most important and is here described as typical of the saccharolytic group. It is a large gram-positive bacillus, short, stumpy and square ended, is non-motile and produces spores only under special conditions. It ferments all hexoses and starch, and in so doing develops large quantities of gas. A milk medium shows a characteristic change—the so-called "stormy clot." It produces a powerful soluble toxin which has a specific effect upon cardiac muscle and against which an antitoxin can be prepared.

*Cl. sporogens* has no great pathogenicity but is usually associated with *Cl. welchii*, whose action it accelerates and accentuates to a marked degree. It is a gram-positive bacillus and is actively motile. It splits sugar to a slight degree but decomposes meat with the production of a foul smelling gas.

All these organisms are anaerobic and the wounds which favour their development are similar to those in tetanus. Essentially gas gangrene is a disease of muscle; other tissues such as subcutaneous fat and fascia may be affected, but only in muscle does this infection assume its formidable character. Involved muscles change their colour becoming a dull brick red (Fig. 8) and lose their contractility. Their fibres and bundles are separated from their sheaths, being surrounded by a clear space full of gas. In this way infection spreads rapidly up a muscle, which is killed partly by the bacterial toxins and also by deprivation of its blood supply by the gaseous distension of its sheath. In such a dead muscle bacilli multiply in large numbers, the vessels are thrombosed and the whole area is converted into a dead mass exhibiting gas crepitation. The discharge is not purulent but sanious, containing many bacteria and few leucocytes and emits so pungent and characteristic an odour that the diagnosis can be made upon that sign alone.

Gas gangrene varies somewhat in its manifestations. The foregoing description is typical of most infections, in which there is a reasonable

expectation that energetic treatment will save the patient's life and possibly his limb also. Less commonly in very severe injuries, especially when the main vessels of a limb are damaged, massive gas gangrene of the whole limb supervenes and the outlook is hopeless. In such cases a gas septicæmia is likely to usher in a fatal ending and at post-mortem bubbles of gas will be found in almost every organ, notably the spleen and liver.

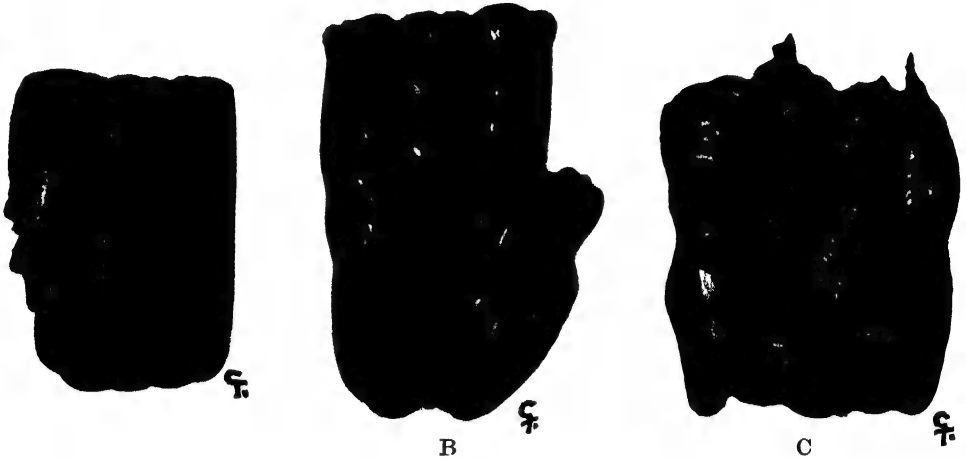


FIG. 8

Gas gangrene

A, normal muscle; B, "red death"—note the cavitation by bubbles of gas;  
C, "black death."

(*Surgery of Modern Warfare.*)

**Types of Wound.**—We have shown that wounds prone to gas gangrene are similar to those in tetanus, viz. : lacerated wounds with much tissue damage and punctures with little or no drainage. It has been our experience in peace time that another injury liable to this infection is one in which an extensive flap of skin has been widely raised from the underlying deep fascia with extensive soiling but without much muscle damage. In war military casualties present a high incidence of gas gangrene, and during the bombing of Britain we found a high proportion of bomb wounds containing *Cl. welchii*. In many of these cases contamination has been by the dust and powder of house debris and not street refuse.

There is yet another type of gas infection, which may be termed "autogenous gas gangrene." Wounds of the buttock and perineum in which gross or unsuspected damage to the rectum is a complication sometimes develop an acute infection from organisms normally present in the fæces. Similarly in rare cases fæcal soiling of the abdominal parietes during operations for intestinal obstruction may have the same unhappy effect.

### CLINICAL PICTURE

The incubation period varies between twenty-four hours and a few days, but is usually short. During the first few hours the wound

appears to be progressing normally and the patient is recovering from the shock of the accident when, with little or no warning, an acute cellulitis and a general septicæmia develop. Slight tension on the stitches, eversion and pallor of the edges and an absence of any but a slight watery discharge are introductory signs. The parts rapidly become very swollen and gas crepitus can be elicited. When the wound is reopened a foul smelling serous discharge pours out, the subcutaneous tissues are seen to be dirty grey or black and the muscles are swollen, emphysematous and of a dull, glazed brick red colour, which later changes to green and then black. The skin is at first bright red, but then becomes dusky, deep purple and then black.

In the early stages if gas gangrene is suspected, but no crepitus can be appreciated, an X-ray photograph may be of great assistance as the bubbles of gas are plainly visible in the films.

**Prognosis** in the majority of patients is quite hopeless, unless active treatment is undertaken at the outset. There are, however, less acute cases which respond to local treatment and in which the toxæmia is not severe; in these patients temperature is moderately high, there are rigors and vomiting and a single group of muscles will be found affected. The toxæmia in fulminating cases is so great that temperature is subnormal, the face pale and haggard, the tongue dry, brown and furred, and vomiting, delirium and coma usher in the fatal ending.

## TREATMENT

**A. Of the Primary Wound.**—The general principles of immediate wound treatment are laid down on pp. 124-126, and a faithful adherence to them will lead to a reduction of clinical gas gangrene to very small proportions. Trueta attributes his success in avoiding this dire infection to his method of immobilisation in plaster of Paris. If at any time within the first few days the temperature rises and the patient complains of pain, the plaster must **immediately** be removed and the wound examined.

**B. Suspected Gangrene.**—If at any time the slightest suspicion of gas infection arises, the wound must be reopened and thoroughly inspected in its every extension. Should no gas be found, nothing more need be done except to powder every surface and recess with sulphanilamide powder, pack lightly with vaseline gauze, insert no sutures and immobilise in plaster of Paris.

**C. Of an Established Infection.**—Here we are faced with the problem of conservative surgery or amputation. We can distinguish between four forms or degrees of lesion, (a) local infection of the wound and its immediate surroundings, (b) infection of a single muscle or group of muscles, (c) generalised spreading gas gangrene of the limb and (d) a severe infection complicated by compound fracture or division of the main vessels. Clearly the last two demand immediate amputation as a life-saving measure, while the first group are ideal for thorough excision and immobilisation. It is the second class which gives such anxiety and calls for a nicely balanced judgment. Attempts to save the limb may lose a life and some amputations will be performed when

excision would have sufficed. If the infected muscle can be wholly removed and all damaged tissue excised conservative treatment is justified. It must be followed by the most unremitting attention to every aspect of the patient's condition.

**D. Chemotherapy.**—It has not been possible to collect sufficient statistics balanced by an equal number of similar but untreated controls to permit a dogmatic statement upon the value of sulphanilamide. Our impression, however, from duty at St Mary's Hospital throughout the whole of the bombing of London has been such that we believe implicitly in the potency of this drug both in wounds and by mouth for several days after injury. We have had a number of cases in which *Cl. welchii* has been demonstrated in the wound both at the primary operation and at the first dressing twenty or thirty days later without a sign of clinical infection. Suffice it to say that we should feel that we had failed in our duty if we omitted sulphanilamide from our technique.

**E. Antitoxin.**—The prophylactic use of anti-gas gangrene serum rests upon less solid foundations than that in tetanus. Early in the war supplies were limited and serum was used for therapeutic purposes only; since it has become more plentiful 1000 units are being given at the same time as A.T.S.

In the presence of an established infection large doses of intravenous serum are administered along the same lines as in tetanus.

The real hope of preventing gas gangrene is in the early treatment of wounds and that again depends upon the exigencies of the military situation. If wounded people could be operated upon within six hours we should be able to reduce gas gangrene to a curiosity. No surgery however brilliant will prevent it in great numbers if cases cannot be evacuated to receiving hospitals within a few hours of the receipt of their wounds.

## TUBERCULOSIS

### ETIOLOGY

Tuberculosis is a chronic infection due to the tubercle bacillus and is responsible for more human deaths than any other disease, namely 15 per cent. The organism belongs to the group of mycobacteria which, on account of their staining reactions in the Ziehl-Neelsen technique, are also known as acid-fast bacilli.

The bacillus was first discovered by Koch in 1882. It is a slightly curved rod about  $4\mu$  long, aerobic, non-motile and difficult to stain and culture. It is surrounded by a lipid envelope which accounts for 25 per cent. of its weight and for its powers of resistance and long life. Four types are recognised, avian, piscine, bovine and human, of which the first and second are non-pathogenic to man.

Although both human and bovine bacilli produce similar lesions, stain identically and grow under the same conditions, they exhibit considerable differences in their virulence and cultural behaviour and especially in their relationship to their human host. On Dorset's egg

medium the growth of each is somewhat different and this variation is markedly accentuated by the addition of glycerin to the medium. But their differentiation is more strikingly revealed by two biological tests, one upon rabbits and the other upon calves. If 0.1 c.c. of a recent culture is injected intravenously into a rabbit, the human strain produces scanty and slight lesions, whereas bovine bacilli lead to a fatal result within a few weeks. Similarly in calves a subcutaneous injection of human bacilli gives a small local reaction, but rapid dissemination all over the body occurs with bovine organisms.

Of greater interest, however, is their relationship to the human being and statistics reveal a remarkable difference in the site of the lesions and the age of the victim. Bovine tuberculosis is rare after the age of 16 years in any part of the body; below this age only the cervical and abdominal lymph glands show a higher incidence of bovine over human infection and only bones and joints are attacked in a reasonably high proportion (26 per cent.) by the bovine bacilli. It is evident therefore that human tuberculosis is preponderantly an infection by the human strain of bacillus. Infection occurs in three ways, by inhalation, ingestion in milk and local invasion via a breach in the skin. This last is distinctly rare.

### MORBID ANATOMY

In the usual mode of infection bacilli enter the lymphatic system and so reach the nearest group of lymphatic glands. Here they may be held up or pass on into the venous system and so to the lungs. In a massive or virulent infection those that pass through the lungs will reach the systemic system and so become disseminated to distant parts of the body.

According to Muir the tissue reaction to the presence of the bacilli varies according to whether the patient is being infected for the first time or reinfected after a previous attack. In the first case the reaction is proliferative (resembling that to neoplastic cells) rather than inflammatory. Epithelioid cells of reticulo-endothelial origin—wandering large mononuclears—become massed around the bacilli, and this group of cells in about two weeks become visible to the naked eye. It is the size of a pin-head, transparent and greyish white, and is known as a *tubercle*. The fatty envelope of the bacillus acts as a foreign body leading to the production, by fusion of several epithelioid cells, of a typical giant cell, with anything from twelve to twenty irregularly arranged peripheral nuclei (Fig. 9).

The "tubercle" being avascular soon undergoes a coagulative necrosis from the action of bacterial toxins in the absence of adequate blood supply, the result being a central core of dead granular cheesy material ("caseation") which may later, when the constituent fatty acids become saturated, liquefy to form tuberculous pus, a sterile, serous solution containing fatty debris.

It is the second or "reinfection" type which is usually seen in man, this being inflammatory in character and more rapid in development. There is a typical leucocytic reaction for the first two or three days



before the changes enumerated above begin. Lesions in this type tend to break down more readily, and each fresh infection produces a higher degree of sensitivity. This, really an allergic reaction, is the basis of the diagnostic von Pirquet and Mantoux tests.

A tubercle therefore consists of a central mass of caseous tissue, either liquefied or not, in which the causative bacilli are seldom found, surrounded by a circle of giant cells, outside which comes the zone of endothelioid cells merging through a small round-celled reaction into normal tissue.

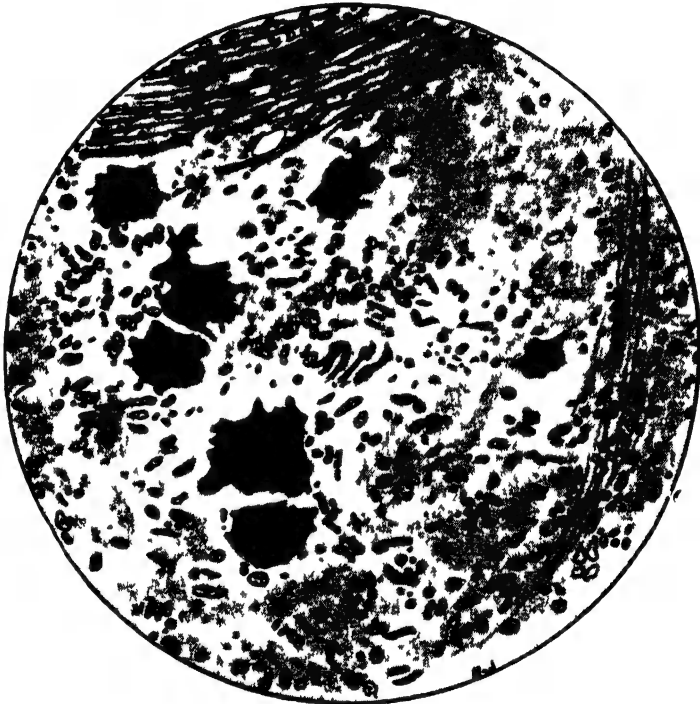


FIG 9

A microscopic drawing of tuberculous giant cells

The tubercle may fuse with neighbouring foci to produce a clinical tuberculous ("cold") abscess; it may produce granulation tissue without caseation (the proliferative, hypertrophic type seen in the cæcum or joints); it may retain its inflammatory character and spread locally (as seen in the meninges of the brain); or it may lead to a fibrous tissue barrier being formed around itself, producing the so-called "healed tubercle." These last quite frequently calcify, but even so they may contain living bacilli.

Spread from a tuberculous focus is by :

1. Lymphatic channels ;
2. Natural passages, *e.g.*, ureter, bronchus ;
3. Permeation, via wandering endothelial cells through neighbouring tissue spaces ; and
4. Blood stream, either directly as seen in miliary (generalised) tuberculosis, or secondarily via lymphatics to venous system.



The clinical picture varies from organ to organ and according to the severity of the infection and the resistance of the patient. It will be described in detail in considering the diseases of the various parts and structures of the body. But it is worthy of note that a definite clinical type (the so-called "scrofulous" type) of person seems to be more prone to the attack of the tubercle bacillus. Such people are fair-haired, often rosy of complexion, commonly freckled with long eyelashes and fine features, and frequently mentally precocious.

Only principles of treatment can be mentioned here. The first and greatest is rest. Every effort should be made to raise the patient's own power of resistance, by good food, tonics, fresh air and sunlight. Any secondary septic foci must be adequately dealt with. Counter-irritants may be used or injections of tuberculin given. This latter treatment is of particular service in genito-urinary tuberculosis. Surgery may be required to secure mechanical rest to an affected part or organ, to excise or even amputate a localised lesion, to curette and cauterise tuberculous foci and to aspirate abscesses.

Tuberculosis is an infectious and notifiable disease.

## LEPROSY

LEPROSY, a rare disease in this country, is caused by the *B. lepræ*, which resembles the tubercle bacillus, but is smaller and straighter. The condition, which, despite its historical notoriety, is only slightly contagious, has an insidious onset some considerable time (even several years) after infection.

Two clinical types are described :—

(a) **Nodular Cutaneous Leprosy** and (b) **Anæsthetic Leprosy**.

The NODULAR type is that usually seen in Eastern Europe. Bright red shiny spots, which later become indurated, occur in crops along the course of a cutaneous nerve. The arrival of each crop is heralded by a pyrexial attack. The skin over the nodules is hyperæsthetic and becomes roughened and pigmented, later ulcerating or atrophying to leave a round septic surface. The nerves concerned are often palpable. Areas commonly affected are the face, forearm and external aspect of the thighs. The lymphatic glands draining these areas may be enlarged.

The ANÆSTHETIC type occurs in the Far East and tropics. In this, one of the main nerves of a limb (*e.g.*, ulnar, median, peroneal, saphenous) becomes excessively painful and the skin of its area of distribution anæsthetic. Trophic changes follow in due course, bones being decalcified, muscles wasted and skin smooth, yellowish and parchment-like. Ulceration and sepsis follow, and deformities are horribly unsightly.

The disease is very chronic and it may be anything from ten to forty years before chronic sepsis or lung infection leads to a usually welcome death. Spontaneous cure has been reported, usually after some acute intercurrent disease.

**Treatment** is non-specific and consists in raising the general,

physical and mental health to such a standard that some particular line of therapy may be applied. Vaccines, both stock and autogenous, have given way to the administration, intramuscularly or intradermally, of the ethyl esters of chaulmoogra oil. This must be continued over several years, and during this period local treatment with trichloroacetic acid greatly reduces local lesions. Lepers should be segregated.

## ANTHRAX

The anthrax bacillus is one of the largest of the species, 10 to 18  $\mu$  in length and 1 to 1.5  $\mu$  in breadth. It occurs in chains, is non-motile, gram-positive and only forms spores outside the body. Its usual habitat is the intestinal canal of herbivora. Man is not in most cases particularly sensitive to its attack—the result being usually only a local lesion either on the skin, in the lungs or the alimentary tract, according to the method of infection. In animals (horses, sheep, cattle) it produces the so-called *splenic fever*.

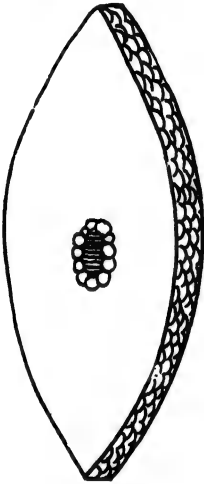


FIG. 10

tule. In the centre is the necrotic area, which is surrounded by a ring of vesicles.

**Clinically.**—1. RESPIRATORY TYPE (“Wool-sorters’ Disease”).—The pathological changes in this type are those of a typical pleuropneumonia, the bacilli being found in the sputum.

2. ALIMENTARY TYPE.—This is characterised by chronic diarrhoea and the passage of blood. Both this and the respiratory type are rapidly fatal.

3. CUTANEOUS TYPE (Fig. 10).—This is the lesion that is usually seen by the surgeon. There is an incubation period of a few hours to a week, the latter being more common.

A red pimple then becomes evident on some exposed part (face or hands), which develops into an isolated, painless papule. About the third day after its appearance vesicles are formed and the skin around has become dusky and indurated. The vesicles contain blood-stained serum (not pus, as the name “malignant pustule” would imply) and soon burst, leaving an umbilicated scar with a blackish slough at the bottom. This scabs over and heals spontaneously in about a fortnight. But meanwhile the discharged fluid has infected the surrounding skin, and so the process continues. The neighbouring oedema is sometimes so marked as to produce a characteristic white ring around the whole area. Occasionally this oedema spreads rapidly and may prove fatal by involving the larynx. More rarely still a true septicæmia develops which is invariably fatal.

The whole lesion is painless, but itching is common. Neighbouring lymph glands, however, are swollen and tender and teem with bacilli.

**Treatment.**—Sclavo’s antitoxic serum prepared from the white ass,

or others produced from horses, has given such good results that no local treatment is either necessary or desirable. It should be given in an initial dose of 200 c.c. followed by 50 c.c. daily. By this means the mortality of the skin lesion has been reduced to under 5 per cent.

## DIPHTHERIA

DIPHTHERIA is an infectious and notifiable disease due to the Klebs-Loeffler bacillus. This is usually a long slender rod, gram-positive, non-motile and beaded in appearance when stained. This irregularity is due to the presence of metachromatic granules, which stain more deeply. Another characteristic feature in a film is the peculiar arrangement of the bacilli giving a pattern akin to Chinese letters. There is an incubation period of two to seven days. Locally the infection usually attacks the pharynx and larynx, leading to a colliquative necrosis and the formation of the characteristic membrane. Surgically this is of importance as it may obstruct the respiratory passages and necessitate a tracheotomy. More rarely diphtheria affects the nose, conjunctivæ, external genital passages and open wounds.

As well as the local effect, viz., the production of a tough, adherent, whitish membrane, the bacilli manufacture an exotoxin, which in the blood stream leads to generalised changes, particularly in muscles (including the heart) and nerves. Hence post-diphtheritic paralysis may occur and require suitable treatment (see Chap. XLIII). The muscles most frequently affected, apart from the heart, are the ocular, palatal and musculo-spiral.

It is interesting that babies are immune to diphtheria, the highest incidence occurring between the ages of 2 and 5. Some individuals are "carriers" of diphtheria bacilli without themselves being affected. Immunisation of all children should be made compulsory.

**Treatment** is by the specific antitoxin, which is given in doses of from 10,000 to 50,000 units, and may be repeated once or twice as indicated.

## GLANDERS

Glanders is a rare disease in man. It is caused by the *B. mallei* and affects chiefly the horse, in which it presents both acute and chronic forms. As it is contagious, man is secondarily infected via some abrasion of skin or mucosa on exposed parts, and the disease is always acute. It is usually initiated by contact with the nasal discharge from an infected horse.

The *B. mallei* in many respects resembles the tubercle bacillus. It is a slender, gram-negative rod, difficult to stain except by carbol fuchsin and best cultured on potato, when it produces a characteristic yellow growth which later darkens to a chocolate tint.

**Clinically** there is an incubation period varying from two to three days to as many weeks—usually the former. The first signs are catarrhal inflammation of mucosæ of mouth and nose, together with

the appearance of papular skin nodules. These develop rapidly into a crop of pustules, which break down to form a ring of irregular ecchymatous ulcers, the floors of which are covered by unhealthy slough and from which comes a foul blood-stained and infective discharge. Pathologically the lesion is an atypical infective granuloma.

Neighbouring glands are enlarged and tender, and constitutional symptoms soon make their appearance. Pains in bones from osteoperiostitis are the most frequent symptoms and may be the first signs of the onset of the disease. Actual metastases occur and produce nodules in lungs, liver, spleen and kidneys. Muscular abscesses and venous thrombosis are common.

The mortality is very high, two out of three cases proving fatal.

The chronic form is seen only in the horse. This affects chiefly the lymphatic system, giving rise to what are called "farcy pipes" and "farcy buds."

A vaccine termed "mallein" has been used both for diagnosis and treatment, but its effects are so marked that it is really dangerous. Diagnosis can be established by injecting some of the suspected material from a skin lesion into the peritoneum of a guinea-pig. In positive cases there is within twenty-four hours an acute inflammation of the tunica vaginalis (Strauss reaction), and fluid from this contains masses of bacilli which give a typical growth on potato.

**Treatment** is purely symptomatic. Excision is the ideal if the disease is recognised early enough, otherwise opening and curetting of the abscesses offers most chance. This, of course, is combined with suitable measures to enhance the patient's general resistance.

## PLAGUE

PLAGUE is due to *B. pestis*, first discovered in 1894. The disease, which is now seen practically only in Asia apart from sporadic cases, is transmitted by one species of rat-flea (*Cheops*) and flourishes in conditions of poor sanitation. There is an incubation period of four to five days, the onset being accompanied by high temperature, headache and often epistaxis. Lymphatic glands (inguinal and axillary) enlarge in the bubonic type and occasionally suppurate. The skin is covered with petechial spots (plague spots). Four out of five cases die. More severe forms are termed pneumonic and septicæmic and are invariably fatal. Buboes may need incision. Inoculation with vaccine (Haffkine's) has been reported to do good.

## RABIES

This is a dire disease, caused by filter-passing virus introduced into the victim by the bite of a mad dog. Infection has also been known to come from wolves, horses, cats, pigs and even birds. It is most commonly seen to-day in India. The tragedy of the disease lies in the length of its incubation period. What seems and is treated

as a trivial injury at the time may lead, months later, to a fatal issue. The incubation may be as short as twelve hours, is much more usually six weeks to two months and may be more than a year.

The **clinical picture** develops insidiously with feelings of malaise, chilliness and probably an aching in the original wound. Giddiness and a subjective feeling of terror follow, and then appear the typical clonic rhythmical spasms affecting chiefly the muscles of deglutition and respiration. The fear of initiating a spasm accounts for the refusal to take liquids (hence the synonym "hydrophobia") and the constant salivation. The temperature rises to great heights, the pulse becomes rapid and irregular, dyspnoea and hiccough supervene, the urine is loaded with albumen and a pre-terminal period of delusions amounting to mania lead within two to four days to a fatal issue. Post-mortem examination shows very little, there being an occasional vascular thrombosis in the medulla, sometimes degenerative changes in the ganglion cells of the cranial nerves, especially the vagus, and usually the presence of the so-called "negri bodies" in the hippocampus and cortex.

**Treatment** is chiefly prophylactic. A suspected wound should be cauterised and allowed to bleed freely, venous circulation to the limb being blocked as quickly as possible. Pasteur's vaccine, made from an attenuated virus, is the great specific and is effective for at least six months. Once the condition has developed, treatment is only symptomatic. Massive doses of morphia are indicated and rectal feeding must be instituted.

## GLANDULAR FEVER

**Infectious Mononucleosis** is an acute infection, the origin of which remains in doubt, though there are some grounds for regarding it as a virus disease. It is characterised by pyrexia, a sore throat, which may be sufficiently acute to resemble Vincent's angina, a swelling of the spleen and lymph glands, particularly in the neck. The blood picture shows a white cell count between 15,000 and 40,000, in which from 40 to 80 per cent. of cells are mononuclear lymphocytes. In addition the patient's blood serum develops an agglutinin to sheep's red blood corpuscles (Paul-Bunnell test). These cases present problems of diagnosis, but general treatment leads to a speedy recovery.

## INFECTIONS DUE TO INTESTINAL BACILLI

### B. COLI

The *B. coli communis* is a normal inhabitant of the intestinal canal. It is gram-negative motile organism, with three to six flagellæ, it grows easily and produces gas and thin pus, which by itself is odourless. It is only when the bacillus deserts the lumen of the intestine that it becomes pathogenic. This, however, is very frequent and it is the commonest secondary infection in inflammations of the intestinal canal, e.g., appendicitis. It is also a frequent etiological

agent in such diseases as cholecystitis, pyelitis, ischiorectal abscesses, etc.

### B. PROTEUS

This is commonly found in the company of *B. coli* and, like it, is only pathogenic outside its natural habitat—the intestinal canal. It is usually present in appendicitis, many infections of the lower urinary tract and abscesses secondary to spread of inflammation from the gut. It is responsible for the particularly foul odour of so-called “fæcal pus.”

### B. TYPHOSUS

This organism is responsible for typhoid fever. It is a short, gram-negative bacillus, with anything up to twenty flagellæ, motile but, unlike *B. coli*, produces no gas in culture. The chief means of differentiating it from similar organisms depend on the fact that it produces specific agglutinins in the patient's blood—this being the basis of the diagnostic Widal reaction.

Infection is usually via polluted water, but food, soil and direct contact are other less common sources. The organisms in the typical case reach the lymphoid patches of the lower ileum, where they set up an inflammation which leads to mucosal ulceration and sometimes to severe intestinal hæmorrhage or perforation. The initial clinical picture consists of headache, malaise, epistaxis, increasing pyrexia, and the appearance of a characteristic rose-red rash. Another complication of surgical importance is the occurrence of thrombophlebitis either in the pelvic or femoral veins.

Detailed accounts of the disease and its treatment will be found in textbooks of medicine, but from the surgical viewpoint it should be remembered that the bacilli may be the etiological agent in cholecystitis, arthritis and osteomyelitis.

### DYSENTERY

Bacillary dysentery is due to infection from contaminated water, food, etc., by the Shiga and Flexner bacilli. Both these types are gram-negative and non-motile. They are found during the disease in the intestinal wall, neighbouring glands, and liver. The clinical picture is dominated by the excessive and continual diarrhoea, which leads to marked dehydration and may be accompanied by severe hæmorrhage.

Ulceration of the intestinal mucosa takes place, and the ultimate result of this may bring the case into the sphere of the surgeon by perforation and subsequent peritonitis, stricture formation and secondary intestinal obstruction, or a disseminated infection seen in joints and bones.

### B. PYOCYANEUS

This is a normal inhabitant of the intestinal canal, but not usually present to any great extent. Occasionally its presence can be recognised

by the peculiar blue-green colour of the pus it produces. When found in sufficient quantity to be easily recognised, the prognosis of the case is correspondingly worse. It is a motile, flagellate, gram-negative organism.

It may be seen every now and again in the discharge from a chronic bone sinus.

## INFECTIONS DUE TO COCCI

### ERYSIPELAS

Erysipelas, known many years ago under the more picturesque name of "St Anthony's Fire" or "Rose," is an infection of the epidermis or mucosæ with a hæmolytic streptococcus (*S. erysipelatus*). The acute inflammation so produced is in many respects similar to cellulitis but effects more superficial layers. Combined types are often seen (cellulocutaneous erysipelas), especially in lax tissue such as the eyelid or scrotum. The infecting organism obtains entry by some break in the surface epithelium, though this may be so small as to be invisible.

Ninety per cent. of cases occur in the face or scalp (Facial or Idiopathic type); the arms and hands account for most of the remaining 10 per cent.; more uncommon but noteworthy sites are the umbilical cord (*E. neonatorum*), operation wounds, scrotum, mucosæ of mouth, pharynx and larynx and vagina (puerperal *E.*).

**Clinically** there is an incubation period varying from twelve hours to three days before the appearance of the typical rash. This is accompanied by marked general signs of fever due to the absorption of virulent toxins. Vague pains and headache are rapidly followed by rigors, occasional vomiting and a high temperature (103° to 105° F.). The pulse is full and over 100, the tongue furred, thirst is complained of and anorexia, constipation, albuminuria and delirium complete a typical picture. The blood shows a marked leucocytosis.

Locally the rash is vivid red in colour and fades on pressure. The advancing edge is irregular, raised and clearly defined. This edge shows all the signs of acute inflammation, and just beyond it the skin lymphatics are crowded with streptococci, which induce a characteristic and marked lymphocytic reaction. As the rash spreads, which it does fitfully, the centre of the affected area quickly fades, but remains shiny. Pain is conspicuous by its absence and pus is never formed.

Occasionally vesicles and bullæ are seen, and as the infection dies down the skin desquamates, fine scales being shed. These scales and the occasional watery discharge from skin blebs are the only infectious elements in the disease. It is thus really only contagious, but for this reason is a notifiable disease. Glands draining the affected area are usually enlarged and may rarely break down and form abscesses.

Spontaneous recovery takes place in two to three weeks, but relapses are common. The mortality is about 5 per cent.

**Treatment.**—Patients must be strictly isolated to prevent the



infection spreading. Treatment has been revolutionised by the sulphanilamide drugs, which control the disease within a few hours.

The peculiarly beneficial effect of an attack of erysipelas on co-existent chronic infections (*e.g.*, chronic ulceration and eczema, particularly of tuberculous or syphilitic origin) and on certain types of neoplasm has long been noted, and has led to the treatment of sarcomata by a bacterial emulsion containing streptococcus erysipelatus (Coley's Fluid).

### PNEUMOCOCCUS

This is a gram-positive encapsuled diplococcus, usually found outside the cells of the organ or tissue affected and lanceolate in shape. It shows a marked propensity to attack serous membranes, and hence, apart from being the chief causal agent in lobar pneumonia, it can produce pleurisy (empyema), peritonitis, meningitis, pericarditis, arthritis, otitis, etc. Any of these infections may be sufficiently severe to lead to a widespread dissemination by the blood stream and a definite septicæmia.

Its pus is typically greenish white in colour and curdy in consistency.

Clinical signs and treatment are considered in descriptions of the various organs concerned (*q.v.*).

## INFECTIONS DUE TO SPIROCHÆTES

### SYPHILIS

The various clinical manifestations resulting from infection with the *Spirochæta pallida* and their treatment are dealt with elsewhere (Chap. V).

### YAWS

**YAWS** (*Frambœsia Tropica*).—A chronic granulomatous disease due to *Spirochæta pertenuis*, it is characterised by ulcerating nodules, usually single, always painful and affecting the face in the majority of cases. The average incubation period is about two months. The disease is contagious, but, unlike syphilis, there is no hereditary transmission. Again, mercury and iodides have no effect whatsoever, but the reaction to either intravenous arsenic or intramuscular sodium potassium bismuth tartrate is magical. Two treatments will probably cause the lesion to heal, although residual scarring is marked and unsightly.

## DISEASES DUE TO FUNGI

### ACTINOMYCOSIS

**Bacteriology.**—This infection is different to any that we have previously described. The causative organisms—*Streptothrix Actinomyces*—belong to higher groups of bacteria, a characteristic of which



is that they appear in long hyphal threads instead of single units, although under certain conditions they may assume a bacillary form. These actinomyces resemble fungi in that they grow in similar filamentous branching threads (mycelium). Certain species produce a specialised type of cell upon culture media (but never in the human body) named conidia. These are not true spores, though in suitable cultural conditions they will develop into mycelium.

In man the streptothrix can best be examined by taking a "sulphur yellow granule" from the discharge from a sinus. Teased out and examined microscopically it will show a tangled mass of mycelial threads, at the periphery of which are grouped a series of thickened radiating outgrowths known as "clubs." These are not spores but the swollen ends of mycelial threads which are regarded as a defensive mechanism on the part of the colony; they are never found upon culture. This radiating club-like pallisade is responsible for the name given to these colonies—the ray fungus (Fig. 11).

**Etiology.**—Similar organisms occur commonly upon grasses, ears of corn, pollens, etc., but these are aerobic and it is doubtful if they are pathogenic to man, although the disease has long been attributed to these fungi. Certain of the lower animals, notably cattle, suffer from similar lesions in the jaw and tongue, to which the names "wooden tongue" and "lumpy jaw" have been given, but in the former at any rate the affection is by an actinobacillus and not a streptothrix. Further, it is cured rapidly and completely by potassium iodide.



FIG. 11

An actinomycotic colony showing the mycelial threads in the centre and the clubs at the periphery.

In man actinomycosis is most common in agricultural people, farmers, farm labourers and all connected with horses and cattle. In all these straw chewing is second nature, but in spite of all this circumstantial evidence it is improbable that the human disease is derived from this source. More probably the infection comes from the animals with whom these people deal.

**Clinically.**—It affects men four times as frequently as women, and has a maximum incidence between the ages of 20 and 40.

Entering via the mouth, it is not surprising that two out of every three cases are cervico-facial in type; the intestinal tract, in particular the ileocæcal region and appendix, account for another 25 per cent.—presumably ingested; and the lungs for some 15 per cent., spread to this locality usually coming from the neck or more rarely the abdomen. It is rare for actinomycosis to spread in any other manner than by direct continuity.

The onset is insidious, and the characteristic lack of pain means

that cases are usually not seen until well advanced. A subcutaneous thickening develops first, slowly becoming a firm mass over which the skin is puffy and discoloured. The mass ultimately softens in the centre, breaks down and discharges to the surface through multiple sinuses. This discharge is semipurulent and contains the distinguishing sulphur granules. The mass steadily spreads and involves muscle and even bone. A marked fibrosis develops around the area, giving a typical brawny induration. Enlarged lymph glands are conspicuous by their absence.

The ideal treatment of the lesion, if discovered early enough, is complete excision of the whole area, but this is unfortunately rarely possible. In more advanced cases it may be of some value to curette the sinuses and open any obvious abscesses, subsequently covering the whole area with a sterile dry dressing. Massive dosages of X-ray therapy hold out the best hope of cure, which may be reasonably expected in disease of the neck. In other situations the prognosis is bad.

Over-optimistic claims have been made for sulphapyridine, but it is becoming evident that it only serves to clean up the secondary infection and has no specific effect upon the streptothrix.

### **SPOROTRICHOSIS**

This disease is one of the less common chronic infective granulomata. Its lesions resemble those of the tertiary stage of syphilis. At first a firm indurated mass forms, which slowly breaks down to produce an abscess and finally an ulcer. This discharges a thin watery pus. The causative fungus (sporotrichium) is not, however, usually found in this, but in scrapings from the ulcer. If cultured on glucose agar a typical blackish wrinkled pellicle forms, and this is the only sure means of diagnosis. The fungus usually gains entrance via some superficial abrasion. The reaction to potassium iodide therapy is good.

### **BLASTOMYCOSIS**

Another rare granuloma, due to a yeast-like fungus (blastomycetes), is characterised microscopically by small spherical bodies (10  $\mu$  in diameter) with a typical double contour and showing free budding. The infection is introduced through minor wounds into the skin and produces a slow localised granulomatosis. Ulceration follows abscess formation, but spontaneous healing occurs at one place, whilst spread takes place at another. It has been reported as very rarely becoming a generalised infection, affecting particularly lungs, bones and muscles.

### **DELHI BOIL**

Baghdad Sore, Aleppo Boil or Oriental Sore is a tropical disease said to be due to a minute parasite carried either by dust or flies. The face is chiefly affected. An indurated papule develops and slowly breaks down into a typically crusted ulcer. In the discharge from this the characteristic clear ovoid cells with well-marked nuclei are found.

**Treatment** is by intravenous injection of a 1 per cent. solution of antimony tartrate.

### MYCETOMA

**Madura Foot** is an affection due to an organism resembling actinomycosis. It occurs almost exclusively in the tropics in natives who go about barefooted. An indurated granuloma forms which slowly breaks down into a chronic abscess, and this ultimately discharges a thin watery pus through multiple sinuses. There is little pain, no glandular involvement and no generalised spread. Iodides have no effect and local treatment is unavailing. The whole foot slowly becomes disorganised and amputation has to be resorted to.

## INFECTIONS DUE TO WORMS

### HYDATID DISEASE

This is the cystic stage in the life-history of the *Tænia echinococcus* for which man acts as one of the intermediate hosts. The condition is fully described elsewhere (Chap. VI).

### TRICHINOSIS

Infection occurs by the ingestion of meat, usually pork, contaminated with the small round worm—*Trichina spiralis*. The worm escapes from the alimentary canal and is carried by the blood stream to various parts of the body. For some unexplained reason the muscles are usually the favoured site, particularly those of the shoulder girdle (trapezius and deltoid). Here the worm settles down, becoming encapsuled and very often ultimately calcified. The blood-stream infection is marked by a feeling of malaise and perhaps slight fever. Within a day or two the affected muscles become tender and swollen. If, as is often the case, incision is made on the supposition that the condition is an acute infection, the muscle will exude a clear serum, often containing worms. These can be seen naked-eye in the muscles as small white dots. After the acute stage the disease is symptomless, although patients are prone to occasional attacks of urticaria. Santonin is a specific for the worm in the alimentary tract.

### CYSTICERCOSIS

This is the name given to the intermediate, or cystic, stage of the life-history of the *T. solium*. This stage usually occurs in the pig ("measly pork"), but man is occasionally affected indirectly and very rarely directly. The cysts become surrounded by fibrous tissue and frequently calcify. Any organ may be affected, and any clinical signs produced are those due to the presence of the calcified cyst, *e.g.*, in the lung, eye or brain. **Treatment** is excision.

**BILHARZIA**

This disease is due to a parasite called the *Schistosomum hæmatobium*—one of the trematode worms. Its life-history is as follows: The ova are shed in human fæces or urine. If they reach water in the process their capsule is dissolved and a freely swimming embryo (miracidium) emerges. This has a life of about thirty-six hours, and if during that time it is ingested by a particular type of water-snail it lives in its digestive gland and continues to develop into a sporocyte. This in about six weeks becomes filled with the primitive worms (cerceria), characterised by a forked tail and two suckers. These are voided by the snail and can live for thirty-six hours in the surrounding water. If during this period they enter the human stomach in drinking water, they lose their tails, burrow through into the portal system radicles and develop into adult worms in about six weeks. The female of the species then swims against the blood stream to either the rectal or vesical mucous membrane (more rarely stomach, vulva, skin, lungs and intestine are affected) from which the ova are shed, so completing the cycle. It is these ova which produce the clinical picture of the disease. They are about 15 mm. in length and vary in shape according to their habitat, the vesical type having a terminal spine, the rectal a lateral one. Their presence in the sub-mucosa is most irritating and leads to the production of masses of soft granulation tissue, which project into the rectum or bladder and bleed easily. Hence frequency, tenesmus, hæmaturia or bleeding per rectum and anæmia are the predominant signs.

**Treatment** by intravenous antimony tartrate is specific. (Dose: gr.  $\frac{1}{2}$ . increased by gr.  $\frac{1}{2}$  daily until gr. ii is reached, this amount being given every second day until a total of gr. xx to xxx has been administered.)

**FILARIASIS**

*The Filaria bancrofti* is a very fine round worm some 3 in. long which lives in and blocks lymphatic channels, especially in the region of the groin. These worms give off countless embryos which make their way to the blood stream, from which they are sucked by mosquitoes and so re-injected into another man. The embryos, about 0.01 in. in length, migrate at night and hence are known as *F. nocturna*.

The lymphatic obstruction produces marked œdema of the legs, scrotum, penis or vulva. The affected tissues are pure white in colour, hard, and will not pit on pressure. Hypertrophy follows in due course, leading to gross deformities, and trophic changes become evident in the overlying skin. Occasionally sepsis supervenes, usually with fatal results. In such cases one meets the rare conditions of chylous ascites, chyluria and chylous hydrocele.

The parasite is best attacked by intravenous antimony tartrate. The œdema may be to some extent controlled by strapping, require partial excision followed by skin grafting, necessitate a lymphangioplasty or Kondoleon's operation (removal of a strip of deep fascia down the whole length of either side of the leg to allow anastomosis

between superficial and deep lymphatic channels), or in bad cases call for amputation (Chap. XVI).

### GUINEA WORM

It is the female of the species (*Dracunculus medinensis*) which is responsible for the clinical picture. The worm reaches the human being by being swallowed in polluted water. It is yellowish white in colour and when fully grown may be as long as 18 in. About a year after ingestion it burrows its way to the surface, usually in the feet or legs, for the purpose of shedding its eggs once more. Its arrival at the surface is heralded by the appearance of a painful red swelling which ultimately breaks down, leaving an ulcer from the centre of which eggs are discharged.

It is only at this stage that the worm can be captured. Bathing in very cold water will make it visible, and it is then very slowly withdrawn—a process that may take over a week.

### AMÆBIC INFECTIONS

The only one of importance in man is that of the *Entamoeba histolytica*, the cause of amœbic dysentery. The typical lesion in this disease is an ulcerative colitis (see Chap. XXIX). The possible sequelæ of intestinal strictures and obstruction should be noted. An important secondary site of infection is the liver (or, more rarely, lung and kidney), where the so-called tropical abscess develops (see Chap. XXXIII).

Emetine (gr. i daily) subcutaneously is specific and most efficacious in the earlier stages.

### INSECT INFECTIONS

CHIGGÈ is the name given to a condition, chiefly affecting the toes and scrotum of children, in tropical climes, due to the irritation of the eggs of sand-fleas.

A. E. PORRITT.

R. M. HANDFIELD-JONES.

## CHAPTER V

### VENEREAL DISEASES

#### GONORRHOEA

**G**ONORRHOEA is a contagious disease usually limited to the urinary and genital organs, the causative organism being a specific diplococcus, first demonstrated by Neisser in 1879. The gonococcus is almost invariably diplococcal, the cocci of each pair being flattened on their adjacent surfaces, which never appear to be in contact. Multiplication takes place simultaneously in each pair and a consequent tetrad form is often apparent. They are readily ingested by polymorphonuclear leucocytes, and are usually found to be intracellular in stained smears. They stain with most basic aniline dyes, are gram-negative and can be cultivated on suitable media.

Transmission of the disease among adults is by venereal means, though in women an infected towel or lavatory seat may very rarely be responsible.

#### GONORRHOEA IN THE MALE

In two to ten days after an infected coitus, the patient is aware of an irritation at the end of the penis and a little discomfort on micturition. Inspection shows the meatus to be somewhat inflamed and a little sero-purulent discharge can be expressed. In the course of a day or two this discharge becomes thicker and more abundant, and as the inflammatory process spreads backward into the posterior urethra, local irritation causes frequency of micturition and painful erections of the penis, known as chordee.

After about ten days the acute inflammation begins to subside, the pain and discomfort passing off and the discharge, though persisting, becoming thinner and less abundant. The discharge finally disappears at any time between three and ten weeks, or a small drop of mucus may continue to be seen on rising in the morning. As long as there is the slightest visible discharge, it is certain that the patient is not cured, and even when this is absent, there is always the possibility that gonococcal colonisation is continuing in the urethra or its adnexa.

In order that the nature and possibilities of gonorrhoea in the male be appreciated, the anatomy of the genital organs must be borne in mind. Examination of the mucous membrane of the anterior urethra, *i.e.*, that part in front of the anterior layer of the triangular ligament, reveals the openings of numerous mucous glands. The larger ones are known as Littre's glands, while in the floor of the bulbous portion of

the anterior urethra are the openings of the two ducts of Cowper's glands. The posterior urethra contains a few rudimentary Littre's glands, the verumontanum with its prostatic utricle, and on each side of it the openings of tubular glands of the prostate and of the common ejaculatory ducts leading from the seminal vesicles.

All these structures are liable to invasion by the gonococcus, and once it has taken up its abode in such inaccessible situations, it proves difficult to dislodge. It is fortunate that nature plays a predominant part in the treatment of gonorrhœa, and provided that free drainage is effected, a large number of cases proceed to spontaneous cure without serious complications. The gonococcus in addition to infecting these glands, possesses the power of penetrating the unbroken mucous membrane and colonising in the submucous tissue.

**Methods of Diagnosis.**—Urethral discharges are not always gonococcal; urethral calculi, *bacillus coli* infections, chemical prophylactics and contraceptives, trichomonas infection, sugar-laden urine and oxalate crystals and the use of too strong antiseptic irrigations are capable of setting up a purulent urethritis. In the acute stage of gonorrhœa a thin smear of the discharge is stained by Gram's method, and large numbers of gram-negative diplococci lying within the pus cells will be seen. In subacute and chronic cases the pus may be scanty or non-existent and the gonococci few in number, and in these cases every part of the urogenital apparatus must be examined. The prostate and vesicles are palpated per rectum and prostatic secretion is expressed and collected on a slide and culture medium for examination; Cowper's glands, which are impalpable when normal, are sought for between the forefinger in the rectum and the thumb on the perineum; the epididymis and spermatic cord are examined for thickening, and finally, the urethra is inspected through the urethroscope. If any doubt remains, a complement fixation test should be carried out, as a positive result in the absence of recent vaccine treatment is strongly indicative of the presence of the gonococcus.

**Treatment** in all stages of the disease is governed by three principles, viz.: (1) free drainage of all infected parts must be ensured; (2) the resistance of all tissues, both local and general, to the gonococcus is to be encouraged; (3) the patient must be prevented from doing anything which will interfere with the natural cure; and (4) chemotherapy.

The patient is instructed to eschew all alcoholic drinks and highly-spiced foods and to drink large amounts of barley water and other bland fluids, which will produce a copious flow of non-irritating urine. In the more severe cases, it is desirable that the patient should be in bed during the first week of the acute symptoms, but only too often this is impracticable.

**CHEMOTHERAPY.**—During the last few years very extensive trials have been made with the various sulphonamide drugs many of which have a strongly bacteriostatic action on the growth of the gonococcus, and it can now be fairly confidently stated that, provided these drugs are used intelligently and in adequate dosage, in the majority of cases of uncomplicated gonorrhœa the infection can be rapidly and

permanently eliminated. In order however to achieve such results, a knowledge of the potentialities of the various drugs is necessary, and close supervision, a prolonged period of observation and particularly stringent tests of cure are essential.

For the out-patient sulphapyridine (M & B 693) is the drug of choice and its administration can be commenced as soon as a bacteriological diagnosis has been made. In order to achieve an adequate concentration in the blood stream the initial dose should be high and medication should always be carried out six-hourly day and night during the first four days of treatment. The tablets should be crushed before they are taken and washed down with a draught of water in order to hasten their passage through the stomach. In the vast majority of cases urethral irrigation is unnecessary, adequate drainage being maintained by an increased fluid intake with a consequent overproduction of urine. The following course of treatment is recommended for an otherwise healthy male of average weight. First day, 2 gm. at once, thereafter 1 gm. six-hourly; second to fourth days, 1 gm. six-hourly; fifth to seventh days, 1 gm. eight-hourly; total dosage, 26 gm.

Seven days after the end of treatment, provided that there is no discharge and the urine is clear, a specimen of the prostatic secretion is obtained by massage per rectum and microscoped for pus cells and organisms. If this is normal the patient is examined weekly for four weeks when, if there has been no recurrence of discharge and the urine remains clear and free from "threads," tests of cure can be commenced.

Sulphathiazole is less toxic and possibly more effective than sulphapyridine, but to obtain good results it must be given at least four-hourly night and day, as it is both absorbed and excreted more rapidly than the latter drug and it is consequently more difficult to maintain an adequate blood concentration. It also has a decided tendency to crystallise in the urine and a daily fluid intake of at least 5 pints is necessary during its administration. A total dosage of 26 gm. of the drug is given as follows: first day, 2 gm. at once thereafter 1 gm. four-hourly; second to fourth days, 1 gm. four-hourly.

Sulphanilamide is not as strongly bacteriostatic to the gonococcus as sulphapyridine and sulphathiazole; it is however a cheap drug and procurable everywhere, and provided that its administration is delayed until the discharge has persisted for at least eight days and the natural immune processes are "under way" a high proportion of permanent cures will follow its use. The dosage is the same as for sulphapyridine but a rather longer period of administration is necessary, a convenient course consisting of a total of 35 gm. given over a period of ten days.

It is important to remember that whichever drug is used its effects should be apparent after forty-eight hours' treatment, and if by this time the discharge has not considerably diminished and the urine commenced to clear, it should be stopped at once and the cause of the failure investigated.

Though a good deal of research remains to be done on the subject, initial failures are usually due to one of the following reasons:—



1. Drainage is inadequate ;
2. The patient's immunity response to the infection is subnormal ;
3. The drug is not being absorbed in adequate amounts from the intestinal tract ; or
4. The infecting organism is sulphonamide resistant, due to previous inadequate dosage which has accustomed it to the drug or to infection with a " drug fast " strain of gonococcus.

A combination of the first two reasons will often explain the failure, and irrigation of the anterior urethra with permanganate of potash (1 : 10,000) will assist drainage, so that a second course of chemotherapy in not less than twenty one days' time will effect a cure. During this time bi-weekly injections of gonococcal vaccines may be given and the prostate very gently palpated per rectum to ascertain its size and consistency. So long as these are within normal limits, no further treatment is necessary, but if the gland is found to be enlarged or tender, it should be very gently massaged immediately after micturition and the resulting secretion examined for pus cells and organisms. If these are present, gentle prostatic massage to assist drainage and to promote the blood supply to the gland may then be commenced provided that the second glass of urine is clear and that the tenderness is not too acute.



FIG. 12

Rash following sulphonypyridine therapy.

Minor toxic effects are common during sulphonamide chemotherapy. These include headache, nausea, giddiness, dyspnoea and skin rashes of an urticarial type ; they are usually most noticeable in constipated patients to whom purgatives should always be given. Urticarial rashes (Fig. 12) are sometimes severe and nearly always commence eight or nine days after the start of the treatment. If the drug has not already been stopped this should be done at once, and if the rash persists for more than forty-eight hours, a differential white cell count should be

carried out. Dangerous toxic effects are rare and among those that have been described are severe dermatitis, sulphæmoglobinaemia, aplastic anæmia, agranulocytosis and hæmaturia due to crystallisation of the drug in the renal tubules. An adequate fluid intake will almost certainly ensure against this last complication if the kidneys are healthy and the recommended dosage is not exceeded. In view of the possibility of these side effects these drugs should only be used under close medical supervision and facilities for blood counts should constantly be available. It appears to be quite unnecessary to forbid foods rich in sulphur such as eggs and onions though it is probably wiser to rely on purgatives other than sulphates.

**Complications** are due to a variety of causes, chief among which is misconduct on the part of the patient. They consist in infection of the urethral adnexa, or of the urogenital system and of metastatic blood-borne infections.

A. THOSE FROM THE ANTERIOR URETHRA.—1. *Periurethral abscess* results from the infection without free drainage of a Littre's gland which proceeds to suppuration. A very painful swelling, which later becomes fluctuant, appears on the lower surface of the penis. The abscess should be incised when definite fluctuation is felt, the cavity being lightly packed with paraffin and flavine gauze. Great care must be taken not to incise the urethra as, if this is done, a urinary fistula will undoubtedly follow.

2. *Chronic littritis* or persistent infection of Littre's glands is diagnosed by palpation of the urethra upon a straight metal bougie, when indurations varying in size from a millet seed to a split pea may be felt in the urethral wall. Treatment will consist in massage of the urethra upon a straight sound or progressive stretching with an anterior Kollman's dilator, either procedure being followed by urethral irrigation to wash away the infected matter expressed.

3. Either of *Cowper's glands* may be the seat of an abscess, which leads to acute pain in the perineum when the compressor muscle contracts at the end of micturition. The abscess may point in the perineum, where it should be incised. Chronic infection occasionally occurs in which the enlarged gland can be felt within the compressor urethræ muscle between a finger in the rectum and a thumb on the perineum. Bi-weekly massage followed by urethral irrigation will aid drainage and assist resolution. In persistent cases, however, the gland should be excised.

4. *Stricture of the urethra* is often a late sequel of submucous infection. Its clinical picture and treatment are described on p. 786.

B. THOSE FROM THE POSTERIOR URETHRA.—1. *Hyper-acute posterior urethritis* gives rise to painful frequency, strangury and often terminal hæmaturia. Treatment consists in rest in bed, frequent hot hip baths and the administration of an alkaline mixture containing tincture of hyoscyamus with potassium citrate. Irrigation must be suspended in all hyperacute conditions.

2. *Acute prostatitis* is a common complication and occasionally goes on to suppuration. Retention of urine and acute rectal pain are

present, and the hot, enlarged and excruciatingly tender prostate can be felt per rectum. The condition is treated by rest in bed, hot enemata and frequent hot baths, the patient being encouraged to micturate while in the bath. If the retention persists, the urethra is anæsthetised with 2 per cent. novocain, and a soft rubber catheter passed. The abscess usually bursts into the urethra, and as soon as the acute tenderness has subsided, drainage is assisted by gentle massage per rectum.

3. *Chronic prostatitis* is the commonest cause of long-standing infection. It may give rise to no symptoms, but many patients complain of vague unpleasant sensations in the perineum, and short threads of muco-pus are present in the morning urine. On rectal examination the gland is found to be enlarged in one or both lobes, to be tender to palpation, and may contain nodules in its substance. The prostatic secretion should be expressed by massage after irrigation of the urethra, and the presence of pus cells and organisms will confirm the diagnosis. Chronic prostatitis is frequently kept up by secondary pyogenic infection, and gonococci are rarely found in long-standing cases. Local treatment consists in bi-weekly massage of the gland to assist drainage, followed by immediate irrigation of the urethra to prevent its re-infection. If the urine is free from pus, a full-sized sound may be passed with advantage before massage to stretch the openings of the prostatic and ejaculatory ducts and so facilitate drainage. Diathermy to the prostate will greatly assist in clearing up the infection.

4. *Acute vesiculitis* is rare. It is characterised by painful blood-stained nocturnal emissions, the enlarged and painful vesicle being felt per rectum. Operative treatment may be necessary if drainage is unsatisfactory.

5. *Chronic vesiculitis* may follow an acute attack or may be chronic from the beginning, and is treated by bi-weekly massage of the vesicles per rectum. If this fails an attempt should be made to sterilise the vesicle by the injection into it through the vas of a 1 : 100 solution of Argylol (Belfield's operation).

6. *Acute epididymitis* results from the spread of the infection along the vas or its lymphatics. There is usually pain, tenderness and enlargement of the vas in the inguinal canal, and later the epididymis swells up and a condition of epididymo-orchitis follows. The pain is severe, and there are sometimes marked constitutional disturbances, while the urethral discharge often temporarily ceases. Treatment consists in rest in bed with local applications to the scrotum of glycerin and belladonna or unguentum hydrarg. co. All urethral irrigation must be discontinued. Resolution usually starts within ten days, but recurrences are not uncommon if irrigation is resumed at too early a date. Bilateral cases are apt to be followed by sterility.

7. *Acute cystitis and pyelitis* are very rare complications.

As a general rule sulphonamide therapy should be tried in all complicated cases of gonorrhœa provided that it is not apparent that drainage is impossible. Thus, it is useless to commence treatment before a prostatic abscess has burst or a periurethral one been incised.

The response is often immediate especially in metastatic infections such as iritis or arthritis.

**C. EXTRA-URETHRAL CONTACT INFECTIONS.**—*Gonorrhœal Ophthalmia* in newly born infants is a well-known condition and needs no description here. It is occasionally seen in adults in whom it is usually carried by the fingers to the eye. The first symptom is an acute conjunctivitis, which if not promptly treated spreads to the cornea and eventually a pan-ophthalmitis results. Treatment consists in, first, immediate sulphapyridine therapy and, second, protecting the other eye with a Buller's shield, washing out the eye with boric lotion, the instillation of atropine and painting the inside of the lids with 1 per cent. silver nitrate. The advice of an ophthalmic surgeon should be sought without delay.

*Gonorrhœal Proctitis* in males is the result of unnatural sexual relations. It is treated by irrigating the rectum with 1:6000 potassium permanganate, and is usually easily cured.

*Papillomata* of the glans penis or prepuce are not infrequently present during an attack of gonorrhœa but often occur in the absence of a gonococcal infection especially in patients of uncleanly habits. Consequently the terms venereal or gonorrhœal warts should never be used. The accompanying balanitis should first be treated with peroxide of hydrogen and when this has been controlled the warty growths can be removed with the electric cautery.

**D. METASTATIC COMPLICATIONS.**—Infection of the blood is not uncommon and the organism has been cultivated from the blood in uncomplicated cases of urethritis. The parts most commonly attacked are the joints (knee, wrist, elbow), bursæ (subdeltoid), tendon sheaths (in the hand, peroneal) and fascial planes (the plantar ligaments of the sole of the foot). These conditions are characterised by the sudden onset of acute pain in the affected part, which often occurs when the patient is in bed at night. Signs of acute inflammation are generally present, but suppuration rarely follows. These processes are described in other sections of this book. The treatment consists in eradicating the primary focus of infection, which is usually in the prostate, diathermy to which gives excellent results.

Metastatic iritis is occasionally seen and is accompanied by some conjunctivitis. Resolution often takes place if the primary focus is treated, but the immediate instillation of atropine is essential for the prevention of adhesions. Gonococcal septicæmia and pyæmia are very serious but very rare, while endocarditis is still more rare and is invariably fatal.

Subacute and chronic metastatic infections are often resistant to chemotherapy and focal treatment and in these cases artificial fever therapy will frequently achieve a cure. The body temperature is raised either by the intravenous injection of T.A.B. vaccine (initial dose 25 millions) or by means of the Kettering hypertherm. This is an insulated air-conditioned cabinet in which the patient is placed and in which the dry-bulb temperature and the relative humidity are thermostatically controlled, the patient's body temperature being continually ascertained from an external indicator connected with a

rectal thermometer. A complete systemic examination should always be carried out before these methods of treatment are embarked upon.

**The Use of Vaccines.**—Gonococcal vaccines are useful especially in the subacute and chronic stages, when the condition appears to be stationary, and their judicious use at this time will often turn the scale towards cure. They are particularly useful in metastatic infections as well as in long-standing chronic cases which have resisted chemotherapy. In such cases an autogenous vaccine, including any secondary organisms that may be present, is a valuable adjunct to local treatment. The writer is opposed to vaccine therapy in the early acute stages, believing that it is better to omit it for the first two weeks of an acute infection.

**Tests of Cure** should always be rigorous and spread over a period of three months. They include a thorough examination of the urethral adnexa and the prostatic fluid must in every case be subjected to microscopic examination. A full-sized bougie (Clutton's 20/24) is passed and the urethra carefully palpated upon it. An anterior urethroscopy should be performed. If no vaccine treatment has been given for two months, the complement fixation test may be done, but often never becomes positive in sulphapyridine treated cases. If all these investigations fail to reveal any trace of infection, a provocative injection of vaccine (300 million (4.C.) is given and the patient is encouraged to drink alcoholic liquor. Any recurrence of symptoms, the reappearance of threads in the urine, or of pus cells and organisms in the prostatic secretion, indicate that the disease is not cured.



FIG. 13

Keratoderma blenorrhagica.

**Keratoderma Blenorrhagica.**—The so-called gonococcal hyperkeratosis is rare, and though it always accompanies a urethritis with metastatic complications, evidence of gonococcal infection is sometimes not forthcoming. The condition, which is found in men only, consists of a vesicular eruption in which the walls of the vesicles become keratinised. The resulting crusts eventually separate leaving a red, moist area of skin. The condition is most often confined to the toes and soles of the feet (Fig. 13), though it is very occasionally seen on the legs, penis, hands and trunk. The presence of the lesion indicates a poor immunity response to the infection and is an indication for hyperthermic treatment. No local treatment is necessary except for strict cleanliness of the affected parts.

**Non-gonococcal Urethritis.**—About 30 per cent. of all cases of urethritis are non-gonococcal, though often venereal, in origin and on this account it is essential that all urethral discharges be subjected to a careful microscopic examination before treatment is commenced. The causes of this condition are "legion" and may be divided into two

main classes, chemical and infective. Chemical prophylactics instilled into the urethra, contraceptive ointments and pessaries used by the female, glycosuria and oxaluria are examples of the former. They are usually rapidly cured by removal of the cause and by an increased fluid intake which will produce a flow of bland non-irritating urine. Primary non-gonococcal infection of the urethra is commoner than was previously supposed, though many of the so-called cases are secondary to a feeding focus in the prostate or vesicles, the residuum of long-standing post-gonococcal secondary infection. Provided however that the history excludes a chemical etiology and that the prostate can be exonerated, bacteriological examination of the discharge will sometimes reveal the causative organism, though only too often the flora will be so varied that it is impossible to determine which variety is to blame. Coliform organisms, staphylococci, streptococci of the *fæcalis* type and diphtheroids are usual and occasionally the *trichomona vaginalis*, a protozoon well known as a cause of vaginitis in women, may be present. In cases where coliform organisms predominate in the smear, a midstream specimen of the urine should always be cultured, as a urethral discharge is not infrequently seen in pyelitis or cystitis due to this group. Though pure coliform infections react well to sulphonamides, those due to other bacteria are often unaffected by any form of chemotherapy and are best treated by daily irrigation of the urethra with a warm solution of oxycyanide of mercury (1 : 10,000) which may be followed by an instillation of a freshly prepared 5 per cent. solution of protargol, the latter being retained in the anterior urethra for five minutes.

It must be remembered that metastatic manifestations such as arthritis, fasciitis and iritis can be caused by a non-gonococcal urethritis or prostatitis and a diagnosis of gonorrhœal infection must never be made without bacteriological or serological evidence.

*Trichomonas* infestation of the urethra is unaffected by sulphonamides or by local treatment but is often cured by making the urine strongly alkaline or when the prostate is infested by diathermy.

### GONORRHOEA IN THE FEMALE

In women the gonococcus usually attacks both the urethra and the cervix uteri, though in very rare cases the infection may be limited to one of these sites. The early symptoms tend to be much less severe than in the male, there often being no pain on micturition and the patient regards the discharge as an attack of leucorrhœa (the "whites"). Hyper-acute forms are known in which the discharge is profuse and the vulva is œdematous and inflamed.

**Treatment.**—Rest is an important part of the treatment in acute gonorrhœa in women and whenever possible the patient should be kept in bed for the first week. Local treatment should be confined to a daily vaginal douche with a weak solution of bicarbonate of soda or permanganate of potash, caustic preparations or strong antiseptic solutions during the acute stage being both futile and dangerous.

**CHEMOTHERAPY.**—Sulphapyridine and sulphathiazole give as equally

good results in female as in male cases, and the gonococci quickly and permanently disappear from the urethral and cervical secretions. Often, however, a resistant infection of the cervix with secondary organisms, which may need active and prolonged local treatment, persists. This is the result of the inflamed endocervix becoming infected with vaginal organisms which are not affected by the sulphonamides and which normally do not have the power of penetrating unbroken mucous membrane and it is good practice to sterilise the vagina by insufflation with stovarsol powder if the cervical inflammation does not rapidly clear up. A daily vaginal douche followed by a thorough dry swabbing of the vagina should always precede each insufflation as otherwise large enough quantities of arsenic may be absorbed as to cause an arsenical dermatitis.

**Complications.**—Persistent urethritis will often be found to be due to an infection of Skene's tubules. These are two small blind ducts opening on to the floor of the urethra just within the meatus. They are best treated by injecting into them, through a blunt hypodermic needle, a few drops of a 5 per cent. silver nitrate solution, or by destroying them with the electric cautery.

**BARTHOLIN ABSCESS.**—Bartholin's glands are often infected and suppuration commonly occurs. Treatment consists in aspiration or incision. Chronic Bartholinitis is best dealt with by complete excision.

**ENDOMETRITIS.**—Infection may spread through the internal os resulting in an inflammation of the endometrium. The condition causes the cervical discharge to be suppressed and the uterus becomes enlarged and tender. The patient is put to bed in Fowler's position and hot water bottles applied to the abdomen. When the condition has become less acute, a small rubber catheter is passed into the uterus and 2 c.c. of glycerin is injected. It must be done slowly, and care must be taken that the catheter does not fit too tightly into the cervical canal.

**ACUTE SALPINGITIS** is fully described in Chap. XXXIX.

**PROCTITIS** is a common complication, the rectum being infected by the vaginal discharge. A warty hypertrophy of the vulva may also be seen (Fig. 14), but gonorrhœa must never be diagnosed on this alone, as it may be due to other infections.

The metastatic complications are similar to those that occur in men, but joint affections seem to be less common in women.

**Test of Cure** is difficult to establish in women. The complement fixation test should remain negative over a period of at least six



FIG. 14

Specimen illustrating the great warty overgrowth of the vulva in a case of chronic gonorrhœa.



months, and a monthly microscopical and cultural examination of both the urethral and the cervical secretion must show no gonococci during this period. The specimens should be taken just after the menstrual period, a provocative injection of vaccine being given a few days before the final ones are collected.

### VULVO-VAGINITIS IN LITTLE GIRLS

This condition, though sometimes caused by other organisms, is commonly the result of gonococcal infection. Epidemics are met with in schools and orphanages, where the disease is spread by infected towels and bed linen. In children living at home it can often be traced to one of the parents.

The infection is in most cases limited to the vulva and vagina, the urethra being occasionally and the cervix rarely attacked. It is notoriously intractable and relapses after apparent cure are common, but it does not usually persist after puberty.

Vulval irritation is soon followed by a vaginal discharge, which, serous at first, soon becomes purulent. Gonococci are abundant in the discharge and after a short time a secondary infection appears.

**Treatment** consists in rest, sitz baths and sulphonamides. Local treatment is unnecessary and always undesirable as it is distressing to the young patient and may easily initiate a habit of masturbation; even in resistant cases it should be confined to simple vaginal irrigation with a very weak antiseptic solution using a soft rubber catheter, which may be followed after the first week by the daily insertion of a small glycerin pessary further to encourage drainage. The infection frequently spreads to the rectum and no case should be discharged as cured until the rectal flora have been investigated and proved to be free from gonococci.

Every case must be rigidly isolated and knickers with no openings should be worn day and night to guard against the possibility of infection being conveyed to the eyes.

### SYPHILIS

Syphilis is a specific infectious disease due to inoculation with the *Treponema* (or *Spirochaeta*) *pallidum*, an organism protozoal in type, which was first demonstrated by Schaudin and Hoffman in 1905. It first appeared in Europe in 1493 and is believed to have been imported from the New World by Columbus' sailors on their return to Spain.

The *T. pallidum* is a minute and very fine spirally-shaped organism having six to fifteen spirals, each curve measuring  $1\ \mu$ . It is actively motile as can be readily seen in fresh preparations under dark ground illumination. It stains indifferently with aniline dyes. Although the treponema has been cultivated outside the body, nothing is known of its life history and every attempt at artificial immunisation has failed. The higher apes have been inoculated with the organism with exactly similar results to those in human beings.



**Transmission of the Disease.**—Syphilis is usually acquired during sexual intercourse, the treponemata in the infecting party being present either in open genital lesions, or as is probably more often the case in the male, in the ejaculated semen, and this may occur even during the incubation period. Extragenital infection (Figs. 15 and 16) sometimes occurs through kissing or using an infected drinking vessel, or inoculation may take place through a minute abrasion on the ungloved finger of an examining doctor or nurse. The organisms are considered able to penetrate an unbroken mucous membrane, and though probably they are unable to gain entry through sound skin, it must be remembered that a completely unbroken nail bed is a rarity, and that the commonest site of a digital chancre is at the junction of the finger-nail and the skin.



Fig. 15

Two primary chancres, one of the upper lip and the other at the right commissure.

Syphilis is a generalised systemic infection and becomes generalised long before the so-called primary lesion appears. Though many of the invading organisms are held up in the vicinity of the site of inoculation by the regional lymph glands, some pass almost at once into the blood stream and other body fluids. It has been shown that if a rabbit's testicle is removed forty-eight hours after inoculation with the organism, a week later its blood is infected to such an extent that 0.5 c.c. of it will transmit the disease.



Fig. 16

A primary chancre of the dorsum of the hand.

In spite of the fact that for clinical purposes the disease is usually divided into three stages, the tissue reaction to an invasion of treponemata is always substantially the same. The pathological process consists of a local proliferation of mononuclear cells, chiefly lymphocytes and plasma cells, with a later multiplication of fibroblasts and a consequent formation of fibrous tissue, nature's attempt to localise the infection. As a result of this reaction many of the organisms are destroyed, and in most cases partial or complete healing with

fibrosis takes place, in much the same manner as it does in tubercular lesions. In untreated cases, however, some treponemata invariably survive, and when the local tissue immunity has worn off, they commence again to multiply and to continue their work of

destruction. It appears probable that asymptomatic survival can occur in the testis or vesicles, which would explain how an apparently healthy man, sometimes years after his original infection, is able to transmit the disease to his wife and to beget syphilitic children.

**Immunity.**—Apart from a local tissue immunity it is certain that a considerable degree of general immunity is conferred by the disease, and it has been said that the one certain proof of the cure of syphilis is the acquisition of a fresh infection. This appears not to be strictly true, and cases of super-infection, though very rare, undoubtedly do occur.

### CLINICAL MANIFESTATIONS

**The Primary Lesion.**—In from anything from two to six weeks after infection there appears at the site of inoculation a small reddish



FIG. 17

A meatal chancre.

papule which usually proceeds to induration and ulceration. This lesion may be so inconspicuous as altogether to escape notice. Syphilis has been called the great mimic, and in no lesion is this more apparent than in the primary one. It may simulate an acne pustule, a patch of scabies, or a mechanical abrasion, and patients presenting a chancre of the frænum preputii are often under the impression that their condition is traumatic. The absolute necessity of submitting the exudate from every suspicious genital lesion to microscopic examination

cannot be too strongly emphasised. In most cases, however, the primary lesion sooner or later takes on the form of the typical *Hunterian chancre*. This consists of a small area of induration in the skin or mucous membrane which soon ulcerates. The resulting ulcer is hard and painless to the touch, its edge is built up and slopes gradually towards its base, which oozes serum but does not bleed readily. This serous exudate will be found to contain many *treponemata*. At the same time the regional lymph glands commence to enlarge. The enlargement is discrete and painless, and there is no tenderness on palpation. When the infection is acquired in a venereal manner the usual sites for the primary lesion in the male are the mucous surface of the prepuce, the glans penis, and more rarely the meatus (Fig. 17). Occasionally the chancre is situated intra-urethrally, in which case a slight serous discharge will appear at the meatus and a painless intra-urethral induration will be felt. The fact that a considerable proportion of cases of stricture of the anterior urethra show a positive Wassermann reaction suggests that their condition is possibly due to the healing with fibrosis of an intra-urethral chancre. The primary lesion also sometimes appears on the shaft of the penis (Fig. 18),

the scrotum, the lower abdominal wall and, as the result of sodomy, at the anal orifice or even inside the rectum.

In the female the usual sites are the anterior and posterior fourchettes, the clitoris, the labia minora and majora, and far more commonly than was formerly thought the cervix uteri. This will explain how so many female syphilitics are able to affirm honestly that they have never noticed "anything wrong below." Even when the primary cervical lesion is viewed through a speculum, it may easily be mistaken for an erosion, and it is always advisable, when there is any likelihood of syphilitic infection having taken place, to palpate the cervix between the two examining fingers for any signs of induration, and to submit the exudate to microscopic examination.

The primary lesion whether genital or extra-genital is usually single, but may sometimes be multiple. This is sometimes the case where two skin or mucous surfaces are in apposition, such as the mucous surface of the prepuce, where another lesion may be present on the contiguous portion of the glans penis, or in the female, on the opposite labium owing to constant friction against the original chancre.

Provided that infection of the primary sore with secondary organisms is not severe, healing will take place within a few weeks and if, as is often the case, there has not been much tissue destruction, little if any scarring will remain. Noticeable enlargement of the regional lymph glands is usual, though by no means invariable, and in females, where the primary lesion may be cervical, the adenitis will be intra-abdominal and not apparent.

**The Secondary Manifestations.**—Between two and four months after inoculation the generalised infection, which has actually existed from the commencement, begins to show itself. Constitutional disturbances, such as headache and slight pyrexia, are common but by no means invariable, and though one patient may complain of being "off colour," another will state that he feels fit enough. Some slight anæmia is nearly always present, and there is usually a deficiency of both red cells and hæmoglobin in the blood.

**SKIN LESIONS.**—It must again be remembered that syphilis is the great mimic and in the skin lesions of this stage it lives up to its reputation. The earliest secondary lesion takes the form of a macular blush that is usually generalised over the whole body. This blush may be so faint that it is not discernible at all on the more exposed parts, where it is liable to be masked by sunburn, and even elsewhere

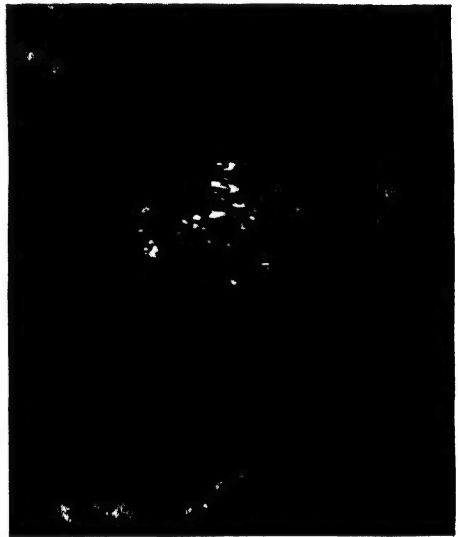


FIG. 18

A chancre upon the shaft of the penis.

it may be so faint that it is not visible by yellow artificial light. Sometimes this rash may fade away after a few weeks, and there may be an end to the secondary manifestations. Usually, however, the colour of the macules deepens to reddish-brown and a typical raw-bacon-coloured rash develops. This may be accompanied or succeeded by a papular eruption. The papules take the form of conical or lenticular raised patches varying in size from that of a pin's head to that of a florin; they are reddish-brown in colour and usually develop at the mouth of a sweat gland. A few days after their appearance they tend to become tipped with small scales, which, when detached, leave a shiny surface underneath. Treponemata can usually be demonstrated in the serous exudate from the larger papules. In moist situations, such as around the anus or vulva, the papules tend to be larger, raw and scaleless owing to the rapid destruction of the already devitalised epithelium. Sometimes in these places the sodden epithelium becomes heaped up, and flat wart-like excrescences develop. These are known as *condylomata*, and treponemata are always present in their exudate in large numbers. Occasionally they may give rise to a good deal of irritation through their becoming infected with secondary organisms and, when situated around the anus, they are not infrequently mistaken by the patient for piles.

In the undernourished and anæmic patient from whom treatment has been withheld there may appear a pustular type of papule which becomes covered by a crusty scab. Tissue destruction proceeds under the crust, which may become raised in characteristic concentric whorls. The fully developed lesion resembles a brown limpet shell adhering to the surface of the skin, a condition known as *rupia*.

The appendages of the skin are often affected in this stage of the disease or later, and the hair may fall out and the finger and toe nails become brittle and fissured. The alopecia of syphilis is distinguished from baldness due to other causes by its characteristic moth-eaten appearance in marked contrast with the smooth, clean-cut patches of alopecia areata.

It should be noted that as a general rule the cutaneous manifestations of syphilis do not give rise to any irritation unless they become infected with secondary organisms. In patients of uncleanly habits this is sometimes the case, and the combination of a macular or papular syphilide with scabies is not uncommon.

**MALIGNANT SYPHILIS.**—This rare condition is characterised by severe ulceration of the secondary cutaneous lesions, the papules rapidly becoming pustular and eventually breaking down to form a greenish slough. The patient is gravely ill, quickly becomes cachectic and may die, often as the result of orthodox treatment, which he is too weak to tolerate. This condition is probably due to a complete lack of antibody formation, and cases are on record in which the Wassermann reaction was negative, although treponemata were present in the tissues.

**MUCOUS MEMBRANES.**—The mucous membranes of the mouth and throat are usually affected about the same time, or rather earlier, than is the skin. The first manifestation in the mouth often takes the

form of a marked erythema of the soft palate which stands out in marked contrast to the paler hard palate. Later, small shallow ulcers may appear on the tongue, buccal mucous membrane, fauces and tonsils. These ulcers, known as *mucous patches* are, in fact, papular lesions which ulcerate almost immediately owing to their warm, moist site. In the throat they tend to run together and to become serpiginous, and they are often covered with a mucoid exudate. From their appearance they are popularly known as *snail track ulcers*. Like most other syphilitic lesions this ulceration gives rise to little or no discomfort, and often a very considerable amount of tissue destruction will have taken place before the patient becomes aware of his condition. Mucous patches are also met with on the mucous membrane of the vagina, the glans penis, and the mucous surface of the prepuce. As in the well-developed skin papules, *treponemata* are usually present in large numbers in these lesions.

**EYE.**—Iritis in varying degrees of severity is not an uncommon manifestation of the disease in the later secondary stage. The patient usually complains of ocular pain and photophobia, and occasionally there is some dimness of vision. The condition by itself is hard to distinguish from iritis due to other causes, but diagnosis is not difficult, as other signs of secondary syphilis are almost invariably present.

**LYMPH GLANDS.**—These tend to enlarge slightly all over the body. This enlargement is rarely great but it may persist for months or years. A convenient position in which it may be felt is in the epitrochlear gland of the elbow, but enlargement of this gland is no more pathognomonic of the disease than that of any other. A feature of this secondary adenitis is the complete absence of pain and tenderness.

**BONES.**—A slight transient periostitis of the long bones is occasionally present and pain is sometimes felt in the shin bones when in bed at night, owing to the increased congestion of the part at that time.

**CENTRAL NERVOUS SYSTEM.**—Definite nervous symptoms seldom appear at this stage, though there is no doubt that infection of the central nervous system takes place in many cases of secondary syphilis. This fact has been proved by animal inoculation of the C.S.F. taken from these cases. Neuralgic pains in the head are fairly common, and it is possible that these might be due to a mild inflammatory oedema of the brain tissue.

**The Tertiary Manifestations.**—There is no hard and fast rule governing the time of appearance of the tertiary manifestations of syphilis. Occasionally they follow close in the wake of the secondary lesions and sometimes they are delayed for years or even decades. The habitat and mode of existence of the *treponemata* in the interim are not known.

One of the best-known and often an early manifestation of tertiary syphilis appears on the skin and is known as the **nodular cutaneous syphilide**. The lesion consists of a curved line of intradermal nodules, dusky red in colour and usually covered with scales or crusts. The curved line tends to meet itself, forming a rough circle of a size varying from that of half a crown to a dinner plate. Some patchy

superficial ulceration is usually present and in parts of the lesion spontaneous healing is seen to be taking place with the formation of scar tissue. If untreated the condition extends, a well-advanced lesion taking on either a concentric spiral or an S-shaped formation. The lesion is occasionally confused with lupus vulgaris, but the differential diagnosis is comparatively easy, as the syphilitic process advances much more rapidly than does the tubercular one.

Syphilis in its tertiary stage is liable to attack any part of the body, and whichever organ is chosen, the tissue reaction to the specific toxin is essentially the same. This consists in an infiltration of mononuclear cells around the arterioles supplying the part attacked, a periarteritis and, later, an obstructive endarteritis of these vessels, resulting in a consequent necrosis of the tissues supplied by them. An increase of fibroblasts in the area results in a fibrosis, which may partially or even totally wall off the infection. The resulting lesion surrounded by its fibrous capsule is known as a **gumma**. When this is near the surface, the skin or mucous membrane is soon involved and ulceration takes place. The ulcer is usually fairly typical; it is punched out, deep and roughly circular, its base being occupied by the necrotic material from the centre of the gumma. Later this "wash leather" slough separates, healthy granulations appear underneath and healing takes place. It will be understood now that the nodular cutaneous syphilide is in fact a succession of small superficial gummata, some of which are breaking down while others are in the healing stage.

Discrete subcutaneous gummata may occur anywhere and are commonly seen on the leg, where, after ulceration has taken place, they must be distinguished from innocent varicose ulcers. This is usually not difficult, as the syphilitic ulcer, in addition to its characteristic punched-out appearance, is in most cases situated in the region of the knee-joint, whereas the varicose ulcer is more likely to appear in the vicinity of the internal malleolus. Nevertheless, in all cases of indolent varicose ulceration a Wassermann reaction should be carried out, as the two conditions may exist simultaneously.

Visceral gummata are not common but may occur in any of the abdominal or thoracic viscera. Cerebral gummata are also sometimes met with, but usually take their origin in the meninges rather than in the actual brain substance.

It is outside the scope of this section to describe in detail the syphilitic process in every part of the body. Let it be enough to say that any comparatively painless tumour of obscure origin is possibly gummatous, and syphilitic infection should always be excluded.

Though discrete gummata are sometimes met with in the bones, testes, meninges and even in the nervous and cardiovascular systems, **diffuse syphilitic infiltration** is the usual process. Here the toxins appear to be "fighting in extended order" rather than in the "mass formation" adopted in the discrete gumma. Consequently, necrosis is not so much a feature of this form of the disease, and a diffuse tissue destruction is quickly followed by fibrosis.

Syphilis of the heart, blood vessels, and the central nervous system is dealt with at length in all textbooks of medicine. As has already

been mentioned the process is usually one of diffuse syphilitic infiltration in cardio-vascular syphilis, and the vasa vasorum of the great vessels are often the first structures to be attacked. These tend to become obliterated and the elastic tissue of the tunica media is replaced by fibrous tissue, which is unable to withstand the intra-arterial pressure; dilatation takes place, and an **aneurysm** results. In syphilitic valvular disease of the heart, incompetence is usually caused by a replacement of the elastic tissue with unyielding fibrous tissue, but occasionally small gummata may form in the substance of the valves.

General paralysis of the insane and tabes dorsalis are both caused by a diffuse syphilitic infiltration of, in the former disease, the cerebral cortex and membranes, and in the latter the nerve fibres of the posterior columns of the cord.

Mention must here be made of **Charcot's disease** of the joints (Fig. 19). This condition is characterised by a rapid painless swelling of the affected joint, which is usually one which has been subjected to over-use. Effusion into the joint cavity takes place, the articular surfaces of the bones are eroded, and considerable grating on movement is felt, and later the joint becomes completely disorganised. In contrast with other forms of arthritis there is extreme mobility. It must be clearly understood that this condition, although almost always a sign of syphilis, is not due to infection of the joint itself, but to disease of the spinal cord, on account of which joint sensation is abolished and the normal reaction to undue stresses and strains is impaired. A similar condition is sometimes met with in cases of syringomyelia, which affects the joints of the upper extremity.

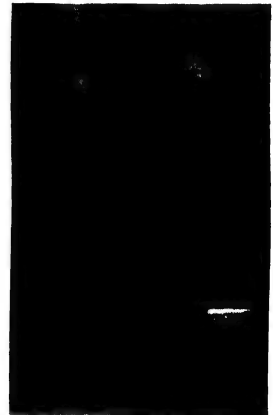


FIG. 19  
Bilateral Charcot's  
joints.

**Perforating ulcers** on the soles of the feet are trophic manifestations of syphilis of the spinal cord. They take the form of ulcerated callosities which refuse to heal, and are often the trouble for which the patient first seeks advice. The clinical signs of tabes dorsalis are almost invariably present in the above two conditions.

### CONGENITAL SYPHILIS

Children born of syphilitic parents are themselves liable to be infected with the disease. The infection either takes place by the maternal blood stream through the placenta, or the ovum or male semen may be infected *ab initio*. At first the mother usually aborts before full term, giving birth to a diseased and often macerated foetus. With successive pregnancies the foetal infection tends to become less severe, until at length a full-time child is born alive. This child may show signs of infection at birth, but often appears to be healthy enough, and it is usually not until it is three or four weeks old that its



infected condition becomes apparent. Frequently this typical sequence is not followed, and it is not uncommon for an apparently healthy child to be born between two obviously syphilitic ones.

Loss of weight, vomiting and irritability are usually the first signs of the disease, and are commonly accompanied by enlargement of the spleen. In many cases there is also an enlargement of the liver due to a pericellular cirrhosis. Often the child's face becomes characteristically putty-coloured and wrinkled, and resembles that of a very old man. Before long the skin and mucous lesions of secondary syphilis appear; these are liable to be exceptionally severe, and treponemata may be found in them in large numbers. A macular eruption is usually the first lesion noticed, which may be accompanied or followed by a papular one. Desquamation of the skin of the palms and soles is common, and is a useful diagnostic feature of the disease. On account of the exceptionally warm conditions in which most babies are kept, the skin lesions, especially the papules, tend to become moist and to ulcerate. True perianal and vulval condylomata may be present and ulceration of the mucous membranes of the nose, mouth and pharynx is common. When the nasal mucous membrane is attacked, the constant discharge gives rise to *snuffles*; later, the infection may spread to the nasal bones which may be partially destroyed. As a result the bridge of the nose falls in, producing what is perhaps the best-known stigma of congenital syphilis, the *saddle nose*.

Cracking of the skin or actual ulceration is common at the angles of the mouth, and when healing takes place, radiating scars or *rhagades* remain.

If the disease in this stage is left untreated, the infant will probably die, if not of the disease itself, from some intercurrent infection. It may however survive, in which case it will probably carry stigmata to the end of its life, and become liable to all the later manifestations of syphilis which occur in the acquired disease. Occasionally all signs of infection are delayed until later on in life; this is, however, the exception, and it will usually be found on going into the history with the mother that an account of some of the earlier lesions can be elicited. It must, though, always be remembered that, as in acquired syphilis, the secondary lesions may be so slight as altogether to escape observation.

The later manifestations of congenital syphilis do not usually occur until after the fifth year, but, as in the acquired disease, they occasionally appear much earlier. Though any of the conditions met with in acquired syphilis may be present, there do appear to be certain organs in the young for which the treponema has a special predilection, the bones, joints, teeth, eyes, ears and testes being particularly liable to attack.

**Bones and Joints.**—Osteochondritis of the epiphyses, especially of the lower limbs, and periostitis of the bones of the fingers, with a resulting fusiform dactylitis, are sometimes present in the early stages of the disease, and a periostitis of the lower end of the tibia can sometimes be detected by X-ray in quite young infants. Later, after the child has commenced to walk, and sometimes not until puberty,



periostitis of the tibia with a resulting thickening of the anterior surface of the bone produces the well-known curved or **sabre-shaped tibia**, which is sometimes mistaken for a rachitic manifestation.

The frontal and parietal bones of the skull are often attacked quite early in the disease, and considerable thickening may take place in the course of resolution. This thickening, which is usually symmetrical, forms the bosses known as **Parrot's nodes**, giving rise to the natiform or "hot-cross-bun" skull. Occasionally the bone is replaced by a thin parchment-like membrane; then the condition is known as **craniotabes**. **Craniotabes** is comparatively rare, and is considered by some authorities to be a manifestation of rickets. A similar condition certainly does occur in the absence of syphilitic infection.

Chronic bilateral synovitis of the knee joints is often present in children and young adults. Considerable effusion occurs and there is no pain. The enlargements are known as **Clutton's joints**.

**Teeth.**—The growth of the milk teeth is often affected adversely as a result of the disease, but no typical abnormality can be described. The permanent teeth are particularly liable to be modified both in size and shape, and are often smaller and more widely spaced than the normal, the first permanent molars tending to be ill developed and domed, when they are known as **Moon's teeth**. The classical dental stigmata of congenital syphilis are known as **Hutchinson's teeth**. In this condition the upper central permanent incisors are peg-shaped and may be notched. This notch is often absent or may be obliterated by wear in later life, but the unmistakable stigma consists in a short peg-shaped tooth considerably broader at its base than at its cutting edge.

**Eyes.**—The commonest ocular manifestation is *interstitial keratitis*. This usually appears between the ages of six and fifteen, though its onset may occasionally be delayed until as late as the twenty-first year. The patient first complains of some pain and photophobia and a ground glass opacity of the cornea develops. Later, leashes of small blood vessels are seen to grow into the opacity, and the characteristic "salmon patch" is produced. Unless vigorous treatment is initiated in the early stages, the prognosis is not good, and some interference with vision will often remain. The condition, though commencing in one eye, nearly always becomes bilateral.

*Choroiditis*, usually combined with *retinitis*, is a common manifestation of congenital syphilis in childhood and adolescence. Diminution of vision is progressive and retinoscopy reveals the characteristic black and white patches of pigment and exudate.

**Ears.**—Otitis is common in syphilitic infants, and is usually brought about by a spread along the Eustachian tube of the syphilitic process or of an accompanying pyogenic infection from nasal or pharyngeal lesions. Syphilitic otitis media also occasionally occurs between puberty and adolescence; the onset is then sudden, it is bilateral, and complete deafness quickly results.

**Testes.**—Syphilitic infiltration of the testis may occur at any age, though it is usually an early manifestation. It is by far the commonest cause of enlargement of the testis in infancy.

**Cardiovascular Congenital Syphilis** is rarely met with in childhood or adolescence, and when it occurs in later life it is often difficult to exclude the possibility of an acquired infection.

**Central Nervous System.**—Juvenile tabes dorsalis and congenital general paralysis are occasionally met with, and present much the same signs and symptoms as they do in the adult. The conditions are rarely noticed before puberty but the Wassermann reaction of the C.S.F. has often been found to be positive in quite young children.

**Third Generation Syphilis.**—It was formerly dogmatically held that syphilis could not be transmitted to the third generation, but recent observations seem to have proved that there are some cases where this does take place. They must, however, be comparatively rare, and the vast majority of congenital syphilitics do not appear to beget syphilitic children. In the investigation of a suspected case the difficulty of proving the absence of an acquired infection in the second generation will be appreciated.

### THE DIAGNOSIS OF SYPHILIS

As has already been pointed out, the early manifestations of syphilis may be so slight as to escape the patient's notice. An intra-urethral or sub-preputial chancre in the male may easily be overlooked, and in the female a cervical chancre will rarely give rise to symptoms. The secondary macular eruption may be so faint as to escape detection and, unless the patient is examined in strong daylight or by the light of a daylight lamp, may be missed by the examining surgeon. In all cases where there is any doubt, the diagnosis can always be confirmed by microscopic and serological tests, and so efficient are these that no diagnosis of syphilis should ever be made without the employment of one or both of them.

**The Primary Lesion.**—Unless strong antiseptics have already been applied by the patient, treponemata may always be readily found in the lesion. The ulcer should be rubbed with dry gauze or gently scarified until slight hæmorrhage takes place; as soon as clotting has commenced, a little of the serum is transferred to a slide, a cover-glass is applied, and the specimen is examined microscopically by the method of dark ground illumination, a special condenser and an exceptionally high-powered lamp being used; the treponemata can then be observed in the living state. In cases where the lesion is not readily accessible, one of the enlarged regional lymph glands may be punctured with a hypodermic needle and some of the gland fluid drawn up into a syringe; the fluid is then examined and the organisms can usually be demonstrated therein. This method of diagnosis should always be tried before subjecting a phimotic patient to the mutilating operation of a "dorsal slit-up" to expose a hidden chancre, as local sub-preputial medication can always be applied with a syringe as soon as a diagnosis has been made.

In the case of a suspected cervical lesion, the cervix should first be well cleaned with a dry swab, and if bleeding does not readily take place, it should be gently scarified. When clotting occurs, the serum can be conveyed to a slide by means of a sterile platinum loop.

Treponemata are invariably present in condylomata, mucous patches and the larger skin papules, and search should be made for them in these lesions, if the primary sore has healed before the patient presents himself for examination. In secondarily infected genital lesions care must be taken not to mistake *Spirochæta refringens* for the specific organism; this is a short, coarse spirochæte which, unlike the delicate, slow-moving treponema, moves with great rapidity across the microscopic field. Spirochætes resembling the treponema are often present in mouth lesions, but once the latter has been observed and carefully studied, there should be little difficulty about its subsequent recognition.

**The Wassermann Reaction.**—This test, which was introduced in 1906, depends upon the presence or absence of hæmolytic power in the serum under fixed known conditions. The test does not become positive until six to eight weeks have elapsed after the original infection; consequently, the early diagnosis of recently acquired syphilis depends upon the finding of the organism in the primary lesions before there has been time for the Wassermann to become positive.

For this test the blood should be collected from a vein in the bend of the elbow, or in an infant, using a Wright's capsule, by puncture of the skin covering the heel, congestion of the part having been effected by bandaging the limb in a downward direction. Great care should be taken that the collecting needle and syringe are free from spirit, otherwise some hæmolysis may take place before the test is carried out, and the result seriously vitiated. Other serological tests for syphilis are the flocculation tests of Kahn, Sachs-Georgi and Dreyer (Sigma test). These depend on the principle that when a cholesterinised extract of heart muscle is added to a heated syphilitic serum, flocculation occurs in the mixture; they are useful as confirmatory evidence, especially in cases where the Wassermann gives a doubtful result, but up to the present none of them has replaced the last-named test in general use.

In this country, where tropical diseases akin to syphilis do not prevail, a positive Wassermann reaction can be regarded as evidence of syphilitic infection. On the other hand, a negative reaction may be obtained in a syphilitic, and will mean one of three things, namely:—

1. That enough time has not elapsed since infection for the test to become positive.
2. That though infection is still present, treatment has caused the reaction to become temporarily negative.
3. Rarely that, though the patient is infected and has not been treated, his blood for some obscure reason has not developed sufficient of the syphilitic antibody, on the presence of which the test depends. It is possible that this occurs when the treponemata are walled off from the tissues within a dense fibrous capsule. Later, if this capsule is autolysed and the organisms are released into the body, the tissues will respond and the test will again become positive.

**Prophylaxis**—*Treponema pallidum* is an extremely delicate organism and, provided that thorough disinfection of the genitalia is carried out within two hours of a venereal exposure, the risk of infection will be minimised. After this time the organism has almost certainly "dug itself in" beyond the reach of the most powerful antiseptics, and prophylactic measures taken then, far from destroying the infection, may have the effect of delaying the appearance of the primary lesion, so that the disease will become well-established before it can be diagnosed and treatment commenced.

IMMEDIATE PROPHYLACTIC MEASURES in the male should include :—

1. Urination ;
2. Thorough washing of the parts with soap and water ;
3. Swabbing of the genitalia, especially the mucous surfaces, with 1 : 2000 perchloride of mercury ; and
4. Inunction of the parts with 33 per cent. calomel ointment, some of which should be squeezed out of a collapsible tube into the urinary meatus and gently massaged into the urethra.

A patient who has run a known venereal risk should be kept under observation for three months, Wassermann reactions being carried out during and at the end of that period.

The practice of giving an injection of one of the salvarsan substitutes as a prophylactic measure after a venereal risk cannot be too strongly condemned. If infection has taken place, it may have the effect of delaying the appearance of symptoms for a considerable time, and it can never be relied upon to destroy all the infecting organisms.

### THE TREATMENT OF SYPHILIS

Certain guiding principles should be followed by anyone who undertakes the treatment of syphilis. These are that :—

1. The earlier treatment is commenced, the better will be the result.
2. Treatment, once commenced, should be as strenuous as is consistent with safety. Half measures are dangerous, and recurrences are common in insufficiently treated cases.
3. Treatment and observation should be prolonged until one is satisfied by the strictest tests that the infection has been eradicated.
4. It should be impressed on the patient at the start that his treatment will take a long time, the danger of premature cessation should be pointed out to him, and he should be reassured of his safety so long as he carries out orders.

The drugs used in the modern treatment of syphilis are the salvarsan substitutes (which include neo-arsphenamine, sulpharsphenamine and silver-salvarsan), solutions and suspensions of bismuth and its salts,

mercurial preparations for intramuscular injection as well as those for oral administration, and potassium iodide. The last-named drug, unlike the others, has no treponemocidal action but aids the autolysis of necrotic substances and of newly-formed fibrous tissue, thus making the organisms more accessible to the other drugs.

**Salvarsan** or **arsphenamine** is an organic arsenical compound; it is rarely employed to-day, as its derivatives, neo-arsphenamine and sulpharsphenamine, are thought to be equally efficient, less toxic, and easier of administration. The usual procedure consists in the intravenous injection of a solution of neo-arsphenamine, or the intramuscular or deep subcutaneous injection of sulpharsphenamine, accompanied by the intramuscular injection of one of the recognised bismuth preparations. The initial dose of the salvarsan substitute will depend on the age, weight and condition of the patient, and in the adult is usually 0.3 grm. If this is well borne, the dose is increased at the next visit to 0.45 grm., and later to 0.6 grm. In heavy, healthy males as much as 0.75 grm. may be safely given, but in the treatment of females 0.6 grm. should never be exceeded. The average interval between doses should be about seven days, but in early infectious conditions, the first four or five doses may safely be given every four days, as by this means the patient is the more quickly rendered non-infectious. The treatment is conveniently divided into courses, though there is at present a lack of uniformity of opinion as to what should be the optimum length of a course. Harrison considers that not less than 5 grm. of the salvarsan substitute and the equivalent of 2 grm. of bismuth metal should be given over a period of not more than three months, and with this view the writer is in agreement. A month's rest from treatment should separate each course, and potassium iodide up to gr. xx t.d.s. should be given during this interval. At the end of this time a Wassermann reaction is carried out.

In deciding the amount of treatment necessary in a particular case, one is guided by the Wassermann reaction, but to guard against relapse, an absolute minimum of three of the courses described above should be given in primary and secondary cases, and usually more for later ones; this, in spite of the fact that the Wassermann reaction may be found to be negative at the end of the first course, or even at the commencement of it. A safe rule is always to give at least two courses after the Wassermann becomes negative. Relapses are common in insufficiently treated cases, and in recent years many neurosyphilitics are found to have been war-treated cases, whose treatment was curtailed owing to the exigencies of the service. After the cessation of treatment and the Wassermann is negative, the patient should be kept under observation for at least two years, and treatment should at once be recommenced should it again become positive. During the period of observation a full investigation of the cerebrospinal fluid should be carried out. Some workers do the final blood test a few days after the injection of a single dose of neo-arsphenamine, claiming that this will have the effect of intensifying the reaction should it be only doubtfully positive.

The scheme of a suggested course of treatment is tabulated on p. 78.

It will be noticed that bismuth is given weekly throughout the course irrespective of the varying intervals between the doses of the arsenicals.

Day.	Neo-arsphenamine or Sulpharsphenamine.	Bismuth Metal.
1st	0.3 gm.	0.2 gm.
8th	0.45 "	"
15th	0.45 "	"
22nd	...	"
29th	0.6 "	"
36th	0.6 "	"
43rd	...	"
50th	0.6 "	"
57th	0.6 "	"
64th	0.6 "	"
71st	...	"
78th	0.6 "	"
85th	0.6 "	"

In the above course it will be seen that 5.4 gm. of the salvarsan substitute and the equivalent of 2.6 gm. of bismuth metal are given over a period of eighty-five days. The course may be modified according to the weight and condition of the patient.

In the treatment of cardiovascular syphilis, it is safer always to employ the intramuscular or deep subcutaneous routes, and in severe cases the doses of the drugs should be small and be given at close intervals, a convenient course consisting of 0.15 gm. of sulpharsphenamine given bi-weekly for ten weeks, the accompanying dose of bismuth not exceeding the equivalent of 0.1 gm. of bismuth metal.

The intramuscular preparations of mercury have been generally superseded by those of bismuth, the latter having proved itself to be equally efficient and less toxic in its action.

In syphilis of the central nervous system strenuous treatment is particularly essential, the usual arsenical compounds appearing to have little effect on the course of the disease. Bismuth given in bi-weekly doses of 0.2 gm. accompanied by massive doses of iodides, preferably given intravenously, seems to give the best results.

Tryparsamide, a pentavalent arsenical compound having a high arsenical content, may be given in courses of ten weekly injections of 3 gm. each concurrently with the bismuth. This drug is often efficient in early cases of tabes, but is contraindicated if any visual disturbances are present or arise during the course of treatment and a close watch must be kept for signs of optic atrophy. Whichever drug is chosen, the intensive administration of iodides should always form part of the treatment.

General paralysis of the insane is always best treated by induced hyperpyrexia by malarial inoculation or a mechanical hypertherm, as at present this appears to be the only treatment which is followed by a remission of symptoms.

In congenital syphilis the procedure is on the same lines as in the acquired disease and the earlier manifestations yield well to treatment,

the Wassermann reaction becoming negative in due course. Though the later lesions, too, usually respond well, the Wassermann is apt to remain positive and treatment must be continued for a long period. Sulpharsphenamine given intramuscularly appears to give the best results, and is well tolerated in small doses even by very young infants, 0.025 grm. being a safe initial dose for a child a month old.

**Intolerance to Treatment.**—A small proportion of patients react unfavourably to treatment with the arsenical compounds. The reactions may be either immediate or delayed, the immediate type usually following an intravenous injection and taking the form of vomiting, rigors, syncope or vasomotor disturbances. These can usually be guarded against by seeing that the patient is not treated on a full stomach and that the injection is made sufficiently slowly. A severe immediate reaction is always a contra-indication to further intravenous treatment. Later reactions include loss of weight, albuminuria, jaundice, dermatitis, purpura and very rarely the fatal complication of hæmorrhagic encephalitis, and are actually symptoms of chronic arsenical poisoning; they are sometimes due to a failure of the excretory mechanism of the body, so that too high a concentration of the drug accumulates in the tissues. To guard against these manifestations the patient should be instructed to increase his intake of water, carbohydrates and proteins, and to abstain altogether from alcohol during treatment. The glycogenic content of the liver can be increased by the administration of a draught of a glucose solution immediately before treatment, and the patient may be instructed to eat a pennyworth of cheap boiled sweets on the day of the injection. Before each injection the urine should be tested for the presence of albumen, and the urobilinogen test should also be carried out.

Immediate vasomotor reactions are best treated by warmth, rest, and the injection of 1:1000 adrenalin, the usual cardiac stimulants being useful in severe cases of syncope. The treatment of the later toxic manifestations must be directed towards securing the rapid elimination of arsenic from the body. With this end in view, a large amount of water must be given by the mouth, frequent colonic lavage carried out, and where the presence of jaundice shows the liver to be affected, a fat-free diet is indicated. The intravenous injection of a solution of sodium thiosulphate and the intramuscular injection of liver extract appear favourably to influence the condition. In cases of generalised dermatitis every precaution must be taken against chill, as the heat-regulating mechanism being seriously interfered with owing to involvement of the sweat glands, pneumonia may become a serious complication.

Arsenicals are contraindicated for many months after a severe reaction, whether immediate or delayed, and when the patient recovers, treatment should be continued with bismuth (or mercury) and iodides only.

Bismuth, though contraindicated in renal disease, is a very safe drug, though, like mercury, it is apt to cause stomatitis, especially when the patient suffers from pyorrhœa, and if the mouth is found to be septic, a visit to the dentist should always be advised.



### THE PROGNOSIS OF SYPHILIS

Since the introduction of the organic arsenical compounds into the treatment of syphilis, the prognosis of the disease has improved beyond measure. It can now be stated with some certainty that an early case of syphilis energetically treated on the lines indicated is extremely unlikely to relapse, and that, in the great majority of cases, the infection is eradicated. At the same time, it should always be carefully explained to the patient on discharge that he is "cured" only so far as is known to modern medical science, and in all cases of tertiary syphilis the period of observation should be lifelong.

**MARRIAGE.**—No person should be permitted to marry until the Wassermann reaction of his blood and cerebrospinal fluid has remained negative for at least two years after the cessation of treatment. Women should always be treated throughout their pregnancies if signs of syphilis are present in their consorts, even though they themselves are not considered to be infected. It should always be remembered that the modern treatment of syphilis has only been in general use during the last twenty-five years, and that it is still not definitely known for how long a completely latent infection can endure.

### CHANCROID

**Chancroid, Soft Sore or Ulcus Molle.**—These names are sometimes loosely applied to any venereal sores which prove to be non-syphilitic, but it is more correct to limit them to the fairly typical infectious venereal condition, which follows contagion with Ducrey's streptobacillus. This organism is a short gram-negative bacillus which, like the streptococcus, tends to adopt a chain formation. On account of an invariable accompanying secondary infection it is not easily recognised in stained specimens and is extremely difficult to cultivate.

In anything from one to five days after infection a red papule, which quickly ulcerates, appears at the site of inoculation. These ulcers, which are frequently multiple, are characteristically painful, shallow, ragged-edged and, unlike the primary syphilitic lesions, have a tendency to spread. In debilitated patients and those of unclean habits the ulcers occasionally become phagedenic, often with considerable loss of tissue. The ulcers are accompanied or followed after healing has taken place by a painful and tender inguinal adenitis, which may be unilateral or bilateral. Suppuration generally occurs and the overlying skin becomes red and inflamed, the resulting swellings being known as "buboes."

**Diagnosis.**—Scrapings taken from the edge of the ulcer and stained by Gram's method will occasionally reveal the presence of Ducrey's bacillus, but more often than not the organisms cannot be found. In all cases repeated search should be made for the *T. pallidum*, and to exclude syphilis the Wassermann reaction must remain negative for eight weeks after the appearance of the ulcer.

**Treatment.**—The patient should be put to bed and the ulcers cleaned



with frequent applications of peroxide of hydrogen, followed by local application of sulphanilamide powder. In phimotic patients a syringe should be used and the preputial sac constantly cleansed. If the pain is severe, powdered aspirin is an excellent local anæsthetic.

Most striking results follow the intravenous injection of Dmelcos, a vaccine prepared in France from Ducrey's bacillus, the initial dose being 1 c.c. (225 million organisms). The injection, which is followed by some pyrexia and malaise, is repeated at three-day intervals, the dose being increased by 1 c.c. daily up to a maximum of 3 c.c. On this treatment the ulcers frequently heal within a fortnight and the adenitis may resolve without any further treatment being necessary. If Dmelcos is unobtainable, similar results will sometimes follow the intravenous injection of T.A.B. vaccine in doses of 25 to 75 million organisms.

In phimotic patients where the sore is sub-preputial, a brown foul smelling discharge suggests the possibility of phagedena. This is a gangrenous process, and the prepuce should be slit up without delay and the ulcer inspected. All sloughs must be cut away and, as in these cases the streptococcus is a common secondary invader, zinc peroxide cream should be applied and sulphanilamide given by mouth in doses of 2 grm. four times a day.

As soon as the bubo suppurates it should be aspirated, a wide-bored needle, to which is attached a 20 c.c. Record syringe, being passed through sound skin into the centre of the gland. After aspiration, the needle is left *in situ* and the cavity injected with a 1 : 20 solution of tincture of iodine equal in volume to the pus withdrawn. If the cavity refills the procedure is repeated daily for three days. After this the bubo should be freely incised, when it will be found that healing will occur more speedily than if incision has been resorted to at first.

It cannot be too strongly emphasised that in this country chancroid is a relatively uncommon disease, and that in all cases the possibility of a syphilitic infection should be rigorously excluded.

## LYMPHOGRANULOMA INGUINALE

This condition—the so-called climatic bubo—is not an uncommon disease in the tropics, but is extremely rare in this country, though recently a few authentic examples have been recorded. The causative organism or virus has not yet been isolated, and the incubation time is not known.

The primary lesion is not typical, though there is generally a history of a small genital abrasion which rapidly healed, and the patient usually first seeks advice on account of a comparatively painless enlargement of the inguinal glands. In women the condition sometimes remains undetected for a considerable time, as the adenitis occurs in the intra-abdominal glands, presumably following a primary lesion on the cervix. Stricture of the rectum from a periadenitis with adhesions may give rise to the first noticeable symptoms.

The adenitis may remain stationary for weeks or even months, but as a rule suppuration eventually takes place.

**Diagnosis.**—The aspirated gland fluid or pus does not contain spirochætes or other organisms, and the Wassermann reaction is negative and remains persistently so. Diagnosis is made by Frey's test. An injection of antigen (0.1 c.c.) prepared from pus from a bubo in a known case is made intradermally into the forearm. A positive reaction, which appears within forty-eight hours, is indicated by the appearance of a raised inflammatory area at the site of inoculation which persists for several days.

**Treatment.**—Very satisfactory results follow the use of "Fouadin," an antimony compound, combined with a course of sulphanilamide.

G. L. M. McELLIGOTT.

## CHAPTER VI

### TUMOURS AND CYSTS

#### TUMOURS

**A**S a result of intense and world-wide study, the nature of true tumour formation is well understood, but its real causative factor (or factors) remains unknown. In the present state of our knowledge, therefore, no exact definition can be formulated. The best available is: "A mass of cells, tissues or organs resembling those normally present in the body, but arranged atypically, which grow at the expense and independently of the organism without subserving any useful purpose therein." The term "tumour" is unfortunately used indiscriminately to include any abnormal swelling, but its use should be restricted solely to true neoplasms, and should not be applied to such processes as simple hypertrophy and inflammatory reactions.

#### ETIOLOGY

Although the essential causative factor is unknown, several facts of etiological importance are recognised.

1. **Age** incidence varies with different types of tumour, but generally speaking the carcinoma ages are between 35 and 65 years, the peak being reached in men at 55 and in women at 50. Sarcoma is not so commonly found in young people as was once thought, and its age incidence closely resembles that of carcinoma. A few rare congenital tumours, certain sarcomata and many teratomata are seen in childhood and adolescence.

2. **Sex**.—Malignant disease occurs more frequently in women than in men in the ratio of 3 : 2. This difference is largely accounted for by the high incidence in the breast and generative organs of the female. On the other hand, cancer of the tongue, buccal cavity, pharynx and larynx is rare in women.

3. **Heredity**.—There is no real evidence to show that heredity has any important etiological significance.

4. **Locality**.—No convincing statistical evidence is available to support the theory of "cancer houses" and "cancer districts," nor is there the slightest evidence that cancer is either infectious or contagious.

5. **Injury and Irritation**.—The history of a blow is quite frequently found in carcinoma of the breast and in teratoma of the testis, and it is possible that the injury may have provided the stimulus to new growth formation.

The clinical and experimental evidence in favour of chronic

irritation forms a more formidable contribution. It is established beyond doubt that long-standing chronic irritation does produce malignant disease. A few examples must suffice, viz., the association of gall-stones and carcinoma of the gall-bladder, scrotal cancer in sweeps, surface growths in paraffin workers, the kangri cancer of the abdominal wall in the natives of Kashmir, who carry their charcoal fires beneath their clothes, the cancer arising in old lupus scars, and finally the experimental production of cancer by the Imperial Cancer Research workers with tar-painting in animals.

### THEORIES OF TUMOUR FORMATION

These cannot be adequately dealt with in a textbook of surgery, and the reader is referred to works on pathology for a full description. The extrinsic theory postulates the existence of a parasite or virus introduced from without, which is responsible for the tumour formation, but no evidence has ever been brought forward to support this view. There are several intrinsic theories, amongst them being (1) the alteration of tissue tension by which the balance normally held between the epithelium and connective tissue of an organ is upset; (2) Cohnheim's theory of the persistence of embryonic cells in the body after birth; (3) theories of alterations and abnormalities of growth; and (4) the theory of heterotype mitosis, which implies that tumour cells are similar to reproductive cells in having half as many nuclear chromosomes as normal somatic cells. Lastly, Gye's work seems to combine the extrinsic and intrinsic theories, for he has described the existence of an ultra-microscopic virus which can be isolated from certain animal growths. This virus cannot, however, produce any effect unless combined with his "specific factor," a virus-free extract of the tumour cells. When this combination of virus and specific factor is injected into an animal of the same species, a malignant new growth develops. This work is not necessarily applicable to the human, and further corroboration is needed before it can be accepted. At the present time it must be acknowledged that the question of the origin of cancer has defied solution.

### STRUCTURE AND GROWTH OF TUMOURS

All tumours consist of two parts, the supporting connective tissue framework or stroma and the tumour cells proper. The relationship between the two varies considerably in different growths, for whereas in epithelial growths they are easily distinguishable, in the connective tissue growths the stroma may be indefinable. The cells in innocent growths are typical, *i.e.*, they resemble their parent cells so closely that their origin is never in doubt, but in malignant tumours the cells are often "atypical," *i.e.*, they differ from their parent cells and tend to revert to embryonic or immature forms, so that it may be difficult to identify the tissues from which they have arisen.

The stroma is derived from the connective tissue of the organ

from which the tumour is growing, and is the framework which carries the blood vessels and lymphatics which supply the tumour cells with nourishment and remove their products of metabolism. The stroma reaction may be so excessive as to strangle the tumour cells and bring about a natural cure, but on the other hand the tumour may outgrow its stroma, so that its central parts may be starved of blood and undergo degenerative changes.

### INNOCENCY AND MALIGNANCY

All tumours are divided into two main groups, the innocent and the malignant, and in the great majority of cases it is possible to say with confidence to which group a tumour belongs, although it is sometimes extremely difficult even for a pathologist to place a tumour in its proper class with certainty.

**An Innocent or Benign Tumour** increases in size by uniform growth throughout the whole mass, *i.e.*, by expansion, the surrounding tissues being compressed or pushed aside. It is enveloped in a true capsule of fibrous tissue derived from the tissues of the host by compression and tissue reaction. It is often multiple, does not recur after removal, and does not produce metastases.

**A Malignant Tumour** is almost invariably fatal unless removed or destroyed. It is not, however, this ultimate result which forms the criterion of malignancy, but rather certain definite properties which these tumours possess. These accepted signs are : (1) Constant and steady increase in size with varying rapidity in different cases ; (2) a tendency for the cells to become embryonic in type ; (3) the tumour extends its borders by an infiltration of the surrounding tissues, which are gradually destroyed or enveloped ; (4) involvement of the skin or mucous membrane leads to ulceration or fungation ; (5) metastases are formed in the lymph glands and viscera ; (6) the growth recurs locally after removal unless all its ramifications have been excised ; and (7) cachexia and anæmia occur as late manifestations.

### METHODS OF SPREAD OF MALIGNANT TUMOURS

A malignant growth spreads locally by "infiltration" and generally throughout the body by "dissemination."

**Local Infiltration** is the process by which the growth extends its borders and spreads into the surrounding tissues. It is the earliest sign of malignancy, because when overactive cells penetrate their limiting or basement membranes and enter the tissues beneath, then infiltration has begun and a malignant process has been established. Active growth in malignant tumours occurs chiefly at the periphery, and the surrounding tissues are invaded by columns or groups of cells, which work their way between muscle bundles and fat lobules into tissue spaces and into lymphatic and blood vessels. These tissues are not displaced and pushed aside, but are enveloped and destroyed by the advancing tumour cells (Fig. 20).

**Dissemination** is the process by which the tumour spreads beyond its site of origin and gives rise to secondary deposits or metastases in other tissues and other organs of the body. It is the general rule that these secondary growths correspond in appearance and behaviour to their parent tumours, but this is not always so. The metastasis may be more or less actively growing than the primary growth; it may completely dominate the clinical picture, though as a rule the primary is larger than any of its secondaries; a primary malignant melanoma may contain little pigment, whereas its secondary deposits may be jet black. Such examples may be multiplied, but usually the metastases breed true to type. Another feature of importance is

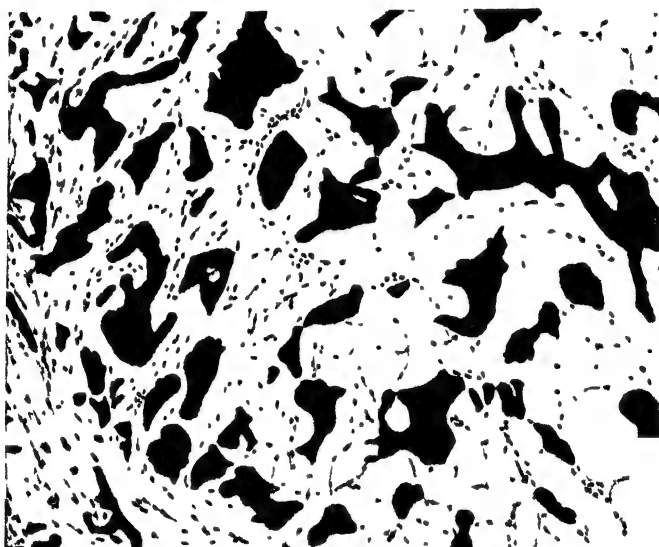


FIG. 20

Section from the growing margin of a carcinoma simplex of the breast. Groups of carcinoma cells are seen infiltrating fatty tissue. (*Kettle*)

the site in which the secondary deposits develop. All forms of carcinoma affect the lymph glands which drain the area, and growths in the area of the portal circulation tend to metastasise in the liver. Certain tissues appear to offer favourable conditions for the development of secondary deposits of given tumours, and they are therefore termed "tissues of predilection." The metastases, for example, of carcinomata of the breast, thyroid, prostate and kidney show a particular tendency to settle in bone, whereas the brain, pancreas, intestine, spleen and skeletal muscle are rarely the seat of secondary growths. Another important fact is the time at which secondary growths appear after the recognition of the primary tumour. Different growths in similar organs vary greatly in this respect. Some tumours disseminate so rapidly that within a few weeks the case is hopelessly inoperable, while in others the patients remain free of any demonstrable secondary deposit for many months.

Dissemination may occur in one of three ways :

1. Permeation.
2. Embolism.
3. Transplantation.

PERMEATION is the gradual extension of a tumour by active growth of cells in and along lymphatic vessels (Fig. 21). It is seen well beyond the periphery of the growth and constitutes the most advanced limit of extension of the tumour. The lymph vessel is occupied and distended by cancer cells, its walls become stretched and later will burst. The presence of these cells within the lymphatic calls forth a perilymphatic reaction, in which round cells and fibroblasts are laid down, until eventually the cancer cells are strangled and killed, the lymphatic vessel being converted into a fibrous cord. At the distal end, however, the

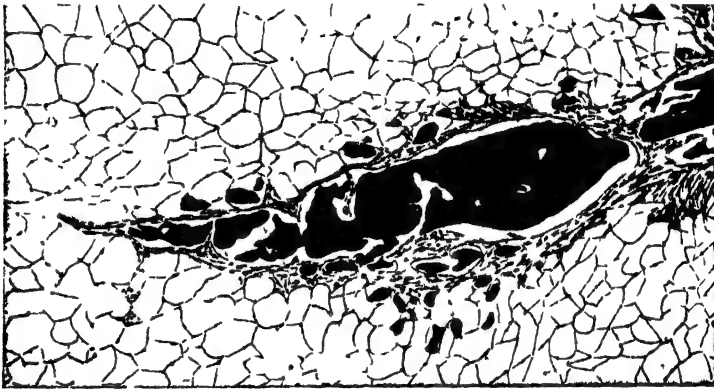


FIG. 21

A section showing the permeation of a lymphatic vessel by carcinoma cells from a primary focus in the breast. (*Kettle.*)

column of cells is still advancing by active cell division, and nodules of growth may appear at some distance from the primary tumour, having no apparent connection with it. Sampson Handley has shown that permeation occurs in vessels of medium size in which the force of the current is insufficient to sweep the cells away as emboli. He has also pointed out that, although the processes of infiltration and permeation have certain superficial resemblances, they are separate and distinct in several important particulars.

Infiltration occurs in tissue interspaces, is the earliest sign of malignancy, is best seen at the "macroscopic" growing edge and is a slow process. Permeation is limited to lymphatic vessels, is of later occurrence, is best seen at the "microscopic" growing edge often several inches from the apparent margin, and is a comparatively rapid process. These two methods of spread are interchangeable, for permeating cancer cells may burst their way through the walls of the lymphatic vessels and infiltrate the surrounding tissues, while an infiltrating group of cells may erode and enter a lymphatic vessel and begin to permeate it.

**EMBOLISM** may occur in either the venous or the lymphatic circulations, small groups of malignant cells gaining admission to the lumen of these vessels by direct invasion of their walls. These cells are then carried along in the circulation until they are arrested in the first capillary system they encounter, *e.g.*, the lungs, liver or lymphatic glands. In sarcoma embolism is the most important method of dissemination, but in carcinoma it appears to occur late in the disease, and although important it is variable in occurrence and in many cases is overshadowed by infiltration and permeation.

**TRANSPLANTATION** of tumour cells from a parent growth to a new situation is an uncommon phenomenon in human pathology.

Transplantation by contact has occurred from lip to lip, from cervix uteri to vaginal wall, and in hollow viscera from one wall to another, as in cases of papillomata of the bladder

Transcœlomic implantation refers to serous membranes, *e.g.*, the pleura and the peritoneum. If a nodule of growth appears on the parietal layer of a serous membrane, small groups of cells may be detached from its surface by the movements of the viscera, and may be grafted on to the membrane either in the immediate vicinity or at a distance. A notable example of this process is the "Krukenberg" tumour of the ovary, usually bilateral, which is secondary to a gastric carcinoma.

Transplantation by inoculation may occur in the course of operation for the removal of malignant growths, tumour cells being spilt in the exposed tissues, an early local recurrence resulting.

### CLASSIFICATION OF TUMOURS

There are so many varieties of tumour and so many transitional forms that a classification of real value is a matter of great difficulty, and for purposes of description the following arrangement has many advantages :—

#### A. Innocent Connective Tissue Tumours :

Fibroma	Chondroma	Lymphoma
Lipoma	Osteoma	Myoma
Myxoma	Odontoma	Neuroma
Chordoma	Osteoclastoma	Glioma

#### B Malignant Connective Tissue Tumours :

Sarcoma.

#### C. Innocent Epithelial Tumours :

Adenoma and Papilloma.

#### D. Malignant Epithelial Tumours :

Carcinoma including Hypernephroma.

E. Melanoma.

F. Endothelioma.

G. Teratoma.



## THE INNOCENT CONNECTIVE TISSUE TUMOURS

### FIBROMA

A fibroma is derived from fibrous connective tissue, which is a component of most parts of the body, but in spite of its wide distribution a true fibroma is of rare occurrence. It is an innocent tumour which compresses the surrounding tissues to form a capsule, and when near the surface of the body or of the alimentary canal it tends to project as a pedunculated growth. It is described as being of two varieties, hard and soft fibromata.

The **hard fibroma** (Fig. 22) is a firm lobulated tumour, which on section is seen to be composed of fibrous tissue having a white, glistening, whorled appearance. It consists of fibrous tissue and fibroblasts arranged in interlacing bundles, so that lobules are formed, which are separated by a delicate stroma of connective tissue carrying fine capillary vessels. They tend to undergo degenerative changes of a mucoid or calcareous type.

The **soft fibroma** is more cellular and contains less adult fibrous tissue. Its tissue spaces are wider, it is more vascular, and there may be œdematous fluid in it. It is a much softer tumour and may be mistaken for a lipoma or a sarcoma.

The diagnosis of a true fibroma should never be made until a microscopic section has been examined, because fibrous tissue is an intrinsic part of every new growth and of so many inflammatory processes. In soft fibromata in particular the appearances may be so difficult that a pathologist may find it impossible to give a definite opinion as to whether the growth is innocent or malignant.

Fibromata may occur in any part of the body, but the following are the commonest situations :—

1. In the skin, *keloid scars* are due to irritation or injury and are sometimes seen in operation wounds (see Fig. 3). They consist of an excessive production of fibrous tissue and are not truly neoplastic but inflammatory in origin (see Chap. I).

2. In the nerves. *Molluscum fibrosum*, or Von Recklinghausen's disease, affects the fibrous sheaths of subcutaneous nerves, leading to the formation of varying numbers of nodules beneath the skin. The condition may be confined to one nerve and its branches, or may be



FIG. 22

A hard fibroma of the palm of the hand.

so widespread that the whole body is studded with nodules. The tumours vary in size and consistence, and after some time become pedunculated from stretching of the overlying skin.

*Plexiform neuroma* is a similar condition except that it is diffuse and not localised. It commonly affects the nerves of the head and neck and rarely those of the trunk. The nerves are thickened and tortuous, and the overlying skin may become hypertrophied and wrinkled.

*Elephantiasis neuromatosis* of Virchow is associated with neuro-fibromatosis of a limb or part of the body, in which the skin and subcutaneous tissues are the seat of fibrosis and lymphatic oedema.

In all these conditions sarcoma is likely to supervene in one or more of these tumours, which will suddenly take on greatly increased growth.

3. In muscles and fascia (*desmoids*) and in connection with the periosteum, fibromata are occasionally seen.

4. In submucous and subserous tissues of the alimentary canal they form pedunculated polypi, covered either with mucous membrane or peritoneum.

5. They are described in connection with certain glands, *e.g.*, the breast, ovary, prostate and kidney, but such conditions are inflammatory in origin or the fibrosis is part of a fibro-adenomatous growth.

6. In the alveolar margins in the mouth, they occur as the *fibrous epulis*.

## LIPOMA

A lipoma is a slowly-growing innocent tumour and consists of fat cells of adult type. The groups of cells are supported by delicate connective tissue, in which run a few well-defined blood vessels. They are of two types, viz., encapsuled and diffuse.

**Encapsuled Lipomata** are classified by their situation as follows :—

- |                   |                 |
|-------------------|-----------------|
| 1. Subcutaneous.  | 4. Parosteal.   |
| 2. Subfascial.    | 5. Subsynovial. |
| 3. Intermuscular. | 6. Subserous.   |
| 7. Submucous.     |                 |

**SUBCUTANEOUS LIPOMATA** vary greatly in size (Fig. 23). They form rounded lobulated swellings which have a well-defined border and are very freely movable. They are not fixed to the underlying deep fascia, but they are attached to the skin by fibrous trabeculæ, so that when they are moved, dimpling of the skin is produced. They are soft and often give a false impression of fluctuation. These tumours may occur in any part of the body, and either grow very slowly or remain the same size for many years. In rare instances rapid growth suddenly occurs and a sarcomatous change should be suspected. Some lipomata have a mixed origin, and fibro-lipomata, myxo-

lipomata and angio-lipomata are described, the last-named being seen in infants and young children. The diagnosis is usually easy, the mobility, lobulation and softness of the swelling serving to differentiate it from a sebaceous cyst or cold abscess.

**SUBFASCIAL AND INTERMUSCULAR LIPOMATA** occur beneath the deep fascia and among muscle bundles. It is usually impossible to detect the lobulation and during contraction of the muscle they may be rendered so tense as to appear hard, and be mistaken for a ganglion, a cold abscess or a sarcoma (Fig. 24).

**PAROSTEAL LIPOMATA** are rare, arise from the outer surface of the periosteum and are usually mistaken for sarcomata.

**SUBSEROUS LIPOMATA** occur beneath the visceral and parietal layers of the peritoneum, and are not infrequently met with in connection with hernial sacs.

**SUBSYNOVIAL LIPOMATA** occur beneath synovial membranes, *e.g.*, in the knee-joint.



FIG. 23

A diffuse lipoma of the neck.



FIG. 24

A large subfascial lipoma arising from the region of the gluteus maximus.

**SUBMUCOUS LIPOMATA** occur rarely in the gastro-intestinal canal and give rise to one variety of polypus.

**Diffuse Lipomata** occur in the front and back of the neck (Fig. 23) over the shoulders and elsewhere in the body. They are more in the nature of a diffuse hypertrophy rather than a true new growth. They may be multiple and symmetrical, and are often met with in men who have lived sedentary lives and drunk to excess.

**ADIPOSIS DOLOROSA** or Dercum's disease affects women at the menopause and results in deposition of masses of fat in various parts of the body. The condition is a manifestation of hypothyroidism and is associated with severe neuralgic pains

### MYXOMA

A myxoma is a tumour composed of embryonic connective tissue comparable to that found in the umbilical cord, and is one of the rarest of all new growths. Mucoid degeneration occurs in many tumours

and a myxomatous appearance results ; again, oedema in a fibroma or in an inflammatory polypus may lead to mistakes in diagnosis.

A true myxoma consists of embryonic connective tissue cells with fine long radiating processes, separated widely from each other. The spaces in this meshwork are filled with mucin. Clinically, they appear as firm, rounded and elastic tumours which contain a glairy fluid. It is possible that a ganglion may be of this nature.

### CHORDOMA

A chordoma arises from the remnants of the notochord at the base of the skull or in the sacro-coccygeal area. These tumours are very rare and of doubtful malignancy. Microscopically they resemble chondromata, but are alveolar in arrangement and more cellular.

### CHONDROMA

A chondroma is an innocent tumour composed of hyaline or fibro-cartilage, being of slow growth and having a definite capsule. When of large size it becomes lobulated. On section it is blue-grey in colour, semi-translucent and homogeneous. These tumours are liable to several types of degenerative processes, such as calcification, ossification, myxomatous and sarcomatous changes. Microscopically their structure differs from normal hyaline cartilage only in the variation in size, number and arrangement of the cells.

Chondromata are quite common and arise in connection with (1) long bones, the short bones of the hand and foot, the pelvis and the ribs ; (2) normally existing cartilage ; and (3) in certain organs which normally contain no cartilage, *e.g.*, testis, ovaries, kidney, etc. These last are examples of teratomata and not pure chondromata. Clinically these tumours are of three types.

**Ecchondromata** may be either single or multiple. They arise from the shafts of long bones close to the epiphyseal line, and it is probable that they grow from islands of epiphyseal cartilage, which have been separated and displaced from their parent cartilage by such diseases as rickets. This type invariably becomes converted into true bone and is then known as a cancellous osteoma. Another type of ecchondroma arises from the shafts of long bones, forming large lobulated tumours, which may undergo several changes, such as calcification, ossification and sarcomatous degeneration. They give symptoms only by pressure on surrounding structures, *e.g.*, pain from nerve involvement or mechanical interference with movement of a limb.

**Enchondromata** are frequently multiple and occur in the metacarpals and phalanges of the hand (Fig. 25), and occasionally in the feet. They arise in isolated cartilaginous rests of the original cartilage from which the bone develops, and are seen in young adults. They produce a fusiform enlargement of the shaft of the affected bone, and if they grow to a large size the shell of bone will give way and the tumour grows into the surrounding structures. Enchondromata never form bone but become calcified and occasionally sarcomatous, in which case a spontaneous fracture may be the earliest sign. Diagnosis is

made by X-rays and treatment consists in local excision of the tumour, followed if necessary by bone-grafting.

**Cystic Chondromata** occur in the bones of the pelvis and the ribs forming lobulated tumours which neither ossify nor calcify, but undergo myxomatous degeneration with the formation of cystic spaces, and later tend to become sarcomatous, especially after an incomplete removal. The treatment of these tumours in the ribs is a resection of that part of the rib which carries the growth; those growing in the pelvis are frequently not amenable to operative removal and if such attempts are made usually recur as rapidly growing chondrosarcomata. X-ray or radium therapy offers the best chance of success.

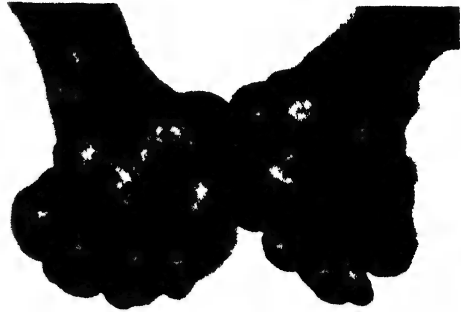


FIG. 25

Multiple enchondromata of the hands in an elderly woman.

### OSTEOMA

Osteomata are benign tumours of bone and are of two distinct types, cancellous and ivory.

**Cancellous Osteomata** are either single or multiple and are the result of ossification of enchondromata. They arise in displaced islets of epiphyseal cartilage and may be associated with rickets. The multiple type often show a familial tendency, several members of the same family being affected. They appear about puberty and continue to grow till the parent cartilage disappears. They consist of true cancellous bone with a covering of compact bone, and have a cap of hyaline cartilage. An adventitious bursa frequently develops over the surface of the cartilage.

Clinically, these tumours appear as hard outgrowths from the bone near an epiphyseal line, usually having a narrow pedicle, the soft tissues being freely movable over them. The only symptoms are pain from pressure on nerve trunks and some form of interference with the movement of the joint, due to the sudden slipping of a tendon across it, which may be accompanied by a slight sickening sensation.

*Treatment* consists in removal if any symptom is present. The pedicle is chiselled through flush with the surface of the shaft of the bone, great care being taken to remove the cartilage entirely, lest a recurrence should occur.

**Ivory Osteomata** arise in the membrane bones of the skull, forming a rounded sessile mass, which grows either outwards beneath the scalp or inwards towards the dura. The external ones are accompanied by considerable pain, but the internal are symptomless unless they reach a large size and press upon the subjacent area of brain. These tumours may affect the orbit and displace the eyeball, or fill up one of the nasal air sinuses.

*Treatment* is not called for unless there are definite symptoms, in

which case the tumour should be removed with a margin of normal bone around it.

Certain exostoses, which are not truly neoplastic, may usefully be mentioned here. Bony outgrowths may occur in certain situations where the bone is subjected to constant pressure or tension; for example, in the condition known as "rider's bone" ossification spreads from the adductor tubercle into the adductor magnus tendon and a traumatic exostosis results. The *subungual exostosis* usually occurs in the big toe, from the terminal phalanx of which a bony spur projects beneath the nail. The latter becomes broken and distorted, and finally the exostosis reaches the surface and is covered with a mass of exuberant granulations. It is inflammatory in origin and gives rise to considerable pain. The treatment consists in removal of the exostosis, the nail and all diseased tissue.

### ODONTOMA

Odontomata are tumours arising in connection with the teeth, which are developed from a downgrowth of epithelium into the anlage of the jaw. This downgrowth forms the enamel organ and is surrounded by a condensation of mesoblast from which is developed the dental sac, dentine and cement substance. The odontomata arise from errors of development of these various structures and may therefore be of mixed origin. Only two of them are important in human pathology.



FIG. 26

An epithelial odontome or fibrocystic disease of the lower jaw.

**The Epithelial Odontome or Fibrocystic Disease of the Jaw** (Fig. 26) arises in the remains of the epithelial downgrowth from

which the enamel organ is produced. Being of epithelial origin its description may appear out of place among the connective tissue tumours, but it occurs in the jaws as a primary growth and it is customary to include it in this category. It affects the lower jaw of young adults, in which it forms a dense hard tumour which grows to considerable size, expanding and eroding the bone. Macroscopically it appears as a fibrous tumour containing cysts of varying size and number. Microscopically it consists of branching masses of epithelial cells lying in a dense fibrous stroma, the outer cells being columnar, the inner ones flattened, and numerous cysts containing mucoid fluid are present (Fig. 27).

*Treatment* is local excision of the growth in its early stages, but when it is large the affected part of the jaw must be removed and a graft inserted.

**The Follicular Odontome or Dentigerous Cyst** results from the imperfect development of a secondary tooth, which remains unerupted. Certain changes occur in the dental follicle which lead to

the formation of a cyst. The tooth is usually imperfectly formed and is attached to the cyst wall in an abnormal position, being either inverted or horizontal. The cyst occurs in either sex and in either jaw, but more commonly in the maxilla which it expands. It is readily diagnosed, because there is a tooth missing in the dental arcade in the region of the swelling, there is no history of an extraction and an X-ray shows the retained tooth.

*Treatment* consists in resection of the cyst intact with the contained tooth.

**The Composite Odontome** is rare and consists of a hard mass in which enamel, dentine and cement are intermingled without any attempt at the formation of a tooth.

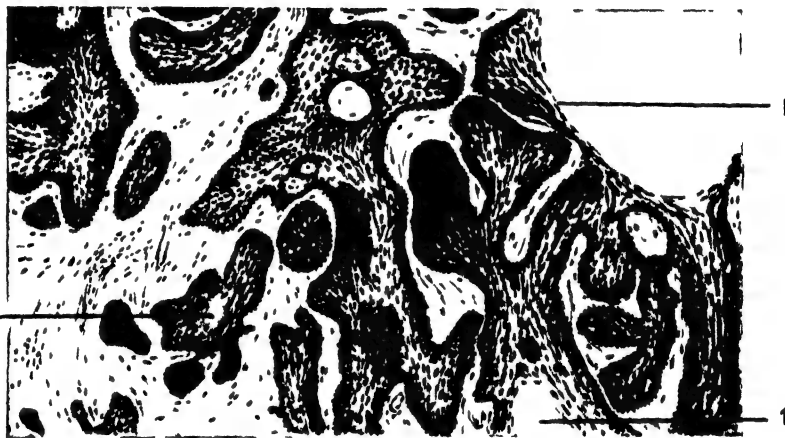


FIG. 27

Section showing the structure of an epithelial odontome. The columns of epithelial cells and the formation of cystic spaces are well seen (1). (*Kettle.*)

**The Cementome** is a tumour consisting of cement substance only, which arises from ossification in the capsule around the developing tooth germ.

**The Radicular Odontome** consists of cement and dentine and develops at the root of a tooth. It gives rise to great pain.

**The Compound Follicular Odontome** is a follicular odontome involving more than one tooth germ, and many ill-formed denticles are present in the cyst.

**The Fibrous Odontome** does not occur in human beings. It is due to fibrosis around the dental sac.

### OSTEOCLASTOMA

These tumours have been known in the past as myelomata or myeloid sarcomata, and considerable difference of opinion has existed as to their nature. The multinucleated giant cells in which these tumours abound have been regarded as derivatives of the myeloplaxes of the red marrow, and the tumours were thought to be growths of the marrow and not of the bone itself. It is now accepted, however,

that they are derived from the specialised bone reticulum from which the osteoclasts are developed, and are therefore truly primary bone tumours. The term "myeloma" is misleading, and that of "myeloid sarcoma" doubly so, and the more accurate term "osteoclastoma" is adopted here.



FIG. 28

The upper end of a tibia replaced by an osteoclastoma, the hæmorrhagic appearance of which is well seen.

**Osteoclastomata** are formed in the long bones, the sternal end of the clavicle and the jaws. They are especially common in the upper end of the tibia, lower end of the femur, lower end of the radius, upper end of the humerus and in the jaws. The cut surface is hæmorrhagic, partly yellow and partly dark red in colour, and cysts filled with blood clot are commonly seen (Fig. 28). Microscopically the appearances are characteristic, the giant cells being very numerous, varying in size and containing large oval nuclei, which are scattered irregularly throughout the plasma. They lie in a stroma of oval or spindle-shaped mononuclear cells and numerous small

blood vessels are present (Fig. 29).

These tumours begin in the interior of the bone, and the cancellous tissue is slowly eroded, the bone being thinned and expanded until finally the growth erupts through it and pushes its way into the soft tissues. They are generally regarded as benign tumours, although a few cases of metastases are on record.

**The Myeloid Epulis** is an osteoclastoma which occurs in the jaws and arises from the bone immediately beneath the periosteum (Fig. 30). It appears as a reddish soft swelling beneath the mucous membrane of the alveolar margin, often in close relationship to one or two teeth.

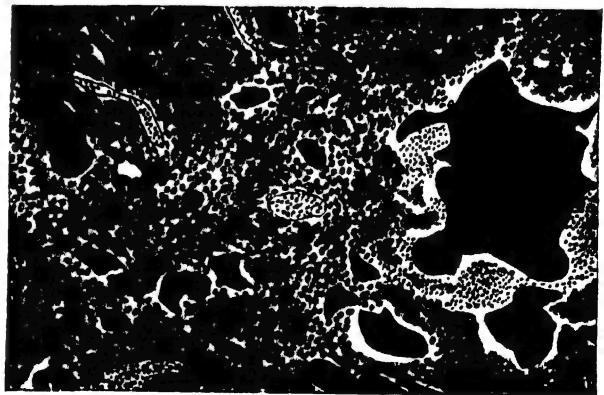


FIG. 29

Section of an osteoclastoma of the femur showing the multi-nucleated giant cells. (*Kettle.*)

*Treatment* depends on the size of the growth, the degree of involvement of the bone and the bone affected. In early cases in which the stability of the bone is not seriously destroyed, local excision of



the tumour and of the affected area of bone should suffice. When the growth has destroyed the bone extensively, a resection of that part of the bone should be carried out and a graft put in to replace it. This procedure will give excellent results in the upper extremity. In the lower limb such an operation may be impracticable without leaving an unstable and useless limb, in which case an amputation should be performed.

**Multiple Myelomatosis** is a rare disease. Multiple greyish-white tumours occur throughout the whole bony system—the ribs, sternum, skull, and small bones of the hands and feet being especially affected. Many of these may appear simultaneously, or one tumour may be present for some time and later followed by others throughout the skeleton. The pathology of these tumours is still undecided, the typical giant cells of the osteoclastoma are absent and the stroma is far more suggestive of a sarcomatous process. The view obtaining most support at present is that these tumours are plasma-celled sarcomata. They are often accompanied by the presence of Bence-Jones' albumose in the urine and by a positive Wassermann reaction, but this is not constantly present. In their late stages pyrexial attacks may occur.



FIG. 30

Section of a myeloid epulis of the jaw. The giant cells are seen encroaching upon the subepithelial connective tissue. (Kettle)

### MYOMA

Myomata are tumours of muscle and are of two types, the leiomyoma of smooth muscle and the rhabdomyoma of striped muscle.

**Leiomyomata** may theoretically occur in any part of the body in which smooth muscle exists, but actually they assume great importance owing to their frequent occurrence in the uterus as *fibroids*. They form rounded and encapsulated tumours, which on cross-section are very tough, ivory white or pinky-white in colour, and resemble a fibroma, having the same whorled appearance. Microscopically they consist of interlacing bundles of smooth muscle fibres and fibrous tissue, the relative amounts of which vary greatly in different tumours.

**Rhabdomyomata** are exceedingly rare, and it is doubtful if a properly authenticated case is on record. They have been described in the oesophagus, kidney, tongue and skeletal muscle. Striated muscle is seen in some teratomata and in teratoblastomata, and rhabdomyosarcomata are recognised, especially in the kidney.

### NEUROMA

True neuromata are rare. Amputation neuromata and neurofibromata are not true new growths of nerve tissue. Certain rare tumours in connection with the sympathetic ganglia, the medulla of the suprarenal gland and the central nervous system are described as ganglioneuromata and neuroblastomata, but these are not pure nerve tumours, being mixtures of neural tissues of many types.

### GLIOMA

Gliomata arise from the supporting tissue of the central nervous system, the neuroglia, and although it is developed from epiblast it has taken on the characters of connective tissue and the gliomata are regarded as tumours of connective-tissue origin; yet their occasional resemblance to epithelial tumours sometimes makes diagnosis difficult. They may be found in any part of the central nervous system or its derivatives, but the majority occur in the brain.

Four varieties are described. A **Medulloblastoma** occurs in the cerebellum of young children, sometimes obstructing the fourth ventricle and thereby causing hydrocephalus. It resembles a small round-celled sarcoma but its cells are oval and have a pseudo-rosette appearance.

**Spongioblastoma multiforme** is a rapid growth of the neurospongium occurring in the cerebrum of middle-aged people. It consists of large irregular cells with little intercellular substance, multiple cystic degenerative changes being often seen.

**Oligodendroglioma** is a less common lesion in the frontal lobe.

**Astrocytoma** is a lesion of early life derived from mature glial cells. It is composed of small glial cells with a rather dense network of fibrils.

These tumours occur only in the brain and spinal cord and their pathological characteristics place them in the category of innocent tumours. Nevertheless they are particularly lethal owing to their compression of the brain or involvement of vital centres in the hind brain.

## THE MALIGNANT CONNECTIVE-TISSUE TUMOURS

### SARCOMA

Sarcomata are malignant tumours derived from connective tissue and are characterised by the embryonic nature of the cells, rapidity of their growth, infiltration of surrounding tissues and their widespread dissemination. Their histological picture often presents great difficulties in diagnosis, for they bear a marked resemblance to the processes of inflammation and repair.

They are composed of actively growing undifferentiated cells, which are scattered diffusely throughout the growth in an intercellular matrix

and have little supporting stroma. The vitality of the cells is shown by the numerous typical and atypical mitoses present in their nuclei.

The blood vessels of such growths are poorly formed and consist of blood spaces and tubes lined by a single layer of endothelium. These lie in close contact with the cells of the tumour, and the fragility of the blood vessels is responsible for the hæmorrhages which so frequently complicate sarcomata and for the early and rapid dissemination by the venous blood stream, which accounts for the frequency of pulmonary metastases in these tumours. Spread also occurs by local infiltration and by lymphatic permeation and embolism.

Degenerative changes are very frequent in sarcomata owing to their rapid growth and to the delicacy of the blood vessels. Hæmorrhage

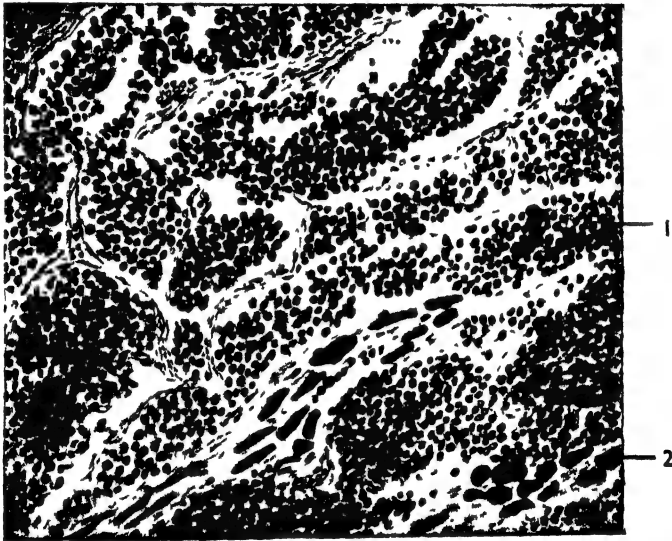


FIG. 31

Section of a small round-celled sarcoma invading muscle.  
(1) Sarcoma cells, (2) muscle fibres. (Kettle)

into the tumour, areas of quiet necrosis and fatty degeneration are all common and myxomatous degeneration is seen in some fibrosarcomata.

CLASSIFICATION.—It is not possible to adopt a purely histogenic classification, for the cells are often too undifferentiated for their origin to be recognised. It is customary, therefore, to group them according to the shape of their cells :

1. Round-celled sarcoma—large and small.
2. Spindle-celled sarcoma—large and small.
3. Oat-celled sarcoma.
4. Giant-celled sarcoma.

Other types present such features that their origin cannot be in doubt, *e.g.* :

- |                    |                   |
|--------------------|-------------------|
| 5. Fibrosarcoma.   | 8. Osteosarcoma.  |
| 6. Myxosarcoma.    | 9. Lymphosarcoma. |
| 7. Chondrosarcoma. | 10. Myosarcoma.   |

**Small Round-celled Sarcomata** are the most malignant of all tumours. They are composed of small round cells closely resembling the lymphocytes of the blood, being slightly larger and having a more deeply staining nucleus (Fig. 31). These tumours are highly vascular and usually exhibit degenerative changes, *e.g.*, hæmorrhage and necrosis. They disseminate rapidly by the blood stream, metastases appearing in all parts of the body, and death occurs within a few weeks or months. These tumours may arise anywhere in the body, and there is often nothing to indicate their exact origin, though they seem to show some predilection for the fascia covering muscles (Fig. 32).



FIG. 32

A large fungating sarcoma of the buttock.

**Large Round-celled Sarcomata** are composed of larger cells than the preceding variety, and their matrix and stroma are more clearly defined. They occur in the skin, the muscles and many of the viscera, and in addition to widespread vascular dissemination they give rise to an enlargement of the neighbouring lymph glands.

**Spindle-celled Sarcomata** are composed of fusiform cells which

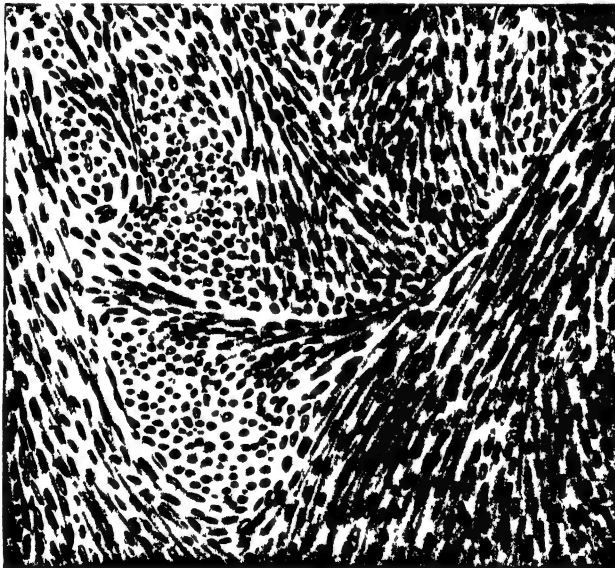


FIG. 33

Section of a spindle-celled sarcoma. (Kettle.)

vary greatly in length and breadth, but which possess a single, long, oval nucleus (Fig. 33). These cells are grouped in bundles which form a fine interlacing network, and so intimate is their relationship to the blood vessels that they appear to form the walls of blood

spaces. These growths form large solid tumours, having so well-defined a margin that they appear to be encapsuled. Hæmorrhage is not a usual complication, but they tend to undergo advanced mucoid degeneration. They are formed in all parts of the body, the smaller-celled type arising in the connective tissues generally, while the larger-celled type is associated with the periosteum and smooth muscle.

**Oat-celled Sarcomata** are midway in both appearance and behaviour between the round and the spindle-celled types. They are met with in muscle sheaths, the periosteum and the connective tissue of the viscera.

**Polymorphic and Giant-celled Sarcomata** exhibit a remarkable variation in the size and shape of their cells, some of which are multinucleated giant cells. They occur in bones and other parts of the body, but in their general behaviour they differ little from the spindle-celled variety.

**Fibrosarcomata** are slowly growing infiltrating tumours which give rise to metastases very late. They form spindle-shaped swellings and often become soft from œdema and myxomatous degeneration.

**Myxosarcomata.**—The majority of tumours thus named are in reality examples of myxomatous degeneration in pure sarcomata, such a change being common in spindle-celled fibrosarcomata. The true myxosarcoma is very rare, highly malignant and forms metastases rapidly.

**Chondro-sarcomata** are derived from cartilage and are seen in connection with bone or normally existing cartilage. They form large tumours, which appear to be simple chondromata, and they vary greatly in their degree of malignancy; in some tumours the sarcomatous elements predominate, while in others these need to be searched for in the peripheral parts of the tumour. They neither grow rapidly nor disseminate widely or early.

**Osteosarcomata** vary widely in their histological picture (Fig. 34), some consisting almost entirely of bone, others being nearly devoid of it, and their metastases vary similarly in the amount of bone they produce. Dissemination may likewise be either early and extensive or late and slight.

**Lymphosarcomata** bear a certain resemblance to round-celled sarcomata, but differ from them in some important respects. They arise in lymphoid tissue, most commonly in that of the intestinal canal, mediastinum, tonsil and cervical glands. They form large, round,



FIG. 34

Section of an osteosarcoma. (1) Spicules of bone in  
(2) sarcinomatous tissue. (Kettle.)

firm tumours, which have a homogeneous appearance on section. They infiltrate locally and form metastases only in the neighbouring lymph glands. Microscopically they consist of small round cells with a well-marked capillary blood supply, and a definite, though delicate, intercellular fibrous framework which will distinguish them from the round-celled sarcomata. The mediastinal tumours may possibly arise in remains of the thymus.

The intestinal growths spread in the submucous coat and do not ulcerate, but, invading the adjacent lymph glands, form a large tumour. While this remains localised, a drastic removal holds out some hope of success.

**Myosarcomata** are of two types derived from either smooth or striated muscle.

**LEIOMYOSARCOMATA** are occasionally met with in the alimentary canal. The cells are arranged in bundles and a longitudinal striation may be seen. They are highly malignant tumours, forming metastases in the lymph glands and the solid viscera.

**RHABDOMYOSARCOMATA** are rare tumours, the essential histological feature of which is the cross-striation, and this may be difficult to demonstrate. Striped muscle is sometimes found in a teratoma.

## THE INNOCENT EPITHELIAL TUMOURS

Epithelial cells form the lining membranes to the many surfaces of the body and the many glandular structures derived from them. Whether arranged as a surface covering or as a solid gland, the epithelial cells are always in contact with one another, and there is no intercellular substance as is present in all connective tissues. These cells are enabled to assume a distinctive arrangement in different parts of the body from the presence of a supporting scaffold of connective tissue, which also transmits the blood and lymph vessels. It is evident that a pure epithelial growth cannot occur, because in every case the stroma must keep pace with the epithelium, but it is the proliferation of the epithelium which is of prime importance in these tumours, and for this reason it is simpler and less confusing to refer to them as epithelial tumours.

The innocent epithelial tumours always reproduce, though atypically, the structure of the parent epithelium; two types therefore exist, the papilloma growing from surface cells and the adenoma arising in glandular or secreting cells. Papillomata always grow away from the surface, the cells being arranged around a central core of vascular connective tissue; adenomata grow beneath the surface in solid or tubular masses. In the latter group the tubules may dilate to form cysts, into which papillomatous processes can grow, giving rise to a papillary cyst-adenoma.

These two groups conform to certain general principles of behaviour. They progress slowly, they neither infiltrate nor disseminate, their cells retain a close resemblance to the normal and they possess a well-defined blood supply. Degenerative changes are

not usual, and are more in the nature of an excess or a perversion of the normal physiological activity of the cells. For example, the "horny degeneration" in a papilloma of the skin is merely an excessive production of keratin, and the over-activity of an adenoma results in a "colloid" or "mucoid" degeneration. In very slowly growing adenomata, fatty, myxomatous, hyaline and even calcareous changes may be seen. Papillomata are liable to inflammation and ulceration. Finally both adenoma and papilloma may undergo a carcinomatous change, especially the latter, which many observers regard as a pre-cancerous condition in certain situations, *e.g.*, the urinary bladder and the breast.

### PAPILLOMA

**Papillomata of the skin** or warts (Fig. 35) are hard tumours with a broad base of attachment to the skin. Their surface is fissured,



FIG. 35

A papilloma of the skin.

but the processes are always short. They are frequently multiple, often show pigmentation and may form a horn from excessive keratinisation.

**Papillomata of a mucous surface** present a villous appearance, being composed of long delicate processes arising from a central stalk. They are commonly seen in the urinary bladder, where they are very soft and bear a striking resemblance to certain forms of seaweed. Another type occurs in the intestines consisting of single, thick,



finger-like processes, hundreds of which may be present in the condition known as colitis polyposa.

**Intracystic papillomata** are seen in cysts of the ovary, breast and thyroid, and vary in structure from coarse branching growths to fine delicate villous processes.

*Microscopically*, papillomata consist of a core of vascular connective tissue, around which is grouped one or more layers of cells of the same type as the epithelium from which the tumour is growing. Secondary outgrowths of epithelium and stroma from the main stem will give rise to a complex compound papilloma, but the essential structure remains unchanged. Many papillomatous processes are not true new growths but are infective in origin, *e.g.*, the multiple venereal warts of the external genital organs and molluscum contagiosum.

### ADENOMA

**Adenomata** are encapsuled tumours composed of epithelial cells which in size, shape and arrangement closely resemble the normal tissue from which they arise, and further show a remarkable tendency to reproduce the function of their parent cells. Since adenomata may occur in any glandular structure in the body, they will present a very varied structure. The histological characteristics of the individual adenomata will be found in the chapters on each region of the body, and only those characteristics common to them all will be described here.

The mode of growth of adenomata depends partly on their site of origin and partly on their parent tissue; those arising from the substance of a gland are spherical and encapsuled and are known as intraglandular, while those growing from a mucous surface are polypoid, pear-shaped and pedunculated. Adenomata of solid organs, *e.g.*, the liver, are composed of solid masses of cells arranged in trabeculae or solid alveoli, whereas those of tubular glands have an acinous arrangement. This distinction is not absolute and both types may be seen in the same tumour.

The power of reproducing the function of the parent tissue is the most important feature of adenomata, those of the liver producing bile, those of the thyroid colloid, and those of the intestinal canal mucin. Since these tumours possess no outlet for the disposal of their secretion, their arrangement must become modified by the distension of their acini with retained secretion, and in this way cysts will be formed throughout the tumour, which is then named a cystadenoma. The cells lining these cysts may either atrophy from pressure, or continue active growth into the lumen of the acini, thus forming the papillary cystadenomata seen in the breast, thyroid and ovary.

The part played by the supporting connective tissue in these tumours has already been explained, but in certain situations there is a definitely co-existing growth of the fibrous tissue. Examples of this type of compound growth will be met with in several organs, a notable example being the fibro-adenoma of the breast. The exact pathological classification of such tumours is not always easy.



## THE MALIGNANT EPITHELIAL TUMOURS

### CARCINOMA

Carcinomata are malignant tumours derived from epithelial cells. Their structure is more complicated than that of the sarcomata, for their supporting connective tissue stroma and its blood vessels play an important part in their life history.

The epithelial cells vary greatly in their arrangement, for in some tumours they tend to form glandular alveoli, acini and tubules, or to assume a papillary form, while in others the cells revert to a more embryonic type and a closely packed mass of undifferentiated cells results. Generally speaking, the more rapidly a carcinoma grows, the more will its cells deviate from normal and the less likely is it to assume any recognisable form.

The stroma consists of a fibrous connective tissue with blood vessels, and varies widely in different tumours. A carcinoma arising in an organ with a well-marked fibrous framework—*e.g.*, the breast—is likely to have an abundant stroma, whereas the reverse is equally true, as is seen in the primary carcinoma of the liver. A far more important factor, however, in the production of stroma is the rapidity of the growth of the epithelial cells. Where this is very slow, the stroma is given time to become abundant, and so dense may it be that an area of slowly growing tumour cells is so completely surrounded as virtually to constitute a natural cure, *e.g.*, an atrophic scirrhus; but when the epithelial growth is vigorous and unrestrained the stroma will be represented only by a network of delicate capillary blood vessels, *e.g.*, an encephaloid carcinoma of the breast. In a great many tumours the stroma contains a round-celled infiltration of lymphocytes, plasma cells and an occasional eosinophil. This adventitious cell infiltration is to be regarded as the body's attempt at defence against the growth.

The naked-eye appearances, gross structure and microscopical characters are too variable to allow a generalised description, and each growth will be set out in detail in regional sections of this book.

Carcinomata may be divided into two main groups: (1) those arising in lining or protective membranes; and (2) those arising from glandular epithelium.

### CARCINOMA OF LINING OR PROTECTIVE EPITHELIUM

These tumours arise from the skin, mouth, pharynx, œsophagus, bladder, ureter and renal pelvis, vagina and cervix uteri. They grow from squamous or transitional epithelium and are all classed as squamous-celled carcinomata, though their appearance differs according to the complexity or simplicity of their parent epithelium.

**Squamous-celled Carcinomata** of the skin, lips, mouth and tongue (Fig. 36) are both characteristic and consistent in their appearances. Branching columns of epithelial cells penetrate the underlying tissues.

The outer cells of these processes are small, deeply staining, and exhibit intracellular protoplasmic bridges, from which the name "prickle cell" is derived; the more central cells are larger, less deeply staining, and contain eleidin granules, while the most central, *i.e.*, the oldest cells, are completely degenerate and are converted into areas of keratin. These are the "cell nests" or "epithelial pearls" which are so characteristic a feature of squamous-celled carcinoma (Fig. 37). The extent of this keratinisation is made use of by some pathologists to grade these tumours according to their relative malignancy. Absence of cell-nests is regarded as a sign of rapid growth, whereas profuse keratinisation is evidence of low malignancy.

Prickle cells, eleidin granules and cell nests are not seen in many of the growths in the oesophagus, pharynx and antrum of Highmore,

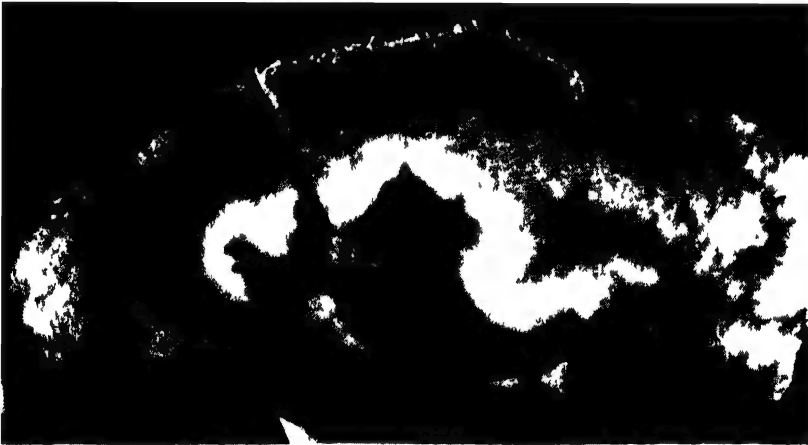


FIG. 36

An ulcerating squamous-celled carcinoma of the tongue.

in which the cells may be drawn out and compressed into a spindle form. Growths of the bladder, ureter and renal pelvis also do not show prickle cells or cell nests, being sometimes known as **transitional-celled carcinomata**.

**Rodent Ulcer** or basal-celled carcinoma of the skin consists of a large collection of oval or spindle cells surrounded by an external layer of high columnar cells—the Pallisade layer (Fig. 38). The exact origin of these tumours is still undecided (p. 239).

A squamous-celled carcinoma is occasionally seen arising from a columnar or cubical epithelium, *e.g.*, in the gall-bladder and body of the uterus. This is an example of metaplasia, *i.e.*, a reversion of cells to a less highly differentiated type.

### CARCINOMA OF GLANDULAR EPITHELIUM

These tumours fall into two groups, the carcinoma simplex and the glandular carcinoma of more highly differentiated cells.

**Carcinoma Simplex** or spheroidal-celled carcinoma consists of masses of densely packed cells, which are moulded into polygonal



FIG. 37

Section showing a squamous-celled carcinoma of the tongue. The epithelial processes are seen to be infiltrating the subepithelial connective tissue and many cell nests are present.

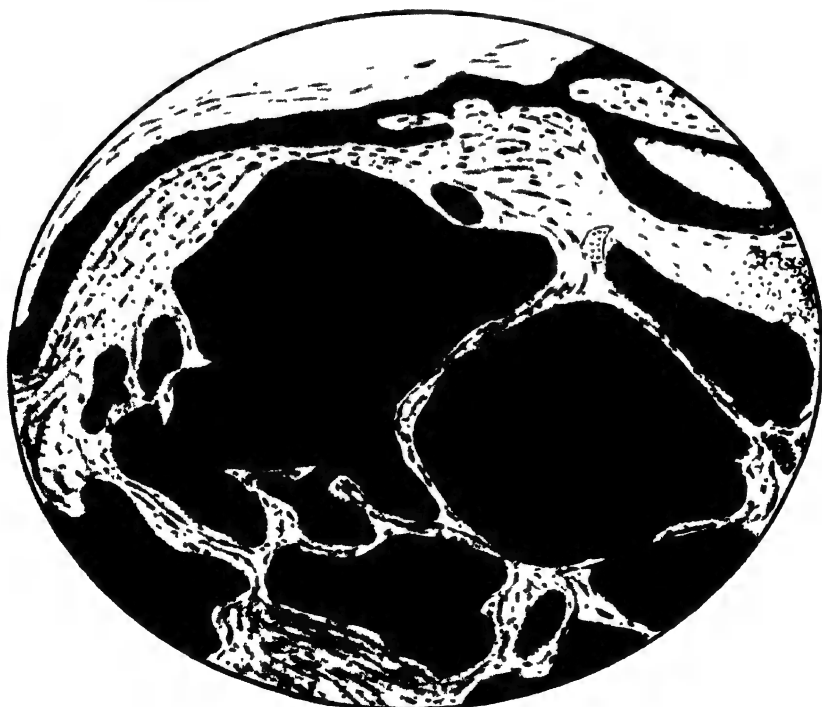


FIG. 38

Section of a rodent ulcer.

or irregular forms. The cells have a deeply staining granular cytoplasm and a nucleus with well-marked chromatin. They may be arranged in long slender processes (as at the limit of infiltration) or in short broad columns, or they may be grouped together in solid alveoli. The stroma varies greatly, depending on the rapidity of the epithelial growth and the efficiency of the host's reaction (*vide* p. 85). This variation in the stroma justifies the division of these carcinomata into two groups, viz., encephaloid and scirrhus. Carcinoma simplex may arise in many glandular tissues, but it can be most perfectly studied in the breast (Fig. 39).

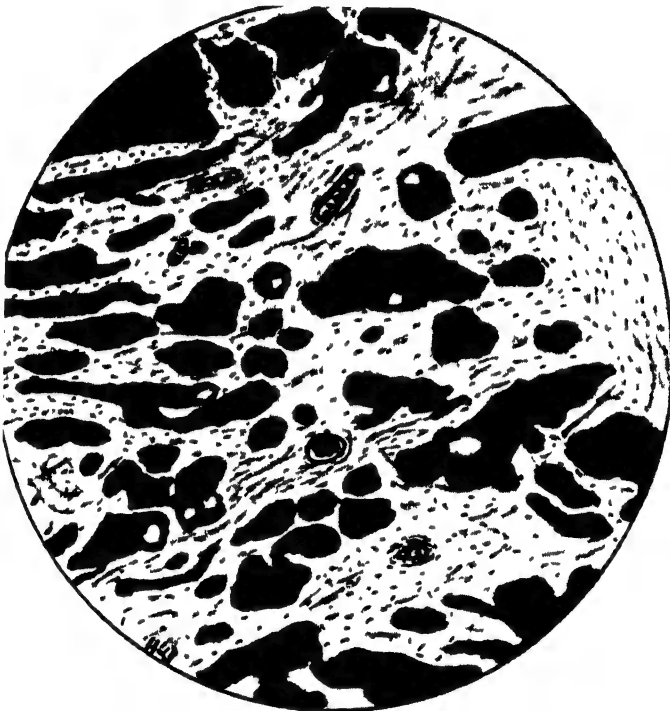


FIG. 39

Section of a carcinoma simplex of the breast.

**Glandular Carcinoma** is derived from highly differentiated cells of complex glandular structure. The arrangement of such carcinoma cells depends on their rate of growth and on the type of the parent epithelium. The more slowly growing tumours attempt to reproduce the form and shape of the glands from which they have grown, and their cells may even reproduce the function of their parent cells, *e.g.*, the presence of mucus-secreting goblet cells in an adeno-carcinoma of the rectum. The result is a very varied structure in different types of glandular carcinoma combined with a somewhat confusing nomenclature. Adeno-carcinoma, papillary adeno-carcinoma, adenoma malignum, duct carcinoma and columnar-celled carcinoma are merely expressions of the type of growth concerned, and more than one type is often seen in the same tumour.

The cells may be columnar or cubical, they tend to be smaller than normal and their nuclei are larger and more deeply stained. The acini vary in size and shape, being lined with one or several layers of cells. Cysts may form and papillary processes project into them.

### HYPERNEPHROMA

**Hypernephromata** form a group of malignant epithelial tumours, whose characteristics are so striking as to justify a separate description.

The great majority of them occur in the kidney, but there are rare examples in the testis and along the course of the ureter and elsewhere. Their nature has been the subject of much controversy, for their resemblance to the zona fasciculata of the adrenal gland has led to many theories of embryonic origin. Grawitz, having demonstrated the existence of "adrenal rests" or misplaced islands of adrenal cells beneath the capsule of the kidney, suggested that hypernephromata arose in these "rests." Others (Willis and Wilson) have favoured the theory that similar "rests" of Wolffian-body cells are responsible, but at the present time it is generally accepted that this tumour arises from the renal tubules and must be classified as highly specialised atypical carcinoma of the kidney.



FIG. 40

Section of a hypernephroma of the kidney. The pale clear cells are well shown bearing an intimate relationship to the vascular spaces (1) (Kille)

Its **macroscopic** appearance is so characteristic that a diagnosis may confidently be made in most cases. The tumour starts either at the upper or the lower pole or occasionally in the middle zone, and for a long time one or other pole remains clearly recognisable. The cut surface, which shows apparent encapsulation, is partly golden yellow in colour and partly mottled with large or small areas of extravasated blood; well-marked striæ of fibrosis appear to divide the tumour into lobules, some of which may be degenerate even to the point of cyst formation. The tumour cells may invade the renal pelvis and the renal vein is often found full of growth (*vide* Fig. 372, p. 747).

**Microscopically** a frozen section stained with Sudan III or Sharlach R shows the cells to be distended with lipid. In paraffin sections the cells are large and polygonal with clear faintly staining and vacuolated cytoplasm, and a well-stained nucleus. They may be arranged in a variety of ways. The tumour may consist of solid

trabeculae of cells closely related to the blood spaces and capillaries, of a tubular or acinous growth or frequently the cells are arranged in a papillomatous manner round a vascular core. The stroma is slight, the cells resting on the vascular spaces, which may be lined with only one layer of endothelial cells (Fig. 40).

**Dissemination** occurs chiefly by the renal vein, and metastases are found in the lungs and long bones. Many examples are recorded of secondaries appearing in bones before the primary growth was clinically demonstrable.

### MELANOMA

The melanomata are characterised by the presence of an iron-free sulphur containing pigment named melanin. They arise from the skin and the choroid coat of the eye.

**Benign Melanomata** of the skin are known as pigmented moles, which are small black or brown tumours of the skin with either a thin

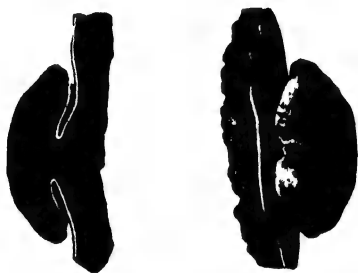


FIG. 41

Two views of a malignant melanoma of the skin showing the jet-black appearance and the greyish ulcerated exterior.



FIG. 42

Section of a pigmented mole. The heavy deposit of pigment can be seen both in the cells and in the stroma. (Kettle)

epithelial covering or a papillomatous surface. They consist of collections of small rounded pigmented cells lying in a dense fibrous stroma. The amount of pigment varies, being sparse in some tumours and profuse in others.

**Malignant Melanomata** may arise in a pre-existing pigmented mole, may follow a blow or wound, or start spontaneously in the eye. The tumours grow at a great rate and form metastases all over the body. The amount of pigment in the primary and secondary growths varies greatly, some being grey, others jet black and others quite colourless (Fig. 41).

**Microscopically** two types exist. In the first the appearance is that of a spindle-celled sarcoma with little specks of melanin scattered throughout it. This type is the melanotic sarcoma, arising in the chromato-phore cells of the connective tissue and disseminating by the blood stream. The second type consists of pigment-bearing cells arranged in acini, strongly suggesting an epithelial tumour. This is the melano-carcinoma, derived from the pigmented cells of the rete

malpighii of the epidermis and spreading primarily by the lymphatics (Fig. 42).

### ENDOTHELIOMA

The complex nature of the endotheliomata carries them beyond the scope of this book. The position is admirably stated by Kettle in his "Pathology of Tumours."

**Vascular Endotheliomata** include :—

1. CAPILLARY ANGIOMA occurring in the skin as a congenital malformation resulting in the familiar "birth mark" or "port-wine stain."
2. CAVERNOUS ANGIOMA of the liver and subcutaneous tissue, also of congenital origin.
3. HÆMANGIOMA SIMPLEX, which occurs in the subcutaneous tissues and infiltrates the underlying muscle. It is a true new growth of vascular endothelium.
4. GLOMANGIOMA, a highly specialised tumour arising in a "glomus" body of the skin (p. 288).

**Lymphatic Endotheliomata** are :—

1. SIMPLE LYMPHANGIECTASIS of the skin of the face and neck, of congenital origin.
2. CYSTIC LYMPHANGIECTASIS, as seen in the cystic hygroma of the neck.
3. CAVERNOUS LYMPHANGIOMA, which is the underlying cause of diffuse enlargement of the lips and tongue, viz., macrocheilia and macroglossia.

**Endotheliomata of Serous Membranes** are rare, the best example being the meningioma or psammoma of the dura mater. This consists of cells of endothelial origin, which have a whorled arrangement. Hard gritty nodules are formed in them by the deposition of calcium salts.

### TERATOMA

The teratomata differ from all other tumours in that they contain tissues formed from two or three of the primary layers of the developing embryo. They are the result of the cells of one individual growing within the body of another of the same species.

True teratomata are found in the ovary and testis, others being rarely found in the mediastinum, the head, and the lower end of the vertebral column, where they are known as "sacrocoxygeal tumours."

**Ovarian Teratomata** usually take the form of dermoid cysts, which contain sebaceous material and hair. In the wall of the cyst at one pole there is a solid projection covered by squamous epithelium, in which one or more teeth may be implanted. Beneath the surface the microscope reveals a multitude of tissues, *e.g.*, bone, cartilage, muscle, nerve, glandular tubules and so on, forming a heterogeneous mixture

without any attempt to produce a recognisable adult structure (Fig. 43). Occasionally there is a definite formation, such as a thyroid cartilage or lengths of intestine. The ovarian teratoma is less commonly represented by a solid tumour, which is invariably malignant, the metastatic deposits being of either mixed or single-cell types.

**Testicular Teratomata** are usually solid. Their appearance varies considerably, but in the majority the tumour seems to be encapsuled, and a fine layer of compressed testicular tissue may be seen stretched over its surface. In many examples numerous small cysts are scattered throughout the growth, while in others there is so much cartilage



FIG. 43

A section of a teratoma of the ovary showing (1) squamous epithelium, (2) cartilage, (3) fat, (4) osteoid tissue, (5) bundles of smooth muscle fibre, (6) glandular tissue. (*Kettle*)

present that it can be recognised by the naked eye. The testicular dermoid is an exceedingly rare form. The histological findings are similar to those of the ovarian tumours, tissues of every variety being mixed up together.

Teratomata are always potentially malignant, but they may behave as innocent tumours for many months. The heterogeneous mixture of tissues gives the suggestion of instability in these tumours, and hence it is not surprising that carcinoma and sarcoma may arise in teratomatous cells, and both may be found in the same tumour. When metastases occur they may reproduce the multiplicity of tissues, but more frequently one type of cell predominates, and it may be impossible to diagnose the nature of the primary tumour from the histology of the metastases.

**Sacrococcygeal Teratoma** is a solid tumour arising from the



postero-inferior aspects of the sacro-iliac region. Its appearance varies from a fully formed Siamese twin to a rounded tumour containing either perfectly formed structures or more likely a heterogeneous mixture of cells of all types. The specimen illustrated (Fig. 44) contained a primitive œsophagus, stomach and intestine together with a plaque of bone and a quantity of nerve tissue.

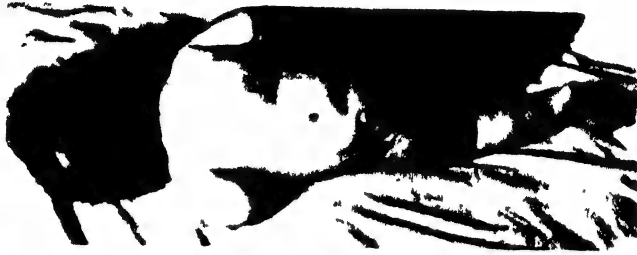


FIG. 44

A sacrococcygeal tumour. Mr V. Pennell's case.

**Epignathus** is a similar condition arising from the region of the nasal bones.

**Teratoblastomata** contain representative cells of two primary layers only. They are seen chiefly in the kidney as the "mixed tumour of infants." They arise from *rests* of the primitive segments of the body and grow from the region of the hilum of the kidney, which is spread out over the tumour as a thin compressed layer. They are greyish in colour, often homogeneous throughout, or sometimes mottled with areas of extravasated blood. Microscopically there is a densely cellular structure of small round cells supporting irregular tubules lined with columnar epithelium and both smooth and striated muscle bundles. These tumours are very malignant and rapidly form metastases.

**Chorionic Carcinoma** differs from all other tumours in that it is the product of a highly specialised tissue of another individual and is therefore in the nature of a teratoma, although it is composed of cells derived from one source only. It is an atypical growth of the cells of the trophoblast of the developing ovum and is therefore associated with pregnancy, and in the great majority of cases it follows either an abortion or a hydatidiform mole. The ovary, fallopian tubes and vagina may be affected as well as the uterus. It disseminates by the blood stream and is one of the most rapidly fatal tumours known, though it has been reported as clearing up spontaneously, and after removal of the primary growth secondary deposits are said to have faded away. It gives a strongly positive Aschheim-Zondek reaction.

The tumour is a dark-red, fleshy, friable mass. Microscopically there are two distinct types of cell bearing a close resemblance in appearance and behaviour to those of normal chorionic villi. The Langhans' cells are polygonal and have a lightly staining cytoplasm and well-defined nucleus, while the syncytium is composed of protoplasm without cell boundaries, stains very deeply and contains numbers of even more deeply staining nuclei. The Langhans' cells form closely packed aggregates with the syncytium either around

them or scattered irregularly through them. The tumour is devoid of stroma and derives its nutrition direct from the blood of the host.

Chorionic carcinoma has been observed in teratomata of the testicle, an observation which has had a profound bearing on the nature of the teratomata.

## CYSTS

Cysts are met with in many pathological conditions, and the term "cyst," applied clinically, should refer only to those swellings in which there is a collection of fluid in a sac which has a lining membrane.

They may be classified as follows :—

- |                       |   |   |   |                                |
|-----------------------|---|---|---|--------------------------------|
| <b>A. Congenital</b>  | . | . | { | Dermoid cysts.                 |
|                       |   |   | { | Embryonic "persistence" cysts. |
|                       |   |   | { | Distension cysts.              |
| <b>B. Acquired</b>    | . | . | { | Cysts of new formation.        |
|                       |   |   | { | Degeneration cysts.            |
|                       |   |   | { | Traumatic cysts.               |
| <b>(C. Parasitic.</b> |   |   |   |                                |

## CONGENITAL CYSTS

**Dermoid Cysts** are of two varieties, viz., the sequestration and the tubulo-dermoid. The so-called dermoid cysts of the ovary and testis are products of teratomata.

**SEQUESTRATION DERMoids** are formed by the inclusion of cells of the epiblast beneath the surface at any situation in the body where lines of developing skin meet and join. They may be seen, therefore, anywhere in the middle line of the body surface, in the face along the lines of junction of the maxillary and lateral nasal processes and in the lateral aspects of the neck from the branchial clefts. The more common sites are at the outer margin of the orbit, near the outer canthus, at the root of the nose, in the midline of the submental region and in the anterior triangle of the neck (branchial cyst).

These cysts are attached to the deep structures, but the skin moves freely over them. They should be excised.

**TUBULO-DERMoids** arise in connection with embryonic glands and ducts, which should normally disappear. They are represented by thyroglossal cysts (p. 353), and those behind the rectum which are derived from the post-anal gut.

**Embryonic Persistence Cysts** arise from specialised embryonic structures, which should normally disappear completely or remain as small vestigial remnants.

In the male, cysts arise in the remnants of the Wolffian body and duct, viz., the organ of Giraldes of the spermatic cord and the vas aberrans of Haller in the epididymis, or from the representatives of the Müllerian duct, viz., the hydatids of Morgagni, or again from the persistence of the central part of the processus vaginalis, from which is derived the encysted hydrocele of the cord.

In the female, cysts of Wolffian origin are those in the broad

ligament, in the organ of Rosenmüller, from Kobelt's tubules and in Gärtner's duct. A persistent canal of Nuck gives rise to a hydrocele of the round ligament.

### ACQUIRED CYSTS

**Distension Cysts.**—EXUDATION CYSTS are the result of either trauma or inflammation in pre-existing cavities and should not be included among true cysts.

RETENTION CYSTS are due to the retention of the normal secretion of a gland from an obstruction of its duct. Numerous examples will be described in the breast, pancreas, salivary glands, etc.

**Cysts of New Formation.**—IMPLANTATION CYSTS sometimes named implantation dermoids—are due to the implantation of squamous epithelial cells in the subcutaneous tissues by penetrating wounds of either sharp or blunt instruments. Their vitality being unimpaired, they continue to grow until a cyst is formed, lined by squamous epithelium and containing degenerate keratinised debris. They are usually seen in the hand, an excellent example being those in the fingers of seamstresses. Foreign-body cysts may be formed around a retained and encapsuled foreign body, being lined with endothelium and having a fibrous capsule.

**Traumatic Cysts.**—Injury in certain parts of the body, *e.g.*, the lumbar and lumbosacral regions of the back and the antero-external part of the thigh, is sometimes followed by a large collection of fluid blood between the subcutaneous tissues and the deep fascia. Absorption and resolution do not always follow, and if the fluid blood is not removed by aspiration, a post-traumatic serous cyst will form. If left sufficiently long, the walls of the cavity will become lined by endothelium, and the cyst will be extremely difficult to eradicate.

**Degeneration Cysts.**—Cysts may occur in tumours either as the result of hæmorrhage or from liquefactive necrosis, due to an inadequate blood supply. Certain rapidly growing sarcomata with a pseudo-capsule contain large quantities of clear fluid.

### PARASITIC CYSTS

**Echinococcal (or Hydatid) Cysts** are the most important of this type of cyst in the human body, and are the intermediate stage in the life history of the *TÆNIA ECHINOCOCCUS*. The disease is much more common in Australasia, but is occasionally endemic in this country. The adult worm, which inhabits the small intestine of the dog or wolf, is  $\frac{1}{4}$  in. long and is composed of four segments. The head is armed with four suckers and forty hooklets in two rows, while the tail segment contains the reproductive organs and is equal in length to the other three together (Fig. 45).

The ovum is roughly one and a half times the size of a human red blood cell, is enclosed in a chitinous envelope and bears three pairs of hooklets. Having been excreted from the dog, it gains entrance to the human being by water or green uncooked vegetables such as watercress. In the stomach its envelope is digested, and the

ovum anchors itself to the gastric mucous membrane and erodes its way into a radicle of the portal vein, whence it is carried to the liver. Here the majority are arrested and settle down to form a hydatid cyst of the liver, but some pass through the liver capillaries and enter the general circulation to be filtered out by a capillary system in any part of the body, e.g., in the lungs, kidneys or long bones. Fig. 46 shows a large hydatid cyst of the liver.

The hydatid cyst is composed of three layers, an outer or pseudo-cyst being derived from the host by compression of the surrounding tissues, a middle or ecto-cyst and an inner or endo-cyst.

The ecto-cyst is a chitinous covering to the epithelial or generative endo-cyst, which buds off daughter and granddaughter cysts. In these cysts little fleshy processes develop, known as "brood capsules," from which are developed the scolices. A scolex is the head segment of a future worm, so arranged that its suckers and hooklets face into a central recess or sinus. The contents of the cyst are clear fluid of sp. gr. 1002 to 1004, inorganic salts, a trace of pyrocatechin (a sugar-reducing agent) hooklets and scolices.

The cyst may continue to grow to great size or certain complications may follow. The parasite may die, in which case the fluid is absorbed, the contents become a pultaceous mass, which after a long time will become calcified. The cyst may become infected and an abscess will form, or it may rupture into related serous sacs, e.g., the peritoneum, and the daughter cysts

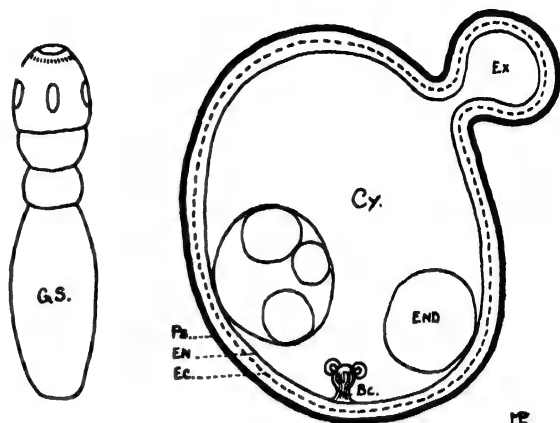


FIG. 45

A diagram representing on the left the *Tænia echinococcus* and on the right the hydatid cyst.

Cy., Ex., the exogenous, and End., endogenous cyst; Ps., pseudo-cyst; Ec., the ecto-cyst; En., the endo-cyst; Bc., a brood capsule surmounted in this case by two scolices; G.S., germinal segment.



FIG. 46

A large hydatid cyst of the liver containing many daughter cysts.

will be implanted over their surface, or into the duct of a gland as in the case of the pelvis of the kidney.

**Diagnosis** is made by the Casoni intradermal test, a complement fixation test and an eosinophilia in the blood. Aspiration of the fluid contents should never be practised.

**Treatment** is complete removal wherever possible, failing which the cyst should be marsupialised to the skin and later opened and irrigated with formalin.

The only other parasite which causes cysts in the human being is the *Trichina spiralis* (p. 51).

R. M. HANDFIELD-JONES.

## CHAPTER VII

### WOUNDS AND BURNS

#### WOUNDS

**A** WOUND is defined as a forcible solution of continuity in soft tissues. Such an injury may be classified in many ways, and there is a special tendency to describe wounds of war and peace as if they belonged to entirely different categories. Essentially their pathology, clinical features and treatment are identical, and no good purpose can be served by keeping them apart. The following classification covers both types :—

- |                                |   |   |
|--------------------------------|---|---|
| A. Closed or non-penetrating . | . | Contusion.<br>Hæmatoma.<br>Abrasion.  |
| B. Open or penetrating .       |   | Incision, stab or puncture.<br>Simple penetration without exit.<br>Laceration.<br>Perforating, <i>i.e.</i> , with exit.<br>Disruption or blast. |

#### NON-PENETRATING WOUNDS

Closed wounds are those which do not penetrate the whole thickness of the skin.

**A Contusion** is produced by applied violence, usually with a blunt instrument, as a result of which there occurs a rupture of blood vessels and a varying amount of damage to soft tissues beneath the skin. If the resultant hæmorrhage extravasates in the skin and underlying soft parts an *ecchymosis* or *bruise* forms.

Its clinical signs and symptoms are pain, discoloration and swelling. Pain and swelling vary inversely according to the tissue damaged, the more lax structures exhibiting little of the former and much of the latter. Thus a contusion of the eyelids and scrotum results in considerable swelling and little pain, whereas the opposite holds good in the fingers and scalp. The discoloration is due to the disintegration of red blood cells and liberation of hæmoglobin : the nearer the surface the contusion occurs, the sooner does the discoloration appear and the more evident it is. The colour is at first a deep purple black, changes to purple, blue, red, brown, green and finally fades away in a yellowish tint within fourteen to twenty-one days. These changes are brought

about by the chemical processes through which hæmoglobin passes during its preparation for final absorption. The colours are due to bilirubin, biliverdin, hæmatoidin and hæmosiderin. When the contusion occurs in deeper planes, swelling and discoloration may appear at the surface some considerable distance from the initial injury.

**A Hæmatoma** is formed when blood is poured out in greater quantities and especially when its diffusion is prevented by fascial planes and muscle attachments. In the first few days it presents a rounded fluctuating swelling resembling an abscess, but without any sign of inflammation. Unlike an abscess, which is at first hard and then softens, the hæmatoma soon acquires a boggy and then a hard edge. This is the beginning of the process of organisation (p. 5) which may lead to complete absorption. Not all clots, however, achieve this happy ending; some leave evidence of their presence in the form of a hard fibrous scar, while others give rise to "serous cysts." These latter occur on the surface of the brain, being known also as "arachnoid cysts," and in the lumbar region, upper part of the buttock and the antero-lateral aspect of the thigh. Fibrin having been deposited at the periphery and the hæmoglobin having been removed, a cyst filled with clear pale yellow serum is formed. In the absence of treatment this cyst rapidly gains an endothelial lining and its removal may prove an extensive and difficult business.

It will be appreciated that extravasated blood is such an excellent culture medium that it is not uncommon for a hæmatoma to become infected in debilitated or toxic patients. Further it should be noted that internal organs such as the brain, liver, kidneys, lungs and gastro-intestinal tract may suffer contusion and hæmatoma formation in exactly the same way as muscle and subcutaneous tissues.

*Treatment.*—An extensive bruise or hæmatoma demands rest and the application of either evaporating lotions or heat. A hot bath will relieve pain and firm pressure limits extravasation, while in the later stages massage will promote rapid absorption of the exudate. If a hæmatoma is slow to resolve, the fluid blood must be removed by aspiration and the parts firmly bandaged to prevent the formation of a serous cyst. If it should be under such pressure as to cause great pain, evacuation of the clot through a small incision with a tenotome will give immediate relief.

**An Abrasion** is produced by friction upon some hard or rough surface and consists merely in the removal of small areas of the epidermis. As a result many superficial nerve endings are exposed and these trivial lesions are unusually painful. Grit and other foreign matter is often engrained and surface sepsis is the rule. Careful cleansing and the use of a mild mercurial ointment is the only treatment needed. An abrasion heals by granulation beneath a scab.

#### CRUSH SYNDROME

A special type of contused wound is that caused by prolonged compression by baulks of timber or masses of masonry. Although this was recognised before this war, the attention of British surgeons was

forcibly drawn to it during the bombing of Britain. A man was admitted to St Mary's Hospital, having been trapped under a ruined building; a large baulk of timber had pinned down his right thigh and masonry had crushed his right side. He was relieved after seventeen hours, and the skin subjected to pressure appeared almost dead. He was surprisingly little shocked and the skin rapidly recovered, but the right leg began to swell until the skin became so tightly stretched that it seemed as if it must burst. Twenty-nine hours after admission he developed anuria and speedily died. Such is an example of the "Crush Syndrome." Little is known of its etiology, though it bears many points of resemblance to the results of an incompatible blood transfusion. Crushed limbs should be treated by compression above the infected parts with a sphygmomanometer air cuff to delay absorption of toxic products, and every effort made to overcome diminishing renal function.

### PENETRATING WOUNDS

Open wounds are those in which a solution of continuity of skin or mucous membrane leads to disrupted soft tissue below. There are numerous varieties of such wounds, but their pathology is basically the same, varying only in degree. We describe three stages:—

1. **DISRUPTION OF SOFT TISSUES** occurs immediately and its extent in the various anatomical planes depends upon the nature and force of the inflicting agent. Disrupted muscle fibres have lost their blood supply, contractility and power to resist infection. Such muscle is obviously a splendid culture medium for anaerobic organisms.

2. **PROTECTIVE INFLAMMATION.**—Very quickly the mechanism of inflammation is called into action (p. 1) and the body mobilises its defences. The exudate causes a visible oedema and this may be so great in certain cases and in certain situations (*i. g.*, the thigh) to endanger the circulation.

3. **ESTABLISHED INFECTION.**—Every penetrating wound, except those inflicted by the surgeon, is potentially infected. The skin is unclean and particles of dirt, debris and clothing are almost certainly carried into the wound. For a period, not less than six and not more than twelve hours, organisms may be said to be still upon the surface of the damaged tissues and not yet embedded and in action, by which we mean multiplying and producing toxin. After this "safe" period the wound is definitely infected.

### INCISIONS, STABS AND PUNCTURES

**Incised Wounds** are produced by sharp cutting instruments such as knives, surgical scalpels, fragments of glass, etc., or more rarely by blunt violence upon tightly stretched skin such as the scalp. An incised wound tends to gape (the extent depending upon the elasticity and tension of the injured tissues); its length is greater than its depth; its edges are regular and there is only microscopic devitalisation of cells; it bleeds freely and is painful because of the number of sensory



nerves cut ; it is accompanied by a moderate degree of shock ; and it heals by first intention within a week unless infection supervenes.

In all accidental incised wounds the whole extent of the track must be thoroughly explored to assess the exact amount of damage to important structures, such as vessels, nerves, muscles and tendons. Regeneration, after primary suture and in the absence of infection, will be good in nerves, tendons, muscles and bone, poor in fat and secreting glands and absent in the central nervous system.

**Stabs and Punctured Wounds** are due to sharp-pointed instruments, such as pins, needles, nails, wood splinters, bayonets and fish hooks. They have a relatively small orifice and their importance lies in the fact that infection or foreign particles can be carried into the depth of the wound, the opening of which is quite inadequate for drainage ; further damage may be done to deep structures which it is difficult to visualise.

In the absence of complications punctured wounds bleed little, are not painful except at the moment of infliction and heal rapidly. If they become septic they must be opened and treated accordingly. If a retained foreign body is suspected, an antero-posterior and a lateral X-ray film should be taken and its position thus determined. Fish hooks are best removed by pushing the shaft still further in so that the barb comes out through a second puncture wound.

**SNAKE BITES** form a subgroup of punctured wounds. In this country the adder is the only poisonous snake, but in tropical parts there are many whose bites prove fatal. A snake's venom is produced in its parotid glands and is led by special ducts to the fangs ; thus the poison is implanted in the very depth of the wound. It is usually an albumose with a markedly acid reaction and as we have already seen (p. 17) each specific venom produces an anti-venom just as bacterial toxins stimulate the formation of antitoxins.

Swelling, pain and local discoloration are rapidly accompanied by faintness, weak pulse, vomiting, dilated pupils and a feeling of terror. In more virulent cases collapse soon follows and death may occur within an hour. Local treatment should be vigorous. A tourniquet is applied immediately, the wounds opened up, free bleeding encouraged and the tissues irrigated with ammonia or hydrogen peroxide. Brandy and heart stimulants are given meantime. In districts where poisonous snakes abound anti-venom is prepared against the bite of all known species. When administered within an hour of the bite this treatment has reduced the mortality to a low figure.

**BITES AND STINGS** from bees, wasps, mosquitoes, certain flies, lice, fleas, scorpions and spiders are also punctured wounds. Quite apart from the importance of such lesions from the point of view of the transmission of infectious diseases, they have an intrinsic bearing upon surgical treatment. The local reaction is similar to that in snake-bite and may be accompanied by an urticarial eruption. The actual swelling may in certain situations be a danger to life, *e.g.*, tongue and larynx, while bites within the danger area on the face may lead to cavernous sinus thrombosis and death. Treatment is by removing the sting—if possible—and dressing the wound with an alkaline solution.

### LACERATED WOUNDS

The wounds of war are only too frequently of this type, and even in peace time in this machine age lacerations form a considerable percentage of the whole. They are produced by a tearing or bursting force and consequently are irregular in shape. The skin is often less extensively injured than the underlying tissues, but is commonly lifted off them over a wide area. The edges are ragged and purple, subcutaneous fat, deep fascia and muscle are disrupted and swollen, and hæmorrhage is often conspicuous by its absence. This is due to tearing of the vessels and consequent retraction of the intima which assists clotting. The mouth of a lacerated wound will often be found filled with clot.

Injury to nerves leads to a temporary local insensibility and a dull ache may be the only complaint. Primary shock is slight in many patients, but secondary shock is likely to be severe.

A wound of this type is probably caused by a jagged agent and contamination is inevitable. Furthermore main vessels may be involved in deep wounds and from every point of view the conditions are ideal for the rapid growth of invading organisms.

Lacerated wounds heal by granulation tissue.

### PENETRATING WOUNDS WITHOUT AN EXIT

Such wounds may be incised, punctured or lacerated. They need special emphasis on account of the entirely unexpected damage they may inflict upon deep structures. Many war wounds are of this type and the path traversed by bullet, shell fragment or bomb splinter can only be guessed at. Such foreign bodies must be localised by X-ray methods and their estimated track from entrance to point of lodgment gives a reasonable picture of possible damage to intervening structures. Treatment will be planned accordingly.

### PERFORATING WOUNDS

These wounds have points of both entry and exit. They are due to missiles which still retain a high velocity and have certain advantages over penetrating wounds; there is no retained foreign body and under some conditions they are less dangerous. They are classified as follows:—

1. Both entry and exit wounds small.
2. Entry small and exit large.
3. Both large.

1. Both entry and exit small. Such wounds are usually caused by high-velocity bullets which pass through the body without hitting bone. In rare instances the bone may be cleanly holed and the hole of exit still remain small. The clinical picture depends upon the part of the body involved and the internal structures injured. In the limbs the missile may pass through without traversing anything but skin, muscle and fascia, and such a "tunnel" wound may need little attention;

it may sever main arteries or nerves, damage to which will be revealed by swelling, absence of distal pulsation and loss of function. In the trunk the line between entry and exit gives an indication of which viscera may be involved.

2. Wounds with a small entry and a gaping lacerated exit are almost always due to missiles fired at close range which have hit bone in their path.

3. Wounds with large holes of both entry and exit are caused by big fragments of bomb or shell-casing or to a bullet which has already hit something and begun to turn over. A wide extent of tissue including main vessels and nerves is likely to be destroyed, and such cases will probably not reach the surgeon.

### DISRUPTION WOUNDS

Wounds due to blast have attained a prominence in this war owing to aerial bombing and the underwater explosion of depth charges. As we have seen every wound has a certain disruptive element in it, but blast injuries are those in which there is no tissue penetration by any tangible foreign body. Two forms have emerged, both resulting from an explosive burst at fairly close quarters.

**Blast Injury to the Lungs.**—A great quantity of material has been collected during aerial attacks upon this country, much of it trivial, some of it misleading. Experiments have shown that both upon land and under water injury to the lungs has been absent or diminished if the victim has complete protection of the abdominal wall by a metal shield. The lung is not damaged—as was thought—by violent retrograde movement of air down the trachea into the bronchial tree, but by forcible compression communicated to the chest by a violent displacement upwards of the diaphragm. In non-fatal cases air vesicles are ruptured and multiple small interstitial hæmorrhages occur. A characteristic X-ray film reminiscent of widespread bronchopneumonia results. Patients will complain of a tightness in the chest, dyspnoea and hæmoptysis. Whilst its importance as a cause of immediate death cannot be denied, the clinical manifestations in non-fatal cases have been somewhat exaggerated (see also p. 464).

**Blast Injury to Soft Tissues.**—The findings in such a case cannot be more clearly explained than by the following report from the author's experience during the concentrated bombing of London in October 1940. Amongst a large number of severe casualties a woman of 24 years was admitted with a large lacerated wound of the left buttock. No point of exit could be discovered but she complained of pain in her left Scarpa's triangle. She was marked "Resuscitation and operation as soon as ready," being very gravely shocked. As the night wore on and as our list of immediate operation cases was being rapidly worked through by four surgical teams, I went repeatedly to the resuscitation room to check progress; other cases were recovering and being sent to the theatre, but nothing seemed to make any impression upon this woman. On several occasions I impressed upon my staff how important it was that she should be operated upon, but it was not until twenty-nine

hours had elapsed that she was fit. Neither at operation nor at post-mortem was a foreign body discovered, the tissues having been torn apart by blast, the effect of which had reached the front of her thigh. There was extensive hæmorrhage but the rectum and all main vessels and nerves had escaped injury. The cause of death was shock.

Similar cases must be familiar to all surgeons dealing with air-raïd casualties ; they are usually fatal from the severe degree of shock from which they are suffering and little can be done to save them.

### GENERAL PRINCIPLES OF WOUND TREATMENT

Every wound, except those inflicted by the surgeon, is potentially infected. As we have seen (p. 120) a certain period elapses before invading organisms actually establish themselves, become embedded in the tissues and start to multiply and form toxins. "How long does it last?" and "Can we utilise it to prevent infection and convert the wound into a clean surgical one?" The answer to these two questions lays the foundations upon which the treatment of *all* wounds is built up.

This safe period lasts for at least six, probably eight, hours and in some cases for twelve ; it is accepted that up to eight hours from the receipt of injury surgical treatment should succeed in preventing all but superficial sepsis. It will do so only if the following general principles be strictly adhered to.

1. Every wound demands operation under full anæsthesia and full aseptic technique. The former should require no comment, but the latter so often is honoured more in the breach than the observance. In civil surgery many minor injuries are dealt with in casualty departments of hospitals by an imperfect aseptic ritual ; in war asepsis is sometimes sacrificed to speed and casualties may be sent direct to the theatre imperfectly prepared, often indeed not fully undressed. There is hardly an emergency which can justify so flagrant a breach of surgical principles.

2. Protect the wound and cleanse the skin. The wounded area is protected by laying on it (not packing into it) sterile gauze, which is maintained in position by hand. The surrounding skin over a wide area is rigorously cleansed with ether soap and warm water ; in air-raïd casualties in whom the skin is engrained with brick and mortar dust I use a sterile scrubbing brush. The area is then dried, shaved and painted with an antiseptic liquid (alcohol when available, liquor antisepticus when not). The field of operation is then towelled up as in a clean operation and the surgeon and his assistant rewash and don sterile gowns and gloves. The gauze is removed from the wound.

3. Excise the wound edges. Using a very fine knife and toothed forceps the edge of both skin and subcutaneous tissue is removed in one piece. It is quite unnecessary to take away more than  $\frac{1}{8}$  in. of these tissues.

4. Explore and identify every lesion. The wound must be explored in its whole extent, and this will frequently entail its extension by one or more suitably placed incisions. Then every lesion must be identified with meticulous care. This is the process of "débridement," which

differs from simple excision in being more searching and thereby more efficient.

5. Remove all seriously damaged tissue. All dead and devitalised tissue, especially muscle and fibrous material, is an excellent pabulum for bacteria. All such components of the wound must be cut away until a healthy bleeding surface is revealed.

6. Obtain hæmostasis. Buried ligatures are to be avoided as far as possible; large vessels must be tied, but smaller points can be controlled by crushing. Our object is to leave the wound as dry as can be.

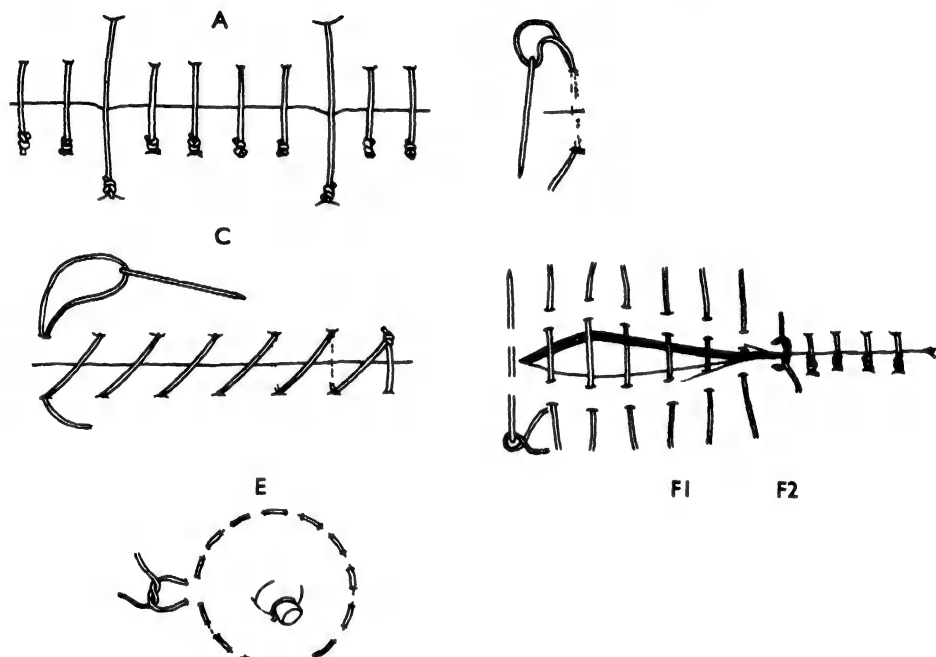


FIG. 47

Diagram of different types of sutures.

A Interrupted coaptation sutures with two deep tension sutures, B a blanket stitch; C, a continuous skin stitch; D, a Lembert's invaginating stitch here shown as interrupted, but it can be used as a continuous suture also E, a purse string suture; F1, a mattress stitch; and F2, an overlapping or sliding stitch.

7. Suture nerves and extrasynovial tendons (see Chaps. XLIII and I.).

8. Insufflate every recess and surface with sulphamamide powder (see p. 38).

9. Drain the wound except in clean operation cases when the probability of collections of blood and serum can confidently be excluded. Rubber tubes should be avoided, except for certain special situations, and soft or corrugated rubber tissue used.

10. Suture the wound *if this can be done without tension*. This proviso is absolute and admits of no exception, it is better to leave a wound open than to stitch it too tightly. The latter impedes the blood supply and predisposes to infection. Fig. 47 shows different types of skin suture.

11. Dress the wound. A clean sutured incision requires a simple, dry, sterilised gauze dressing covered by a layer of wool. The whole is retained by a bandage firmly and evenly applied to support the parts. When a tourniquet has been employed, a Robert Jones pressure bandage should be used, as for example after the removal of a semilunar cartilage from the knee. A layer of gauze and wool is loosely fixed by a sterile bandage, over which is laid a thick layer of wool from ankle to groin and this in turn is bandaged more firmly. Finally another layer of wool is applied and the third bandage can be pulled tightly and evenly.

The time during which dressings must be retained varies considerably. In general we can say that bulky dressings are kept in place unnecessarily long. Not only are they uncomfortable but both gauze and wool are expensive and scarce. Clean surgical wounds are sealed within twenty-four hours and need nothing more than a single layer of gauze held in place by mastisol, collodion or plaster strapping. Other wounds such as that following a radical mastectomy require large dressings and firm pressure, while all discharging wounds obviously call for generous applications of absorptive material.

12. Immobilise the wounded area. Certain anatomical regions either cannot be put at rest (*e.g.*, the gastro-intestinal tract) or do not require it. Wounds of the extremities, especially those of war, have been shown by Trueta and confirmed by all those who have dealt with air-raid casualties to progress more favourably when encased in plaster of Paris (see below).

13. General treatment involves attention to the patient's diet, bowel and bladder. In traumatic surgery pain will not be a prominent feature if the closed plaster technique has been adopted, but after many surgical operations it may be severe and call for the administration of opiates for some days.

Finally every patient sustaining a potentially infected wound must be given a prophylactic injection of both anti-tetanic and anti-gas gangrene serum (pp. 34 and 38).

### MODIFICATION IN TECHNIQUE FOR INFECTED WOUNDS

We have seen that after eight, twelve or at most eighteen hours a wound is inevitably infected and its "débridement," as detailed above, is no longer the method of choice; in fact such a procedure would do more harm than good. In these cases wounds must be enlarged to ensure free drainage and to remove foreign bodies and all obviously dead tissue (Figs. 48 and 49).

**Closed Plaster Method.**—The wound is lightly packed with vaseline gauze. Although the original Winnett-Orr technique of applying plaster directly to the skin is followed by many surgeons, there is grave danger in such a procedure, and it is our practice to bandage the limb with a thin layer of vaseline gauze in the whole extent to be covered. Plaster is then applied so as to immobilise the joints above and below the wound and to maintain them in their most favourable functional position. When the plaster has firmly set, the limb is elevated to 45 degrees to



FIG. 48

Private H. A. Admitted 1st June 1940, two days after being wounded. Showing the large exit wound, grossly infected. Treated by the closed plaster technique. (*Surgery of Modern Warfare*)



FIG. 49

Private H. A. The plaster was renewed on the 14th, 21st and 34th days. Showing the clean granulating wound on removal of the final plaster. (*Surgery of Modern Warfare.*)

assist venous and lymphatic return. We believe that this small detail of technique is of great importance and one which does not receive the attention it deserves. The plaster is left on for three or four weeks.

It is essential that the indications for the removal of the plaster be thoroughly understood. They are : (1) presence of pain and swelling, (2) looseness of the plaster from muscular wasting, (3) a sudden sharp rise of temperature maintained for more than twelve hours, (4) secondary hæmorrhage, (5) evidence of gas gangrene and (6) smell. The last is inevitable but can be controlled for a time and to a certain extent by the use of deodorising bags.

**Carrel-Dakin Treatment.**—Continuous irrigation of wounds by eusol has been eclipsed by the closed plaster method. Nevertheless it has its place in the treatment of heavily infected wounds with a tendency to deep pocketing, and it is a perfect preparation for skin grafting or secondary suture in the final stages.

**Late Closure.**—After a time the wound has become filled by granulation tissue, which now forms a surface flush with the surrounding skin. Much time can be saved and a considerable amount of subsequent contraction of scar tissue can be avoided by one of two methods.

**A. SECONDARY SUTURE.**—A wound is suitable for secondary suture provided that (1) a swab shows a reasonable freedom from infecting organisms, (2) no sloughs are present, (3) the granulations are not too fragile, *i.e.*, do not bleed at the least touch, (4) no sinuses leading to retained foreign bodies or dead bone are present, (5) epithelium is already beginning to grow in from the edges and (6) not more than twenty-one days have elapsed since the receipt of injury.

The wound is dusted with sulphanilamide powder, the skin is carefully cleansed and deep tension sutures are introduced together with a few more superficial interposed stitches. The area is dressed with vaseline gauze and immobilised.

**B. SKIN GRAFTING.**—Many wounds do not fulfil these conditions and are best treated by skin grafting by one of the methods described on p. 168.

### CROSS-INFECTION OF WOUNDS

The “secondary” infection of certain specific lesions with non-specific pyogenic cocci has long been recognised as an unusually serious complication. We are taught, for example, that a tuberculous abscess must on no account be contaminated by skin cocci lest a persistent sinus or even amyloid disease result. But it is not so generally appreciated that the prevention of secondary infection is of equal importance in every wound. I have drawn attention for many years to the urgent necessity of excluding staphylococci from the drainage incision for a streptococcal tenosynovitis in the flexor sheaths of the hand. The Medical Research Council have done great service in emphasising the urgency of this question in their memorandum upon “Hospital Infection of Wounds.”

Cross-infection may result from many factors, among which the following are the most important :—

1. Faulty technique in dressing wounds. A “dressing” requires



two persons, the actual dresser and his assistant. The former is concerned solely with the removal of old materials, toilet of the wound and reapplication of gauze, wool and bandages; the latter is in charge of the dressing trolley, and hands by means of forceps sterile instruments, lotion bowls, dressings, etc., to his colleague. Our hands can never be sterilised and sterile rubber gloves cannot be worn for ward work except in very special circumstances, so that all manipulations must be done with forceps. The hands must never touch the wound, its surrounding skin, the inner dressing, any tubing or waterproof covering or any sterile bowl, lotion or material.

Details of the packing and sterilisation of drums and the equipment of a dressing trolley vary greatly in different hospitals. They should be so arranged that successive dressings can be done rapidly without imperilling the sterility of any of their components.

2. Imperfect sterilisation of ward utensils. All bowls, kidney dishes and irrigation vessels are sterilised by boiling in the usual way, but it is frequently forgotten that special baths for arms and legs as well as the ordinary ward bath are in equal need of sterilisation. Small hand-baths can be boiled if a large enough steriliser is available. All other baths demand disinfection by scrubbing them with a mixture of domestic cleaning powder and undiluted lysol.

Lotions are often supplied in large open-neck flasks and are used for a number of patients. This is a potent source of cross-infection and is avoided by having half-pint bottles with screw caps reserved for each separate dressing. Irrigation fluids, either for continuous or intermittent treatment, are apt to become contaminated; this is overcome by using a blood transfusion type of apparatus (p. 147).

3. Spread by droplet and contact. Many people carry pathogenic organisms in their throats quite unknown to themselves. Surgeons, nurses, students and porters suffering from recognised throat infections must be banished from surgical wards, and their throats swabbed after recovery. Unrecognised carriers can be rendered impotent as a source of danger if the wearing of masks is made compulsory in surgical wards by everyone concerned in a surgical dressing.

Similarly the smallest septic focus upon the hands of anyone engaged in surgical work must be reported at once and the victim sent off duty.

4. Dust. The morning ritual of ward cleaning is often marked by zeal rather than intelligence. After it is finished a full hour should elapse before dressings are commenced. During their progress blankets should be moved slowly and quietly and no procedure such as dividing or removing a plaster is allowed in the ward. Experiments with various oils for use upon floors are being tried out. So far none have shown any merits which would outweigh their disadvantages.

## BURNS

A burn is an injury produced by excessive heat applied by flame, hot solids, steam and hot liquids (the two latter giving "scalds"), by chemicals having a corrosive action, by certain electrical currents and by over-exposure to sunlight.

## CLASSIFICATION

The classical differentiation by Dupuytren into six varieties was so long and so universally accepted that only great experience of

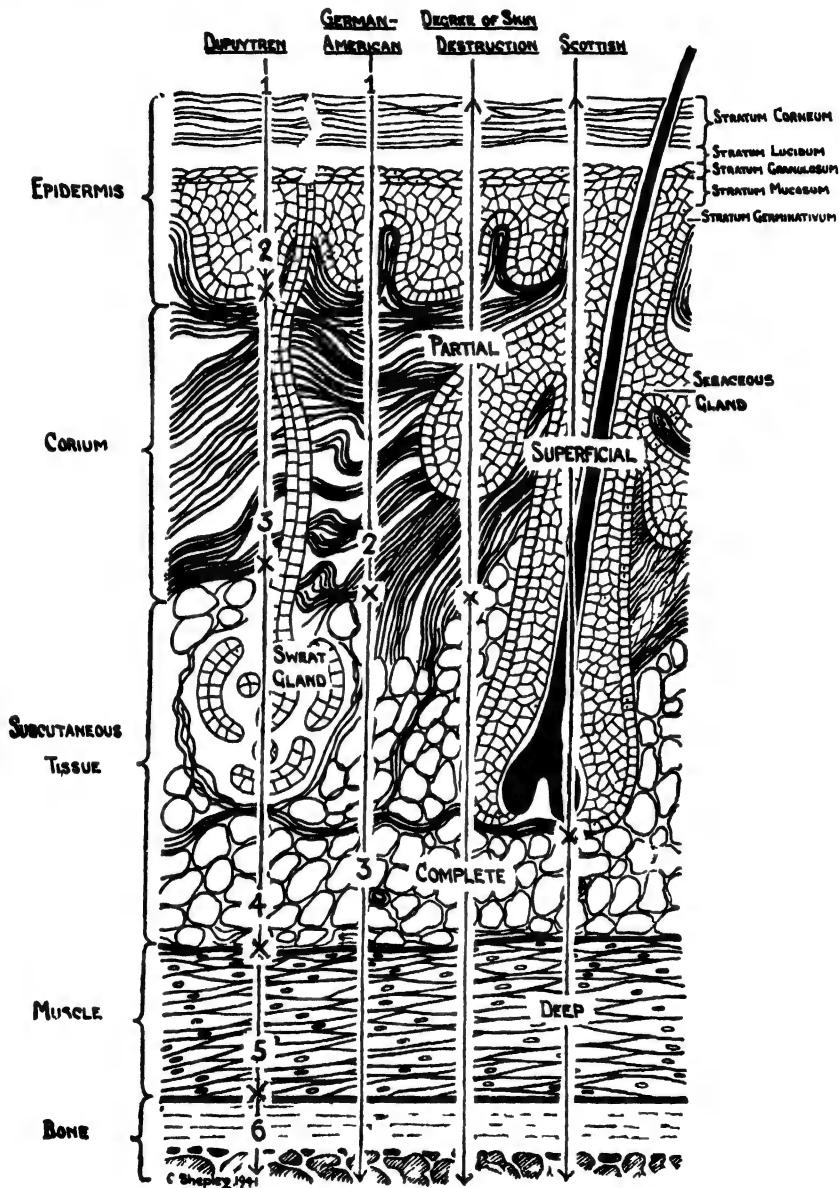


FIG 50

Schematic drawing illustrating various classifications of burns. (By kind permission of H M Stationery Office in Scotland, from the E M S Memorandum No 8)

war burns has shown that a simpler classification is more in keeping both with the clinical picture and the objects of treatment. No final agreement has been reached and none of the following is altogether satisfactory (Fig. 50).

<i>Dupuytren.</i>	<i>American.</i>	<i>Degree of Destruction.</i>	<i>Scottish.</i>
1st degree—erythema	1st degree	...	...
2nd degree—vesication in epidermis			
3rd degree—involvement of corium	2nd degree	Partial	Superficial
4th degree—subcutaneous tissue	3rd degree	Complete	Deep
5th degree—muscle			
6th degree—bone			

Of these the American is the best in that it recognises three groups, but it is apt to carry confusion because of the unequal use of the word "degree." I suggest that the most suitable classification is:—

Simple erythema.

Superficial skin burn.

Deep burn { (a) Whole skin.  
(b) Whole skin and subcutaneous structures.

### PATHOLOGY

**Local.**—The appearance of a burn depends upon the temperature of the agent, duration of application and depth of penetration. Very great heat applied for a fraction of a second may cause a small shallow burn, whereas more than five seconds' exposure to a heat of only 85° C. is likely to destroy the whole thickness of skin.

Heat acts by coagulating the tissue proteins to a variable depth and damaging others more deeply placed. The reaction of the body to this type of injury is that of the classical process of acute inflammation (p. 1), certain features of which play a dominant part in the clinical picture.

**SIMPLE ERYTHEMA** is due to dilatation of the vessels. A red suffused blush is present in the surface layers of the skin accompanied by a barely perceptible swelling. The colour fades slowly and should have disappeared within a week, the slightly damaged cells "scaling" or "peeling" off.

**SUPERFICIAL BURN.**—This can affect any part of the true skin, i.e., both epidermis and corium, but does not reach the subcutaneous fat. It is characterised by the formation of a blister, which appears either within a few minutes or after a few hours. Fluid collects in one of three situations, namely, (1) immediately beneath the *stratum lucidum* as in the hands and feet, (2) in the *stratum germinativum*—the most common site—and (3) between the epidermis and corium, when the papillæ are exposed in the floor of the blister. Exudation continues for thirty-six to forty-eight hours and the vesicle slowly enlarges. Its roof is composed of cells which have undergone coagulation necrosis and as a result it will be shed after the blister has ruptured. Superficial burns of this type are almost painless, but when papillæ are exposed in the floor they become exquisitely painful and tender.

Provided the corium is not completely destroyed, perfect repair will follow, new epithelium springing from interpapillary downgrowths, sweat and sebaceous glands and hair follicles. The time before healing is complete varies with the depth of the burn; when the corium is

not exposed epithelial recovery is complete within ten days, after partial destruction of the corium from three to four weeks must elapse before cure. The newly formed skin is perfect, carrying hairs, sweat and sebaceous glands.

A special type of this superficial burn is produced by intense heat applied for a fraction of a second. The lesion is similar to the above except that the surface layers are instantaneously detached and consequently no blister forms.

**DEEP BURN.**—It is sometimes difficult, if not impossible, to assess the exact depth of a burn by simple inspection, but destruction of the whole thickness of the skin is accompanied by a peculiar nauseating odour. The description given in the Emergency Medical Services Memorandum No. 8 is so clear that I quote it in full :—

“If the injury has been caused by flame, the dead area becomes brown or black, leathery and insensitive. Occasionally the skin is split, exposing subcutaneous fatty tissue, and an oily fluid containing melted fat escapes from the fissures, the patient being pervaded with an unpleasant odour of roasted flesh. Usually the scorched brown epidermis remains firmly adherent; if, however, it can be removed the surface thus exposed is frequently grey and opaque, but occasionally the subjacent corium acquires a mummified appearance, light brown in colour and semi-transparent, and through it the dark blue outlines of thrombosed subcutaneous veins are clearly visible. If the deep injury is produced by scalds, the surface is dull white, marble-like and of slightly sodden appearance. In such lesions of the hands and feet the nails are usually detached. There is no visible exudation of fluid from the surface of deep lesions, but in the subjacent surviving tissue hyperæmia and œdema are present.

“Later reparative processes begin which lead to a separation of the dead tissue; a layer of granulation tissue forms beneath the slough, the deeper parts of which become softened and liquefied by leucocytic digestion, hastened all too often by the action of bacteria which have invaded the demarcating layer. If secondary infection is prevented, spontaneous separation of necrotic material is a slow process and may not be completed for over eight weeks. Since all epithelial elements have been destroyed, the granulating surface is entirely dependent for a new covering upon epithelial growth from the periphery. The resulting scar remains thin, shiny, dry and hairless and subsequent keloid formation is not infrequent.”

**General.**—The pathological changes in the body as a whole are becoming more clearly understood, but active research is engaged in solving still undecided problems. These changes are of the greatest importance since they exercise a profound influence upon treatment. The generalised disturbances produced by a burn necessarily depend upon its extent and depth; it is rare for a patient to survive if more than half the total skin surface is burnt.

**CIRCULATORY FAILURE AND PLASMA LOSS.**—The early changes can be compared to the classical picture of traumatic shock. In and around the affected area an immense number of small blood vessels are damaged. Capillary dilatation follows and great quantities of plasma are poured

out, not only upon the burnt surface and into blisters but also into the tissue spaces of the part. It seems that reabsorption by the lymphatics is so reduced that the amount of plasma saved in this way is negligible. The blood therefore is gradually drained of plasma, the volume of which and the total blood volume steadily fall. At first the vital centres and organs are adequately supplied with blood by peripheral vasoconstriction, but eventually this compensatory mechanism fails. Reduced blood volume with vasoconstriction is shown by a slowly falling systolic and a rising diastolic blood pressure ; failure of compensation introduces a fall of both readings, which may be slow and persistent but is apt to be sudden and severe. As the pressure falls all the vital organs suffer from anoxia and a stage is reached when all hope of recovery is lost.

**CHANGES IN THE BLOOD.**— These are of little practical importance, being somewhat variable and slight in degree. They may be summarised as increased viscosity of the blood, hemoconcentration (hemoglobin may rise to 120 per cent.), a transference of sodium and chlorides from plasma to cells and of potassium from cells to plasma, a rise in non-protein nitrogen and a lowered  $\text{CO}_2$  combining power.

**TOXÆMIC PHASE.**— In the majority of non-fatal cases shock passes off, the circulation recovers and general systemic complications are infrequent unless bacterial invasion occurs in the burned area. Rarely, however, severe constitutional symptoms appear which have many points of resemblance to the "crush syndrome." This phase is marked by pyrexia, mental disturbances, oliguria, albuminuria, a fall in plasma protein, a rise in blood urea and occasionally jaundice. Death is likely to follow upon the renal and hepatic damage. The pathology of this condition is not known ; it is possibly due to the absorption by the blood of large quantities of autolytic products from the burned area.

**BACTERIAL INFECTION.**—The nature of the injury (in spite of the sterilising effect of heat), the circumstances of its infliction and the problems associated with both first aid and later surgical treatment make bacterial invasion almost inevitable ; fortunately, however, the presence of organisms does not necessarily have much effect upon either local or general symptoms. Nevertheless, secondary infection is to be regarded as a grave complication, since septicæmia may easily follow when a large surface provides so extensive an area for absorption. Many organisms are found : *S. albus*, *S. aureus*, *Streptococci*, *B. coli*, *B. subtilis*, *B. proteus* and *B. pyocyaneus*. While the others may delay wound healing, hæmolytic streptococci are responsible for the more severe complications.

### CLINICAL PICTURE

Symptoms can be classified under five headings :-

- |                     |                        |
|---------------------|------------------------|
| 1. Primary shock.   | 3. Acute toxæmia.      |
| 2. Secondary shock. | 4. Bacterial invasion. |
| 5. Healing.         |                        |

Of these acute toxæmia is seen in very few people, but in spite of improved methods of treatment bacterial invasion remains comparatively frequent.

**Primary Shock** is of immediate onset and short duration (under two hours). It is closely akin to the vasovagal syndrome. Patients complain of cold, thirst, nausea but of little pain, though the memory of the agony of the actual burning remains. The forehead is cold and clammy, face cyanosed or grey. Pulse rate drops to about 60, blood pressure falls to 85/50 and the capillary hæmoglobin is 115 per cent. This combination of slow pulse, low pressure and hæmoconcentration clearly defines primary from secondary shock, and calls urgently for resuscitation therapy.

Two hours after injury this initial shock is passing off and the general condition improves. The pulse rises to 90, blood pressure is up to 105/80 and hæmoglobin is 125 per cent.

**Secondary Shock.**—This improvement is transient and within the next hour the patient is becoming restless and anxious. So great is the thirst that it cannot be assuaged, more especially as vomiting occurs at each attempt at swallowing. The face is grey, cold and clammy, pulse rate is mounting from 110 to 120, blood pressure is falling and still further hæmoconcentration follows. If treatment is not available at this stage a fatal issue is inevitable.

Under usual conditions, however, treatment will have prevented any severe degree of secondary shock and the clinical picture is different. Resuscitation has been started in the period of primary shock and the general condition and blood pressure have returned so nearly to normal that local treatment of the burned area is undertaken. During the twenty-four hours that follow careful readings of pulse and blood pressure are recorded hourly and a decline will be observed on two or three occasions. Each fall will be controlled by plasma transfusion and by the end of twenty-four to thirty-six hours the general condition of the patient will be stabilised, and in many cases no further complication arises and a smooth, if tedious, convalescence lies ahead.

**Acute Toxæmia.**—A small number of patients will suffer from toxæmia. After thirty-six to forty-eight hours the temperature rises to 102° or higher, pulse is about 100, blood pressure 100/65 and hæmoglobin 90 per cent. The patient becomes restless and mentally confused and vomiting occurs. The urine output is scanty and blood urea rises sharply from 150 to 200 mg. If recovery ensues jaundice may appear, but other toxic manifestations slowly disappear after seventy-two hours.

**Bacterial Invasion** may occur at any time, but is apt to be most severe when the sloughs on deep burns are in process of separation. Toxæmia is usually of moderate intensity, but its duration of one to four weeks leads to prostration, emaciation and anæmia. In a small number of patients septicæmia occurs and death ensues or a prolonged illness with metastatic collections of pus complicates an already grave condition.

**Period of Healing.**—A time arrives when all sloughs have been shed and all deep areas are covered with healthy granulation tissue. This "turning of the corner" is so abrupt and dramatic that the general condition alters within twenty-four hours. The patient sleeps profoundly, **eats** voraciously, puts on weight, regains a normal blood

picture and undergoes a complete mental change. Healing goes steadily forward and, although periods of sluggishness and stagnation occur, epithelialisation is finally completed.

**Overlapping of the Phases.**—The above description presents clear-cut phases, but these overlap so that it is not always quite obvious what the exact state of affairs is. This is illustrated by the following scheme from the E.M.S. Memorandum No. 8 (Fig. 51).

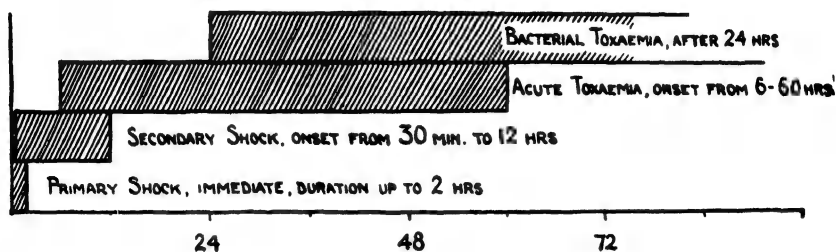


FIG. 51

Schematic drawing showing the overlapping of the stages of shock and toxæmia.  
(By kind permission of H.M. Stationery Office in Scotland, from the E.M.S. Memorandum No. 8.)

## TREATMENT

### GENERAL

**A. On Reception.**—Immediately on arrival a burned patient is put to bed and swathed in blankets while a large radiant-heat cradle is placed overall. No attempt to remove the clothing or to assess the extent of the burn is permissible at this stage. If morphia has not been administered before arrival, an intramuscular injection of  $\frac{1}{4}$  gr. is given forthwith. Pulse and blood pressure readings are taken at ten-minute intervals to determine the type and degree of shock. Unless the patient's condition obviously demands resuscitation, no further interference is desirable for half an hour, at the end of which warmth and morphia are taking effect. At this stage a brief inspection is carried out to define the surface extent of the burn.

**B. Stage of Primary Shock** is treated by morphia, heat and rest. During this period our attention is directed towards the prevention of secondary shock, if this is possible. No local treatment of any sort is permissible until this has been done.

**C. Stage of Secondary Shock.**—Prevention and treatment of secondary shock are identical: our study of pathology has taught us that the volume of the circulating blood and its plasma content must be restored as rapidly as possible. For this reason transfusions of plasma are given, whole blood and normal or glucose saline being equally contraindicated, the former leading to increased hæmoconcentration and the latter to a serious depletion of plasma protein owing to increased capillary permeability.

Suprarenal extracts such as cortin or similar synthetic compounds are powerful adjuvants to plasma transfusion, and oxygen inhalations through a B.L.B. mask are helpful (p. 161).



**D. After Local Treatment.**—For twenty-four hours repeated falls of blood pressure are apt to occur and regular pulse and pressure readings will be taken every half-hour to detect them. Each will be treated by plasma transfusions and during this period two or three will probably be needed.

**E. Stage of Toxæmia.**—Since its etiology is obscure, the treatment of toxæmia is unsatisfactory. There is some evidence that the incidence is less in cases treated by coagulation therapy. Glucose should be given generously by mouth in order to prevent liver damage, and cortin combats circulatory failure.

### LOCAL

Our study of pathology of burns suggests that our objects in treatment are to—

1. Cleanse the burned area and its surroundings and to prevent infection ;
2. Cover all exposed sensory nerve endings to reduce pain and shock ;
3. Seal all exuding surfaces to prevent external loss of plasma ; and
4. Provide ideal conditions for healing.

Coagulation therapy fulfils all these requirements most admirably. Its few disadvantages were given such undue prominence during the first year of this war that there was a real danger of its falling into disrepute. I cannot emphasise too strongly that, except for certain burns of special areas, coagulation is superior to all others and must be considered as the standard method of treatment.

**A. Preparation.**—There is a certain similarity between a superficial burn and an open wound. Both are potentially infected and just as the latter has a "safe" period of from eight to twelve hours, so a burn can be saved from sepsis if treated within twenty-four or at most thirty hours.

When shock has been controlled the patient is taken to the theatre and given a general anæsthetic, gas-oxygen-ether being the method of choice. All clothing is carefully removed and the patient transferred to the operating table. Under no circumstances should large areas be exposed at a time, but in extensive burns small sections should be dealt with in succession. Every blister is punctured and all detached epithelium removed by gentle swabbing with gauze and normal saline. Rough handling and the use of a scrubbing brush are absolutely forbidden as are all irritating or dehydrating antiseptics.

**B. Coagulation.**—The burned area is now sprayed with 15 to 20 per cent. solution of tannic acid or with 5 per cent. tannic acid followed by 10 per cent. silver nitrate. This is continued until the surface turns brown. Whilst this spraying is in progress the whole area is being dried by a stream of hot air from an electric hair drier, to the outlet of which a filter must be fitted to trap organisms which would otherwise be driven on to the burn in large numbers. When the operation is completed all burned areas are covered with sterile towels and the patient returned to the ward. If the position and extent of the damage



permits, the whole surface is exposed to radiant heat beneath a special cradle and no clothes or dressings are allowed to touch it. Drying is continued for twenty-four hours, after which the pellicle has turned black (Fig. 52), is firm and securely adherent to underlying tissues. Attention is directed to preserving it from cracking, anticipating any premature separation and to the recognition of sepsis below it at the earliest moment. Any fresh blisters which form at or near the periphery are pricked and tanned as are all fissures. Eventually the pellicle will separate normally after epithelium has grown in beneath it, this process being a perfect example of the classical "healing beneath a scab."

In extensive burns it is impossible to protect every part from pressure. Patients should lie upon the least damaged area which is coagulated and then protected by a gauze dressing: in some cases it



FIG. 52

The appearance of an extensive burn of the back after the application of tannic acid.

may be preferable to use a tannic acid jelly from the outset for these areas.

**C. Dye Solutions.**—The effect of certain dyes is not truly coagulative but rather antiseptic. They form a crust of dried exudate which acts in a manner somewhat similar to a true pellicle. They are used either alone or in conjunction with the coagulants. Crystal violet and brilliant green may be employed separately, but are usually combined in the form of "triple dye":—

Crystal violet	.	.	0.5 per cent. watery solution	} Equal parts.
Brilliant green	.	.	0.1 " " "	
Acriflavine	.	.	0.1 " " "	

This treatment is in special favour for burns of the face and hands in which coagulation therapy is contraindicated (see below).

**D. Deep Burns.**—The difficulty of assessing accurately the depth has been stressed above. For this reason the whole area is coagulated although theoretically a deep burn has already provided its own protective coagulum since the skin has undergone coagulation necrosis

Coagulation, however, ensures that all surface lesions are adequately protected and all areas of exudation are sealed. After a long period the dead skin separates and finally healing may be accelerated by skin grafting.

**E. Established Sepsis.**—If more than thirty-six hours have elapsed since injury before the patient reaches hospital, the burned areas are infected and coagulation therapy is no longer safe. Again, apparently clean burns may become grossly septic beneath a pellicle which will be rapidly cast off as a result. Such septic burns require different treatment and many methods are available, but irrigation in conjunction with a Stannard envelope is most satisfactory for the limbs; on the trunk, head and neck other means must be used.

The Stannard envelope is made of waterproofed silk and is designed in various shapes and sizes for the upper and lower limbs (Fig. 53).



FIG. 53

The envelope method of treating burns. Note the entrance and exit diverticula for irrigations. (*Surgery of Modern Warfare.*)

Its upper end is sealed to the skin by adhesive strapping and the burned area is completely exposed within it. Irrigation is carried out with 1:20 Milton (electrolytic sodium hypochlorite) at 100° F. for twenty minutes three times a day. After each treatment the contents are drained away and the envelope filled with oxygen and both inlet and outlet tubes securely clamped. Results have been excellent and this method is a valuable addition to burn treatment.

Saline packs are useful for septic burns of the trunk. The damaged area is covered with tulle gras and thick pads of gauze soaked in normal saline. They must never be allowed to become dry; either they must be changed every two hours or some method of drip feed installed to maintain their moisture.

Oily dressings such as cod-liver oil emulsion and glycerin-sulphapyridine paste are useful in deep burns, and in encouraging granulating areas to heal or to prepare them for grafting.<sup>1</sup>

<sup>1</sup> After this was written and already in the press, Lieut.-Col. Porritt has returned to England and tells me that in the Middle East he and his colleagues have had excellent results from glycerin-sulphapyridine pastes for all burns. In his opinion this method is equal, if not superior, to coagulation therapy.

Closed plaster has been applied to burns, but at present there is not sufficient evidence for us to assess its value.

### BURNS IN SPECIAL SITES

**Burns of the Fingers and Hand** present special problems. A coagulum contracts on drying and when surrounding a finger completely is liable to constrict its digital vessels and so imperil its nutrition. Further on the dorsal aspect of the hand the skin is thin, subcutaneous fat is absent and tendons, joint capsules and bones are highly vulnerable. Contractures will occur unless special attention is directed to position in treatment and re-education afterwards.

The dangers of coagulation are possibly overrated, but it is wise to forbid this method in this situation. Less severe burns are best treated with triple dye while others respond most satisfactorily to irrigation with a Stannard envelope. If dressings are applied the hand must be placed in the position of function (Fig. 54).

**Burns of the Face** demand special notice because of the likelihood of unsightly scars and contractures affecting the eyelids, lips, ears and nose. Sepsis is apt to give trouble especially if the burn has extended into the hairy scalp. Superficial burns which do not involve the eyelids, lips or ears may safely be coagulated, but it is essential that the scalp be shaved for at least 2 in. beyond the limit of the burned area. Some authorities prefer triple dye to tannic acid even in these "safe" areas.

Burns of the eyelids, lips and ears are best dealt with by an oily application, *e.g.*, cod-liver oil, glycerin-sulphapyridine paste or soft paraffin.

Deep burns may also be coagulated, but it is important that they should be rid of their pellicle or slough within four weeks and the exposed surface immediately prepared for grafting. It is better to anticipate contractures and scarring as far as possible than to be faced with extensive plastic operations later.

**Burns of the Eyeball** are treated with liquid paraffin drops as an immediate first-aid measure and, later, attention is directed to the prevention of injury to the cornea and to adhesions between the globe and eyelids.

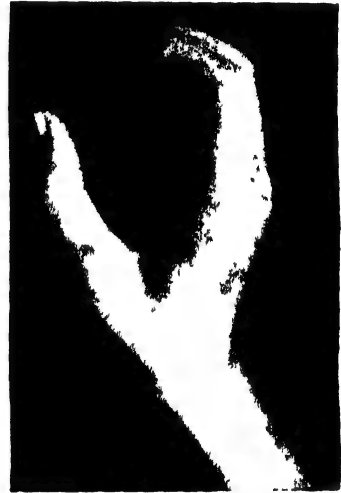


FIG. 54

The position of function.

### FROST BITE

Frost bite is actually a localised form of gangrene and may be produced either by direct action of cold, leading to vasospasm, or to the effects of too rapid thawing, when excessive exudation causes obliteration of the vascular supply of the part. The most exposed and most distal parts are naturally those usually affected (nose, ears,

fingers and toes), and children and the aged are the worst sufferers. The direct cold type is a dry painless gangrene, affecting a larger area on the surface than in the deeper tissues. The part becomes white and waxy and shrivels up, then later turns black and is separated and cast off.

In the congestive type there is a great deal of pain and inflammation followed often by vascular thrombosis. Recovery may occur without loss of tissue, but superficial ulcers are usual.

*Treatment* is prophylactic, being the avoidance of or protection against exposure and the slow and careful thawing of a frost-bitten part. If gangrene occurs, the part is kept warm and dry until a satisfactory line of separation develops. Trench foot is prevented by cleanliness, drying of the feet night and morning, the application of talcum powder and a change of warm socks twice a day.

### ELECTRICAL INJURIES

These conditions include lightning stroke, electrical burns and injuries from X-rays and radium.

If lightning stroke is not immediately fatal its effects are similar to those of severe electrical shock. In lightning shock it often happens that, without any external signs on the surface of the body, the clothes have been torn off, bones broken and viscera ruptured. External markings, when present, are usually where some piece of metal, *e.g.*, a stud, was in contact with the skin, and are often arborescent in character.

The severity of electrical burns varies not so much with the voltage as with the amperage. Alternating currents are more dangerous than constant, and length of contact is an important factor. Some tissues conduct electricity better than others, *e.g.*, cerebrospinal fluid, blood and the viscera. Pregnant women, sleepers and those actively expecting a shock are less susceptible to the effects.

The symptoms are (1) pain which is agonising and generalised and is often accompanied by such sensory changes as blindness, deafness and hallucinations; (2) all sense of time is lost and the patient feels as though the body had shrunk; (3) unconsciousness usually follows, associated later with some degree of retrograde amnesia; (4) burns, which are characterised by their unexpected depth in comparison to their surface extent, by the friability of the vessels in the neighbourhood, by their tendency to spread and by their very tardy healing.

Secondary effects include reactionary hæmorrhage, falling out or blanching of the hair, longitudinal fractures of bone, œdema, optic atrophy and cataract, and both organic and functional changes in the nervous system. The cause of death is a combination of respiratory failure from inhibition of the medullary centres, and cardiac failure from ventricular fibrillation.

*Treatment* comprises removal of the patient from the source of the shock, immediate and prolonged artificial respiration, and general measures as for a serious head injury. Lumbar puncture is said to be particularly efficacious. Opinions vary as to the treatment of the

burn, some surgeons advising conservative treatment, others radical excision and grafting.

### RADIUM AND X-RAY BURNS

These result from over-exposure during therapy. It must be realised that to obtain a maximum therapeutic effect the radiologist must produce a mild burn, such a full dose being known and referred to as an *erythema* dose. After the redness has died away, the skin may become dry, hard and itchy, and surface desquamation ensue. Hair is lost and the nails become brittle. Some degree of pigmentation may persist and the condition tends to relapse. Such an X-ray dermatitis may progress until it assumes the characters of a low-grade squamous-celled carcinoma. Various tissues react differently to X-rays, the scalp being very resistant while the normally moist areas (axillæ, groins, vulva, etc.) are peculiarly sensitive.

Longer and more intense exposure to both X-rays and radium produces a definite necrosis. These ulcers are very painful and form large sloughs which are slow to separate and slower still to heal.

*Treatment* consists in complete excision if possible; otherwise diathermy, ultra-violet light or short-wave therapy may assist in the separation of the slough and the healing of the wound. Grafting should be done as soon as the state of the wound permits.

### CHEMICAL INJURIES

Strong caustics and corrosives, particularly acids, produce localised necrosis if they come in contact with the skin. The degree of tissue destruction depends on the strength of the solution and the length of application. Such burns are characterised by considerable pain, excessive slough formation and slow healing, followed by marked contraction of the scar. Treatment consists in the application of antiseptic dressings and prevention of contraction by splints and skin grafting.

R. M. HANDFELD-JONES.

## CHAPTER VIII

### HÆMORRHAGE AND SHOCK

#### HÆMORRHAGE

**T**HE term hæmorrhage implies a loss of blood from the vessels, and in that there are three types of blood vessels, so hæmorrhage is classified as being arterial, venous and capillary. Hæmorrhage is usually due to trauma, but it may also be the outcome of certain constitutional diseases, either congenital in origin, such as hæmophilia, or acquired, as in purpura, leukæmia and scurvy, or be secondary to pathological changes occurring in the vessel walls, *e.g.*, aneurysm or atheroma.

Any of the three types of hæmorrhage may be further subdivided into external and internal.

**External Hæmorrhage** may occur from the skin as the result of wounds, or from one of the natural orifices of the body. In this respect certain terms are in common use: *epistaxis* (blood from the nose), *hæmoptysis* (blood coughed up from the lungs or respiratory passages), *hæmatemesis* (blood vomited up from the stomach), *melæna* (blood passed per rectum) and *hæmaturia* (blood voided in the urine).

The blood in epistaxis is bright red, as is that of hæmoptysis, having been recently oxygenated. This recent admixture with air also accounts for its being frothy. The blood in hæmatemesis may have been swallowed from the nasopharynx before being vomited. Its colour depends on the time it has been in contact with gastric secretions, prolonged action of which produces dark-brown clotted fragments—the coffee grounds vomit. In hæmaturia the colour depends on the site of the bleeding and its rapidity. In melæna the blood has been altered by the intestinal secretions so that the stools are coloured black, but bleeding occurring low in the rectum is bright red.

**Internal Hæmorrhage** may be of two varieties, the subcutaneous, which is more or less obvious, and the deep or concealed, which occurs in the deeper tissues or cavities of the body. In both groups there are several terms which must be defined. *Extravasation* of blood implies an escape into the submucous, subcutaneous or subserous tissues; almost synonymous is the term *ecchymosis*, although in this case the blood has usually worked towards the surface from the deeper planes. The end result in both these types is called a *bruise*. If the blood poured out is limited by tissue planes and is in any quantity, the resultant collection is known as a *hæmatoma*, whilst small multiple extravasations beneath the skin or other lining membrane are termed *petechiæ*.

Concealed hæmorrhage occurring into the pleural cavity is called *hæmothorax*; into the peritoneum, *hæmoperitoneum*; into the tunica vaginalis of the testis, *hæmatocele*; into a joint, *hæmarthrosis*; into the fallopian tubes, uterus and vagina, *hæmatosalpinx*, *hæmatometra* and *hæmatocolpos* respectively; and into the spinal cord, *hæmorrhachis* if extradural and *hæmatomyelia* if intramedullary. To bleeding into the substance of the brain the term *apoplexy* is often applied. These conditions are fully described in the chapters concerned.

### THE CLINICAL PICTURE OF HÆMORRHAGE

**Local.**—The signs of external hæmorrhage are obvious and will be referred to in detail in the description of the three main types of bleeding (see below).

In internal hæmorrhage local signs are absent, but the history and general symptoms combined with signs of fluid in an internal cavity will assist diagnosis. In this respect it must be noted that the quantity of blood poured out will affect the length of time it remains fluid. In small extravasations, as, for example, in the tunica vaginalis, fluctuation may be obtained in the early stages and this, combined with an absence of transillumination, would strongly suggest a hæmorrhage. Fluid in the pleura and peritoneum may be recognised by certain clinical signs, but in general it will be seen that these signs are simply those of an effusion, which can be identified as blood only if the general clinical picture suggests this possibility.

**General.**—Constitutional effects naturally vary with the amount of blood lost and the rapidity with which it is shed. If half the total volume is lost in a very short time, the result is fatal, whereas a gradual loss of 80 per cent. is still compatible with life. In infants and the aged the effects of hæmorrhage are more marked, but in the former recovery is very rapid; as a general rule, women suffer less severely from considerable loss of blood than men, and in pregnancy their powers of recovery are remarkable. The presence of certain accessory factors render the effects of hæmorrhage more serious. In anæmia the vital hæmaglobin content is already low; shock, which so often accompanies bleeding, is responsible for a continued depression of blood pressure which prevents the usual post-hæmorrhagic revitalising rise; jaundice due to the presence of bile salts leads to a diminution in the normal clotting power of the blood; while sepsis, septicæmia, hæmophilia, scurvy and purpura are all examples of diseases in which a small hæmorrhage may have very serious results.

Massive hæmorrhage may lead to death in a few minutes from syncope. In non-fatal cases two factors are responsible for the general symptoms, viz., loss of body fluid (dehydration) and loss of hæmoglobin (the oxygen-carrier). Dehydration in the normal course of events is rapidly made good by replacement with tissue lymph, but red blood corpuscles are regenerated slowly. Cerebral anæmia may result in unconsciousness, usually temporary (a fainting attack), but subsequent effort may cause a repetition. The patient is very pale, cold and clammy, the red margins of the lips, the nail beds and the inner

lining of the lower eyelids being the tissues to give the earliest and most valuable indication of loss of blood. The pulse is rapid, feeble and irregular, and the blood pressure falls rapidly, but should soon start to recover after the bleeding has ceased, owing to reflex peripheral vasoconstriction and to the influx of tissue fluids into the blood stream. Respiration is rapid and shallow with long gasping sighs at intervals, as the patient strives to obtain more oxygen. This is described as *air hunger*, and is accompanied by marked restlessness. Great thirst is complained of, as are tinnitus or even deafness, flashes of light or dim vision, and severe headache from cerebral anæmia. The temperature is subnormal.

**Natural Arrest of Hæmorrhage.**—It is well known that many minor hæmorrhages and not a few major ones will stop spontaneously. The essential factor in bringing this about is the coagulability of the blood. Clot is formed of fibrin, which in the early temporary stages of arrest is soft and jelly-like and contains many red blood corpuscles. This "red clot" is formed both within and outside the damaged vessel, *i.e.*, internal and external clots. The fibrin is produced by the action of thrombin on the fibrinogen of the circulating blood, and this thrombin is the result of interaction between the calcium salts of the blood with a substance called prothrombin. Whether prothrombin action results from its activation by the thrombokinase freed from damaged cells, or from neutralisation of a normally present antithrombin by a substance named thromboplastin is not yet definitely decided.

Subsidiary factors which influence the formation of a clot are retraction of the intima and contraction of the media. The absence of this factor in partial wounds of vessels or in incised as against lacerated wounds accounts for the more prolonged hæmorrhage met with in the former case. If hæmorrhage is considerable a fall of blood pressure occurs, and anæmia of the medullary cardiac centre leads to diminution in force of the heart beat. This produces a slow blood stream and an increased viscosity, whilst, finally, the compensatory back flow of tissue fluid into the vessels to replace the blood loss brings with it copious supplies of fibrinogen.

The red clot formed by this combination of factors soon becomes infiltrated with platelets and white corpuscles, and it is this "white clot" which is organised by simple plastic inflammation in a similar manner to an inflammatory exudate, so producing permanent arrest of hæmorrhage by fibrosis.

### GENERAL TREATMENT

General treatment consists essentially in arrest of the bleeding, but many patients after a severe hæmorrhage are too ill to withstand any surgical measures. The methods of general treatment will therefore be described first.

Complete rest and quiet are the first essentials in every major hæmorrhage. The head should be kept low to ensure that the vital medullary centres get as great a share as possible of what blood there is. This may be achieved by removing all pillows, raising the foot of the



bed on blocks and, in very severe cases, by bandaging the limbs firmly from the periphery towards the trunk. Patients must lie as quiet and motionless as possible; warmth, supplied by a radiant heat cradle or by carefully placed hot-water bottles, is an immense advantage, and certain drugs are of value. Morphia ensures that rest which is essential to the recovery of vitality and to the arrest of the hæmorrhage by clot formation. To increase the coagulability of the blood, calcium lactate may be given either intravenously (gr. v of a 10 per cent. solution), or rectally in gr. xv doses, and 25 c.c. of horse-serum or 2 c.c. of hæmoplastin given intramuscularly serve the same purpose. The general vasoconstrictors, adrenalin (Mv of a 1:1000 solution of adrenalin chloride) or pituitrin (1 c.c.) subcutaneously, will help to stop bleeding, but as they also raise the blood pressure, they may neutralise their own good effects. Stimulants, such as strychnine, have the same disadvantage, but may perforce have to be given in cases of syncope.

The remaining methods of general treatment involve the replacement of either the fluid content of the blood or of blood itself. The latter is obviously the method of choice, but certain technical difficulties and certain possible sequelæ make it applicable only under favourable conditions.

INFUSION may be intravenous, rectal (proctoclysis), intramuscular or intradermal (hypodermoclysis). In intravenous infusion, the most rapid method, some relatively superficial vein (the median basilic or cephalic at the elbow, the internal saphenous at the ankle, and the superior longitudinal sinus in infants) is selected, and if sufficiently obvious is pierced by a medium-bore needle subcutaneously. If the vein is impalpable, a transverse incision is made over its course and the vessel exposed. It is then tied distally and a glass or metal cannula inserted proximal to the ligature through an oblique slit in the vein wall (Fig. 55). Leakage is prevented by a second ligature tied tightly round the cannula, and when the infusion is completed the cannula is withdrawn and the ligature tied. Whether a needle or cannula be used it is important to see that it is full of fluid before insertion to avoid the introduction of air bubbles into the circulation. The danger of air embolism, however, from the injection of small bubbles of air into a peripheral limb vein is probably very slight. The fluid used is usually normal saline (3i of sodium chloride to 1 pint of sterile water), though a 6 per cent. solution of gum acacia in saline is thought to have more lasting effects. The fluid should be warmed to 110° F. and introduced slowly. Two or 3 pints can be given more rapidly at first, but later an apparatus providing a "continuous drip" is to be preferred (Fig. 56).

Absorption of fluid from the bowel after rectal infusion varies considerably with the individual and with the contents of the bowel. Ten pints of saline or water can be given in twenty-four hours by this route, a continuous-drip apparatus (*e.g.*, Murphy's) being used. Absorption is not so satisfactory when a large quantity is run into the rectum rapidly.

In cases of great urgency, and if the operator is single-handed, a needle may be introduced beneath the skin into the loose connective

tissue of the submammary region, buttock, thigh or abdominal wall and connected to a receiver (about 5 ft. above the patient) containing normal saline. This will provide a steady if somewhat slow absorption of fluid. Excessive subcutaneous tension and a fluid temperature above 110° F. must be avoided or sloughing will occur at the site of infusion. Absorption is safer and more certain if saline is introduced into large muscles.

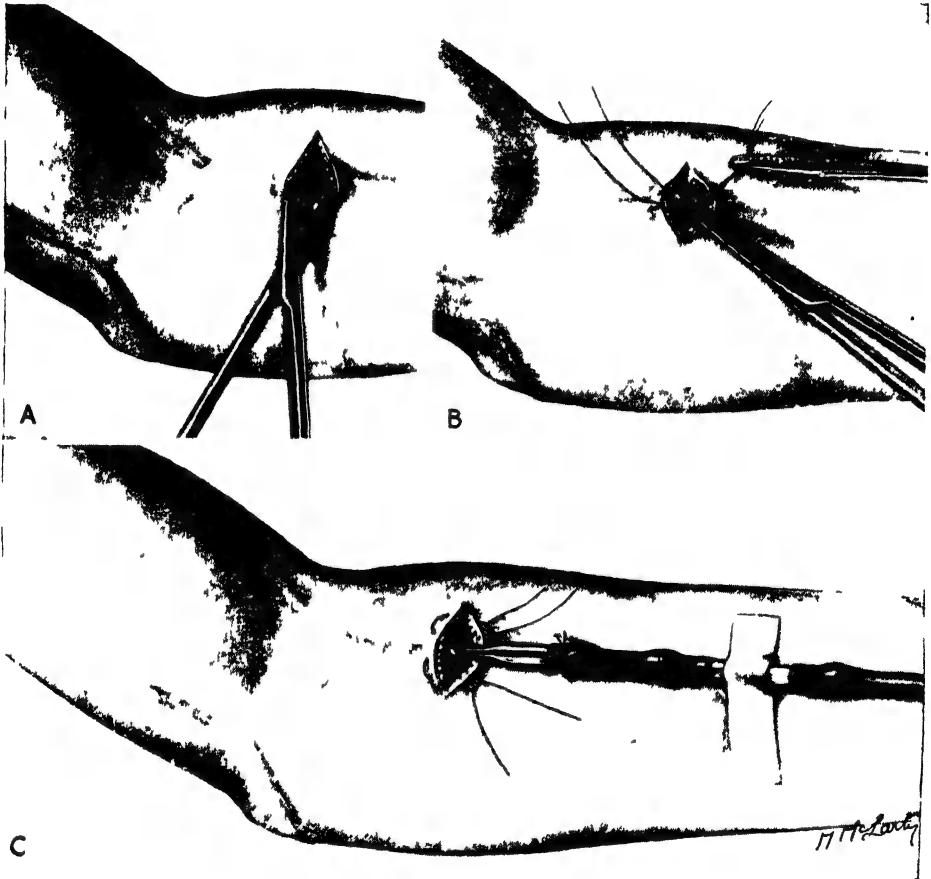


FIG. 55

Drawing to show the method of exposing the vein and of tying in the cannula. (Farquharson)

### BLOOD TRANSFUSION

The first essential is to find a prospective donor whose blood is safely miscible with that of the patient. Blood contains bodies known as agglutinins and hæmolysins, and in incompatible bloods the donor's red cells may become agglutinated by these substances in the recipient's serum. This agglutination may be, but is not necessarily, followed by a breaking up of the donor's corpuscles, *i.e.*, hæmolysis. It is necessary therefore before transfusions to test the donor's corpuscles against

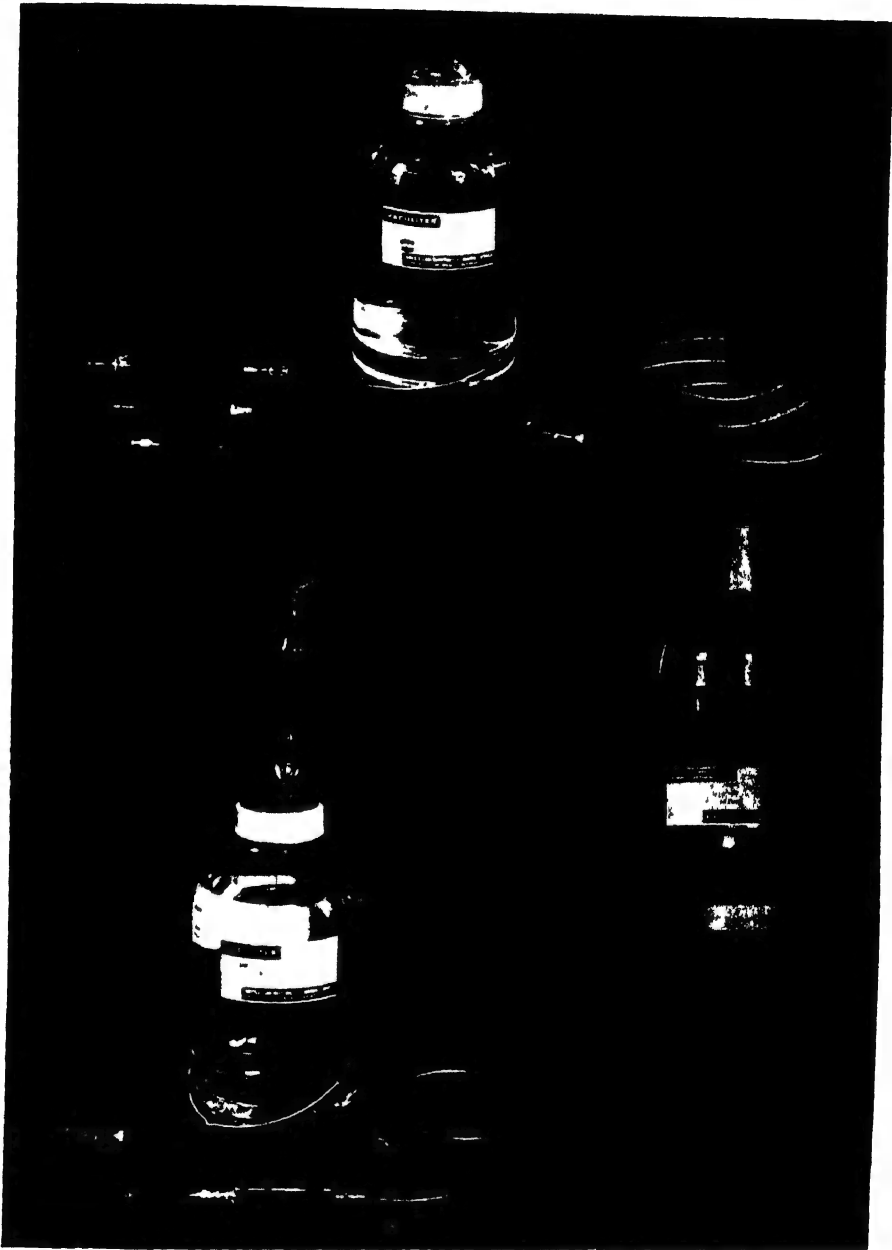


FIG. 56

"Vacoliter" saline infusion apparatus and method of use. (*Fa-guharson.*)

- A, 1000-c.c. flask with delivery apparatus, consisting of dropping tube, 6 ft. of rubber tubing and clip, glass connection and intravenous needle.
- B, The metal cap is removed and the rubber diaphragm cut away. The dropping tube, which has been connected to the rest of the delivery apparatus, is plugged into one of the two holes in the rubber bung, indicated by an arrow. The other hole carries a glass air inlet tube already fixed in position.
- C, The flask is inverted and suspended by the handle, ready for use.

the recipient's plasma. The reverse is of small importance, because of the very rapid dilution of the donor's plasma once it gets into the recipient's circulation.

Human beings fall into four "blood groups." In the past there have been various methods of group nomenclature, but to-day the Landsteiner classification finds universal acceptance. The following table gives the reactions of the various groups and their percentage occurrence :—

Donor's Cells.	Recipient's Serum.				Percentage Occurrence.
	Group AB.	Group A.	Group B.	Group O.	
Group AB . .	-	+	+	+	5 per cent.
Group A . .	-	-	+	+	40 "
Group B . .	-	+	-	+	10 "
Group O . .	-	-	-	-	45 "

+ Agglutination. - = Non-agglutination.

It will be seen that a Group AB serum can receive blood from any donor (universal recipients), whilst a Group O donor can give to any other group without agglutination (universal donors). In most cities to-day and in many hospitals there are efficient transfusion services by which potential donors of known group, tested blood and robust health are made available as required. But even when a donor of known group can be supplied, compatibility tests between donor and recipient should be carried out before transfusion, except in most dire emergencies. In such cases a universal donor should be used, except for repeated transfusions or in the case of a late pregnancy or the puerperium.

**Group Tests.**—Specific tests of donor against recipient can be carried out by direct or indirect methods. In the indirect technique, the corpuscles of donor and recipient are tested separately by obtaining a drop of blood (from the lobe of the ear or the finger). This is mixed with a drop of fresh stock test sera of Groups A and B bloods, the appended table showing the possible results :—

	Corpuscles.			
	AB.	A.	B.	O.
Serum A . .	+	-	+	-
Serum B . .	+	+	-	-

+ = Agglutination. - = Non-agglutination.

If the diluted blood is mixed with the stock serum on a glass slide or white tile, agglutination can be readily observed by the appearance of fine dark "sand" in the drop. This usually occurs in a few minutes, but delayed agglutination is one cause of false grouping.

In the direct method a few cubic centimetres of the recipient's blood are withdrawn from a vein, placed in a test tube and allowed to clot. A drop of the resultant serum is then mixed with a drop of the potential donor's blood and agglutination looked for.

**Effects of Incompatible Transfusion.**—Administration of a grossly incompatible blood, *i.e.*, of a wrong group, produces agglutination and lysis of the red cells of the donor's blood. This grave—and easily avoidable—error leads to disaster. In a number of patients death follows within an hour or in the course of a few days: others survive after a serious illness. The first symptom occurs while the transfusion is in its earliest stages, the patient complaining of severe pain in the kidney area of both loins. Should this occur transfusion must immediately be stopped. A rigor, difficulty in breathing and circulatory failure follow rapidly and later hæmoglobinuria, jaundice, urticaria and symptoms due to small emboli in the brain, heart muscle and gastro-intestinal tract make their appearance. Should the patient survive this initial “hæmolytic shock,” a period of renal failure has to be overcome before convalescence is achieved.

Unpleasant reactions and even tragedies may occur, however, in cases of correct grouping, but in the absence of direct matching of recipient and donor. These complications are practically unknown in single transfusions except late in pregnancy, but multiple injections for such things as severe war wounds always carry this risk. We must understand why these phenomena happen.

**SUBGROUPS  $A_1$  AND  $A_2$ .**—Groups AB and A are subdivisible into subgroups  $A_1$ ,  $A_2$ ,  $A_1B$  and  $A_2B$  with the result that in rare instances specific agglutinins  $a_1$  and  $a_2$  are formed in considerable amount. The importance of these subgroups will be understood when it is realised that the agglutinins thus produced not only affect their opposites in their own groups but are also anti-O, the universal donor.

**FACTORS M, N AND P.**—These inherited factors are of no practical significance in intragroup incompatibility, only four cases having so far been recorded.

**RHESUS FACTOR.**—Recently the Rhesus factor has been shown to be present in 85 per cent. of human bloods. The remaining 15 per cent. Rh— contain no antibodies. As a result after repeated transfusions, or from a foetus in utero, these people may develop the corresponding agglutinin. As Whitby points out the detection of the Rh factor requires special technique, but for repeated transfusions and for pregnant women Rh— donors must be used. It is probable that this factor is responsible for the great majority of hæmolytic reactions in homologous Landsteiner group transfusions, especially after using a Group O donor.

**COLD AGGLUTININS** are occasionally the source of great confusion and difficulties both in group testing and treatment. Cold agglutinin is active only at or below 30° C. Whitby reports cases in which a patient is said to be Group AB, whereas on direct matching he appears to be incompatible even with a universal donor. This nuisance can be overcome by testing and transfusing at 37° C.

**Methods of Transfusion.**—Whatever method is used one thing

is imperative. The first 20 c.c. must be injected very slowly and the transfusion abandoned immediately if severe pain in the back is produced. This constitutes the "biological test" and must never be omitted.

Kimpton's paraffined tube has been entirely discarded, as has direct vein-to-vein connection. The most common method is that in which the blood taken from the donor is either citrated or defibrinated and infused into the recipient at leisure. Defibrinated blood, obtained by continually shaking the blood round a glass rod in the collecting bottle, is said to give fewer unpleasant after-effects than citrated blood. In this latter method the donor's blood is run into a bottle containing 150 c.c. of sodium citrate solution (2 grm. to 100 c.c. of distilled water) to the pint (approximately 568 c.c.) of blood. Blood is obtained from

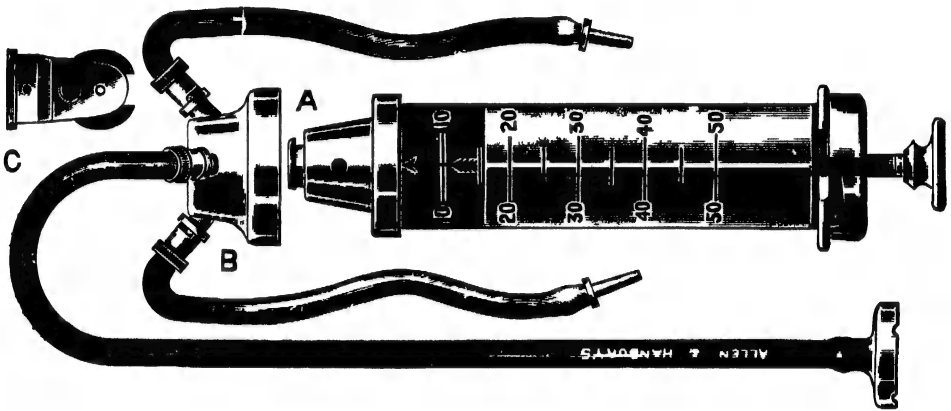


FIG. 57

The Rotanda three-way syringe. (Allen & Hanbury's)

A, B and C are the component parts of the nozzle head with its three-channel distribution.

the donor by puncturing a vein with a wide-bore needle to which is attached a short length of rubber tubing leading into the collecting bottle, which is immersed in hot water and gently rotated during the operation. If a vein in the arm is used, a sphygmomanometer cuff is placed on the arm above, and the pressure regulated to ensure a steady flow of blood.

The blood is introduced into the recipient either via a cannula tied into a vein, when the gravity method from a funnel may be used, or through a needle passed into a vein, in which case some type of syringe will have to be used. The three-way Rotanda syringe (Fig. 57) has been found most serviceable, one lead being connected to a flask of warm saline, with which the whole circuit is first filled, another to the blood container and the third to the patient. The blood should be given slowly, at least twenty minutes being taken in giving 1 pint. A modification of the greatest value is the continuous-drip method of transfusion (Fig. 58) by which large quantities of blood can be given over a long period of time, thereby overcoming all the disadvantages of the rapid transfusion. The method entails apparatus

for keeping the blood warm and constantly mixed, and this is done by allowing oxygen to bubble through it.

In infants the superior longitudinal sinus is usually used as the portal of entry and the amount given is 15 c.c. per pound weight. As an alternative the internal saphenous vein at the ankle is used in children.

**Stored Blood.**—The great demand for large quantities of blood which occurs when many casualties have to be dealt with in a short time presents a considerable problem to a transfusion service. To meet this "blood banks" have been formed in which large quantities of citrated blood are stored for immediate use. Care is needed in collection, sterilisation and group labelling, but this method has proved a success during aerial bombardment of cities, and it allows the transportation of blood from central stores to distant battle areas by aeroplane. Reactions are apparently little more frequent than with fresh blood, but doubt persists as to how long it is safe to keep blood in store. Except in periods of great pressure blood older than fourteen days should not be used.

**Plasma Transfusion.**—For many purposes blood plasma has been found to be an excellent substitute for blood and in certain conditions, *e.g.*, burns, it is superior. It can be stored in its fluid state or dried and subsequently reconstituted. Its preparation requires both skill and care, but it has the great advantage of reducing unpleasant reactions to a minimum. Serum has also been used, but has no advantages over plasma; its preparation, however, is a much more intricate process.

**Indications for Blood and Plasma Transfusions.**—The great therapeutic value of each is likely to be lessened if they are regarded—a too prevalent belief—as equal and interchangeable in all conditions. Blood is obviously required when blood has been lost or destroyed by disease, but its use in cases of non-hæmorrhagic shock, *e.g.*, burns, may have a most damaging effect. Plasma has a definite value in severe hæmorrhage, but its chief use is in those conditions in which the body is suffering from fluid loss and its resultant hæmoconcentration.

The late general treatment of hæmorrhage includes the use of iron tonics, sunlight, fresh air, plentiful nourishing diet and regulated rest.

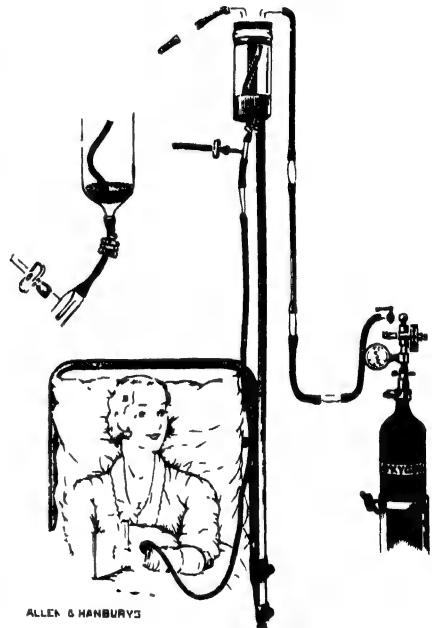


FIG. 58

Kekewich's apparatus for the continuous-drip administration of a blood or saline transfusion. (*Allen & Hanbury's.*)

### LOCAL TREATMENT

The aim of local treatment is to occlude the bleeding vessel until such time as natural arrest can take place. The following methods are available ;—

1. **Pressure** as a temporary and an emergency measure, either at the site of bleeding (particularly applicable to venous hæmorrhage) or over the main vessel of supply at some distance, is invaluable when feasible. Pressure can be exerted by the fingers, by a pad firmly bandaged on, or by a tourniquet, of which there are many types. In general, compression should not be continued for periods over an hour in duration.

2. **Position**.—In bleeding from a limb vessel, elevation will assist hæmostasis, the consequent emptying of the veins leading to a reflex arterial vasoconstriction.

3. **Heat** may be applied in the form of very hot water (150° F.) which causes constriction of the vessels by stimulation of its medial coat, or by the cautery used at dull red heat, or by diathermy. These methods are applicable to bleeding from a large area, particularly if septic, or from a cavity.

4. **Cold** applied either as ice or very cold water acts in a similar manner. It finds its chief application in oozing from the mouth, pharynx or even the stomach.

5. **Chemicals**.—Those used locally are divided into two main classes, viz., styptics and astringents. The former cause direct coagulation, the latter a mild type of vasoconstriction. Of the styptics, which are only of value in small wounds, perchloride of iron, alum, silver nitrate, tannic and gallic acids and creosote are in common use. The most popular astringents are turpentine, hamamelis and lead acetate.

Two other agents are worthy of mention: Adrenalin (1 : 1000) applied locally will stop oozing, and small free muscle grafts, probably owing to their prothrombin content, have a special field of usefulness in minor hæmorrhage from the surface of the brain, the lung and elsewhere.

6. **Acupressure** is a method of historic interest. A straight rod was inserted beneath the vessel and a figure-of-eight knot was tied over both vessel and rod.

7. **Forcipressure**.—The artery forceps of Spencer Wells and Kocher may be used to stop hæmorrhage from small vessels by causing adhesion of the intima from crushing, the subsequent curling up of the intima and media allowing natural clotting to occur. They are also used universally as a temporary hæmostatic until the bleeding vessel can be ligatured (Fig. 59).

8. **Clamps** are simply forceps of special design, and are used for big vessels or vascular pedicles (*e.g.*, the renal). They can be used as crushing instruments as a preliminary to ligation, or can be left on for forty-eight hours to allow a natural clot to form behind them.

9. **Torsion**.—Small vessels, particularly of the skin, may be twisted



by a pair of forceps. This ruptures the inner coat and allows rapid clotting.

10. **Ligature** is the commonest method of dealing with a hæmorrhage of any magnitude. The materials used are either catgut (prepared from the submucosa of the small intestines of sheep), silk thread, linen thread and silk-worm gut. Of these, catgut is the most universally used, having the advantage of being ultimately dissolved by the tissues, all other materials persisting as foreign bodies. Silk and thread can be sterilised by boiling; catgut is prepared in a variety of ways.

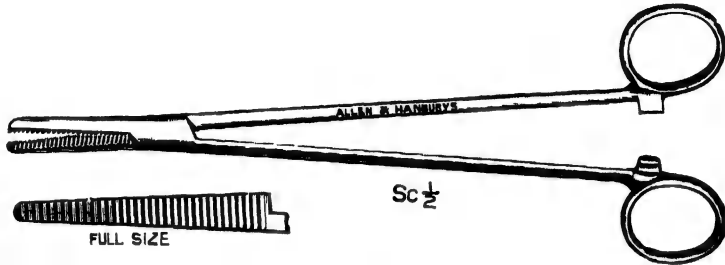


FIG. 59

One type of straight artery forceps. (*Allen & Hanbury's.*)

It should be remembered that wherever anastomotic circulation is free, a vessel, either artery or vein, may bleed from both ends, and each will require ligation.

### VARIETIES OF HÆMORRHAGE

**Arterial Hæmorrhage** may be primary, reactionary and secondary and may occur from a divided artery in an open wound, subcutaneously or internally. Arterial bleeding occurs in jets synchronous with the heart beat, is bright red in colour and often appears at both ends of the cut vessel, being more profuse from the proximal side.

**PRIMARY HÆMORRHAGE** occurs at the time and as a direct result of an injury. Its treatment demands the securing of the bleeding vessel by one of the methods enumerated above. Occasionally the wound may have to be enlarged before the vessel can be found.

**REACTIONARY (OR INTERMEDIATE) HÆMORRHAGE** occurs within twenty-four hours of an injury and is an expression of the failure of the temporary natural arrest or of faulty ligation at the time of original treatment. It is usually due to a rise in blood pressure coincident with recovery from initial shock and hæmorrhage.

**SECONDARY HÆMORRHAGE** occurs after the lapse of at least twenty-four hours from the time of injury, but more commonly eight to ten days later. It is almost always due to sepsis in the wound, the bacterial ferments present under such conditions leading to proteolysis and softening of the temporary clot naturally formed in the damaged vessel. Subsidiary etiological factors are arterial disease, high blood pressure and toxæmia.

Secondary hæmorrhage therefore occurs in a septic wound and its arrival is usually heralded by one or more small "warning" hæmorrhages. If the warning is not taken and adequate preparations made to deal with the subsequent bleeding, it may be so copious as to be fatal. The longer the time from the initial injury the more likely is the hæmorrhage to be severe, as the largest arteries take the longest time to rupture. Treatment demands digital pressure of the main trunk, a tourniquet or firm plugging as emergency measures. The wound must then be opened up freely and the bleeding vessel located and retied at each end as far away from the site of infection as possible, and the wound cleaned and drained as efficiently as possible. Proximal ligature of the main trunk or even amputation may be necessary in the worst cases.

**Venous Hæmorrhage** occurs in a steady stream (except when a big vein is lying in contact with a big artery from which pulsation is transmitted, or in the case of veins emerging from the thorax and affected by respiratory movements), and the blood is dark blue or even black in colour, coming chiefly from the distal end of the divided vessel, unless the vein is varicose.

Venous bleeding from small veins requires little more than pressure by way of treatment. Large veins, if only partially injured, may be sutured, but if completely divided must be ligatured at both ends. Secondary hæmorrhage from large veins is uncommon and is treated on similar lines to that in arteries. Wounds of veins in the neck may lead to air being sucked into the circulation and, if this occurs into one of the big veins near the heart, an "air embolus" may result and cause death from interference with the heart's action.

**Capillary Hæmorrhage** consists of a generalised oozing from the raw surface of a wound. The colour of the blood changes gradually from a bluish to a reddish tint as it wells up. It is treated by pressure, heat, cold, styptics, etc., as described above.

**Hæmophilia** is a hereditary disease associated with severe and prolonged bleeding from wounds and a tendency to spontaneous hæmorrhage. It was said to be peculiar to males, though transmitted through the female, but recent observations have revealed an occasional case of female "bleeders."

The *etiology* seems to lie in the failure of the platelets, which are present in normal numbers, to deliver up their thrombokinas, and hence there is a delay in the conversion of prothrombin into thrombin and correspondingly delayed clotting time. Obviously, in these patients operations must be avoided except in the gravest emergencies. Any trivial wound may lead to fatal hæmorrhage, *e.g.*, cut finger, tooth extraction and circumcision. Hæmorrhages into joints and under mucous surfaces are fairly common. The prognosis is not good, over 50 per cent. dying before the age of 10, and only 10 per cent. reaching adult life.

*Treatment* consists in blood transfusions, intravenous serum, intramuscular hæmoplastin, calcium chloride and the application of ice, adrenalin or whole blood to the wound.

## TRAUMATIC SHOCK

This Second World War has provided material under ideal conditions for a vast amount of research into the pathology and treatment of shock. Looking over the stricken field we cannot but feel that this research has had much the same effect upon this subject as has aerial bombardment upon the bricks and mortar of our cities. It has destroyed most of the old theories but has put few solid concrete facts in their place.

It has seemed to us that much of the confusion has arisen from an inexact and misapplied nomenclature. The term "shock" is used to cover the state resulting from such diverse etiological factors as grave injury, internal and external hæmorrhage, burns, perforation of abdominal viscera, biliary, intestinal and renal colics, a blow upon the solar plexus and even the receipt of bad news or injury to our moral susceptibilities. It is hardly surprising that chaos has resulted. It seems desirable, therefore, that in all cases the word shock should be preceded by a definitive adjective to avoid all misconception. Accordingly we are about to describe "Traumatic Shock."

**Its Varieties.**—It has been the accepted custom to divide shock into two types, viz., primary and secondary, since their etiology and clinical picture differ in many respects. Their differentiation is not always easy and, moreover, they are apt to merge indefinitely the one into the other. Indeed there is a tendency to-day to abandon altogether the term "primary shock." Nevertheless although the distinction is not entirely satisfactory, the full clinical picture and treatment of shock cannot be understood unless this classification is retained.

### PRIMARY SHOCK

Primary shock is a condition of syncope following upon injury. It has been a marked feature of air-raid casualties who reach reception hospitals soon after receipt of their injury. It is believed to be due to over-activity of the nervous system and two varieties are described.

**Psychogenic Shock** results from stimulation of the medullary centres by psychogenic impulses. Distress, fear, terror and physical pain even without injury are potent factors. This type is usually recovered from rapidly; if, however, the noxious stimuli are sufficiently intense, widespread or long-continued, they will predispose to the development of secondary shock.

**Neurogenic Shock** results from the action of sensory stimuli of somatic and autonomic origin upon the medullary centres. This is the type of shock which complicates surgical operations, especially those upon the abdomen.

Primary shock is produced by "a general decrease in vascular tone and reflex inhibition of the heart through the vagi. Influences acting through the nervous system, such as direct trauma, a severe blow upon the abdomen, a laparotomy or psychic stimuli bring about a diminution of constrictor tonus resulting in vasodilatation. A fall in blood pressure follows this latter, a continuation of which leads to

a diminution in the venous return to the heart. Anoxia develops later, causing further serious complications" (Raven).

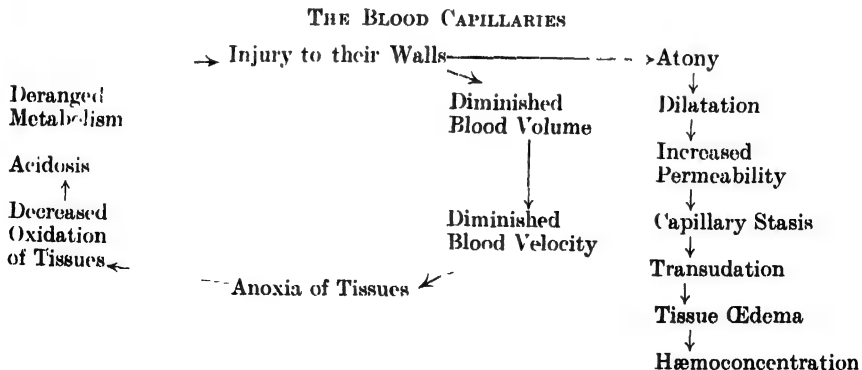
*Clinical Picture.*—Primary shock develops immediately after receipt of injury. It may rarely cause instantaneous death when the great sympathetic centres, *e.g.*, the solar plexus, are affected. Usually its duration is short and in difficult cases Whitby suggests that if the blood pressure remains below 100 after one hour, secondary shock has made its appearance. "The patient appears to be in a prolonged fainting fit, ushered in with feelings of great weakness. Pallor and cold sweating are present together with a fall in blood pressure. Pulse rate is slow and its volume poor, while respirations are slow and shallow. The fall in blood pressure is not serious provided the respiratory centre continues to function well. The extremities are usually warm owing to peripheral dilatation. If the condition is not speedily fatal, the stage of reactions sets in, being heralded by disappearance of pallor, improvement in pulse volume and an attack of vomiting. A careful record of pulse and blood pressure must be taken every fifteen minutes" (Raven).

*Treatment.*—In mild cases a short rest and a simple stimulant suffices. More severe examples are treated in recumbency with hot-water bottles or a radiant heat cradle, and all noxious stimuli either of psychogenic or neurogenic origin eliminated. Fear and despair must be counteracted, every injured part protected or splinted and hæmorrhage controlled. Morphia may be used in small, but never in large, doses. A blood transfusion should not be given in undoubted cases of primary shock. If, however, doubt exists as to the presence of early secondary shock, then a transfusion is justifiable. All our efforts both in diagnosis and treatment are directed toward the prevention or immediate recognition of secondary shock.

## SECONDARY SHOCK

### PATHOLOGY

That little is known of the pathology of secondary traumatic shock the multitude of theories goes to prove. Before we inquire into its



possible causation, let us examine the bodily changes it entails. Raven has embodied most attractively the sequence of events and the interplay of different factors at work in the above diagram.

This is described as the "vicious circle" of secondary shock, and if it is not broken death is inevitable.

We will now pass on to a consideration of the various theories.

1. **Loss of Blood and Fluid.**—In so far as the principal manifestations are so closely linked with changes in the circulatory system, secondary shock has been termed by Blalock "hæmatogenic." This worker, followed by many others recently, is inclined to regard local loss of blood as the chief etiological factor. Important in initiating shock it undoubtedly is, but it does not explain many of the phenomena seen in these patients. We deplore the tendency evident in the past three years to speak of hæmorrhage and shock as identical states.

2. **Presence of a Toxin.**—After the First World War the views of Cannon and Bayliss, supported and elaborated by Dale and Laidlaw, were widely accepted. They postulated the presence of a toxin derived from the traumatised area circulating in the blood stream. Histamine and similar H substances, as we have shown in Chap. I, play a local part in the pathology of acute inflammation; but all recent work has proved beyond doubt that they have no relation to secondary shock.

3. **Nervous Stimulation** has once again been revived as an etiological factor. This theory suggests that nervous impulses from the site of injury bring about a disturbance in the vagosympathetic system. Lorber's experiments seem to demonstrate that the nervous system undoubtedly exerts a powerful influence in the production of shock.

4. **Vasoconstriction and the Adrenal Cortex.**—Excessive adrenal activity has been held to account for the vasoconstriction which is present in the earlier stages. It is probable that this is an effect rather than a cause of shock; in the depressed state of the circulation vasoconstriction protects the vital centres and is therefore a defensive reaction.

The functions of the adrenal cortex have been questioned in another way. The later phases of Addison's disease, severe shock and bilateral adrenalectomy, produced a similar condition in the blood, there being an increase in the non-protein nitrogen and a decrease in sodium and chlorides. Moreover Coller has pointed out that shock is reduced by a proper maintenance of the balance between fluid, blood chlorides and the sodium-potassium ratio. The probable explanation, however, is that secondary shock calls urgently for cortical hormone, and early cases may derive benefit from injections of cortin or eucortone.

#### CLINICAL PICTURE

The patient lies very still in an apathetic condition. If conscious his mind is clear but listless, but as shock deepens unconsciousness and delirium make their appearance. The cheeks and eyes are sunken, the nose pinched and the brow creased; the skin is cold and clammy and presents a marked pallor which later merges into an ashen hue. Cyanosis is present and the finger-tips, nose, ears and lips assume a livid tint. The tongue is dry and furred, and great thirst is complained of.

Blood pressure and pulse rate are the most important signs; the

former falls steadily as the condition progresses, at first being about 100 and then dropping to 50 or lower. Its return to 100 is the surest criterion of the success of resuscitation methods. Pulse rate is more variable as it may be normal, or even slow, but in general it rises as the pressure falls.

The heart sounds are faint and a "tic-tac" or "gallop" rhythm may be present. The apex beat is neither to be seen nor felt and all peripheral veins are empty and collapsed, so that it may be quite impossible to introduce a needle for transfusion. Respiration varies little in the early stages, but later deep breathing is interspersed with short rapid respirations. Cheyne-Stokes breathing may be seen and the end is ushered in with irregular gasping movements assisted by forced use of the accessory muscles of respiration.

The output of urine is diminished, but its specific gravity is not markedly raised. Thirst and vomiting are constant symptoms and incontinence of both sphincters occurs before death.

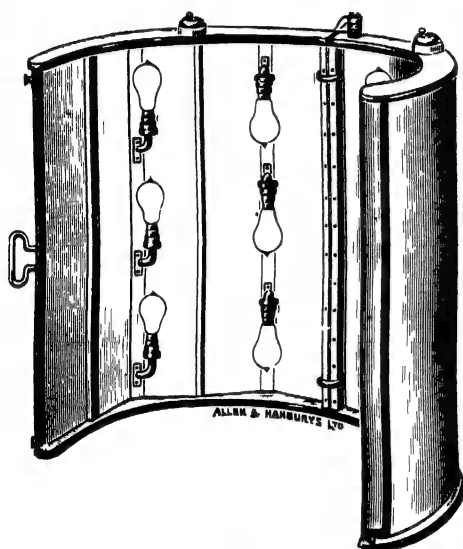


FIG. 60

A large radiant-heat cradle. (Allen & Hanbury's.)

## TREATMENT

**A. Prevention.**—1. IN SURGICAL OPERATIONS.—Fear and anxiety must be allayed and the patient convincingly assured of a successful outcome. All unnecessarily rigorous preparation should be avoided and a peaceful night's sleep obtained with the aid of bromide, medinal or similar sedative.

Many patients by reason of their disease are suffering from malnutrition, *e.g.*, in pyloric obstruction or from dehydration. Maintenance of the body's water balance requires about six pints of fluid in twenty-four hours, and this amount will need to be increased if vomiting and diarrhoea occur. Five per cent. glucose saline is given by either rectal or intravenous drip infusion, and if acidosis is present sodium bicarbonate must be added. If the plasma proteins have fallen below their normal concentration a plasma transfusion is indicated. Similarly many patients come to operation suffering from secondary anaemia as a result of either hæmorrhage or disease; they will require a blood transfusion before operation. In grave emergency when hæmorrhage is threatening life (as in repeated hæmatemesis from a gastric ulcer) we may be forced to operate when the hæmoglobin is as low as 35 per cent., but when possible surgical intervention should be delayed until the reading has reached 70 per cent.

During operation rough handling must be avoided. Of the many great lessons taught by Moynihan the paramount importance of

gentleness was the greatest. Exposure of internal structures and hæmorrhage are reduced to a minimum, and clean cutting always takes the place of tearing. As small a surface area as convenient is displayed and warmth is carefully conserved during operation as well as during transit to and from the theatre. When facilities exist a patient should be moved direct from the table to a warmed bed, which is then removed to ward or room. Heat is most conveniently provided by radiant-heat cradles (Fig. 60).

2. IN INJURED PATIENTS.—In peace an injured person usually reaches hospital so quickly that shock may be successfully prevented: in the bombing of cities casualties also are frequently brought with

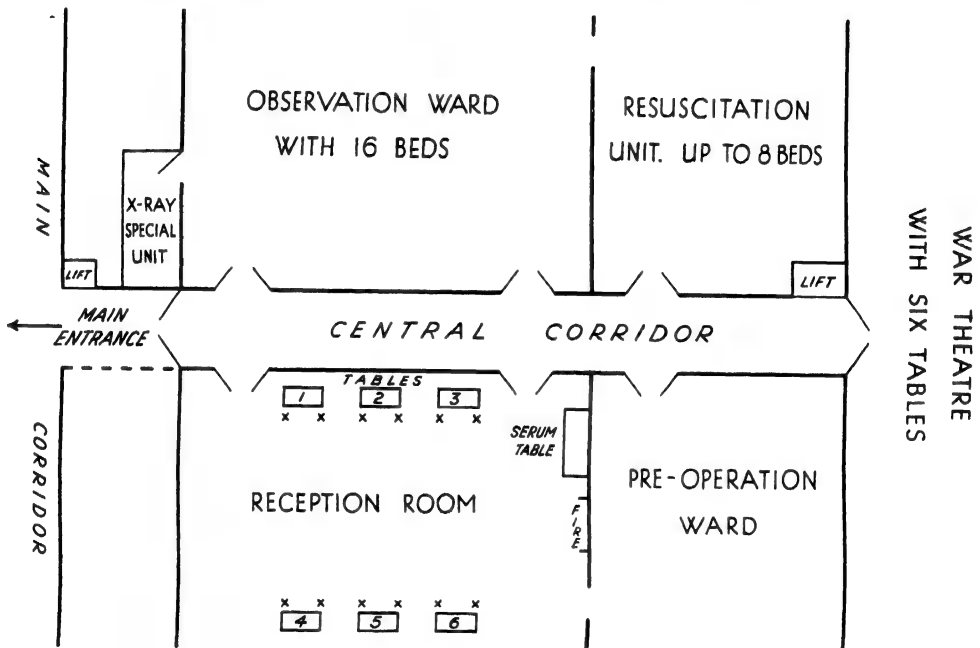


FIG. 61

Ground-floor plan of war surgical unit at St Mary's Hospital.

commendable speed. But, generally speaking, battle casualties are delayed in transit to surgically equipped hospitals and first-aid measures assume great importance. Again gentle handling in first dressing and in transport is essential. Hæmorrhage is controlled by firm bandaging or tourniquet and the injured part is immobilised. Morphia (gr.  $\frac{1}{4}$ ) should be given to allay pain and the time of its administration *must* be noted on the casualty card. Hot drinks may be given provided no abdominal wound is present.

**B. Active Treatment.**—We have shown that unless the vicious circle of shock is broken death must ensue, and the earlier this is done the better. We shall describe the measures to be adopted when battle casualties are arriving in considerable numbers, but the general principles apply equally to single patients in times of peace.

1. THE RECEPTION UNIT.—A ground plan of the unit at St Mary's Hospital is given here (Fig. 61), which functioned without a hitch

throughout the bombing of London. Casualties are received in the reception room which has six tables, each with a pair of trestles to receive a stretcher. Slung on each pair is an inverted "Restor" heat cage (Fig. 62) and at each table sits a reception clerk (in our case one of our almoner staff). A senior medical officer is in control, and every patient is carefully examined and the nature and extent of his or her injuries are assessed. Full particulars are entered upon the casualty card, which is also stamped with a coloured star<sup>1</sup> indicating the patient's destination, for it is at this early stage that this vital decision must be taken. Although rest, a minimum of handling and preservation of body heat are desirable, patients must be thoroughly examined lest a grave internal injury be altogether overlooked. The patient is then sent to either (1) the resuscitation unit; (2) the pre-operation room; (3) the main surgical wards; (4) an observation ward; or (5) to the casualty department for minor dressings. It is of great advantage if the whole unit together with the theatre is contained in one compact unit on the same floor as in our plan. If this is impossible, then the resuscitation unit must be adjacent to the theatre.

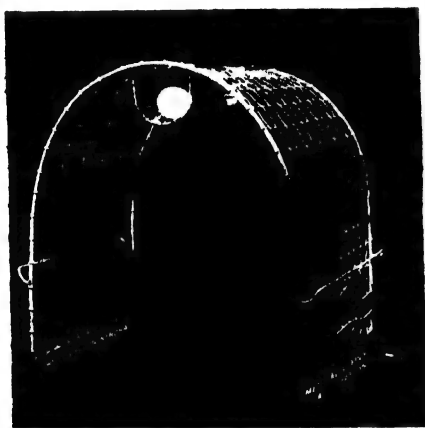


FIG. 62

The Restor electrically heated resuscitation cage. (*Surgery of Modern Warfare.*)

2. RESUSCITATION UNIT.—The efficacy of this life-saving organisation depends upon team work, the training of which calls for as much intensive training as does that of first line assault troops. The ideal type of man to be in charge is an experienced medical registrar.

Junior medical officers, nurses and students are under his command and each knows exactly what his or her duties are. The room must always be ready for instant use at any time of day or night, and its temperature is maintained despite any considerations of fuel economy (the unit is there to save lives not fuel). Each bed is made up with a large radiant heat cradle in position, and every piece of apparatus ready in its appointed place. The whole unit is ready for action the moment the light switch is turned on at the door. Finally let us remember what the objects of this unit are in the treatment of gravely shocked patients. It is to revive them and restore their circulatory system as soon as possible to such a condition as permits surgical treatment with reasonable prospects of success. The following general principles govern the working of this unit.

*Absolute Rest.*—In spite of the intense activity which will prevail, absolute quiet is maintained, and if bombing and gunfire are still in progress patients ears are plugged with cotton wool. All go about their duties with that speed, efficiency and silence which intensive

<sup>1</sup> Coloured arrows prominently displayed in the corridors guide stretcher-bearers to their proper destination.



training can alone guarantee. Active treatment must be arranged for with as little disturbance of the patient as possible.

*Heat must be Provided.*—The room is warm, all draughts being eliminated, and the bed is warmed. If the patient's general condition permits all clothing is removed and warm, dry night attire substituted. Warmth in bed is provided by radiant heat, but if this is not available hot-water bottles or a brick wrapped in a blanket will suffice. Care must be taken to avoid overheating, for MacMichael has pointed out that this may result in loss of fluid by sweating as well as an anoxæmia of the vital centres due to peripheral vasodilatation.

*Sedatives* are usually needed for restlessness or pain. Morphia in repeated small doses is of great value; a full dose should never be given as it further depresses the vital centres. Moreover its routine use in the absence of real indications is to be deprecated. In head injuries bromides, chloral and paraldehyde are given per rectum and luminal sodium intramuscularly. Coramine is an excellent *stimulant* and can be given repeatedly on numerous occasions. Verital, an isomer of ephedrine, is most useful in raising blood pressure. Extracts of adrenal cortex, *e.g.*, cortin and eucortone, have been used, but their value has not yet been determined.

*Oxygen* is of the greatest importance. It is best administered by means of the B.L.B. mask (Fig. 63).

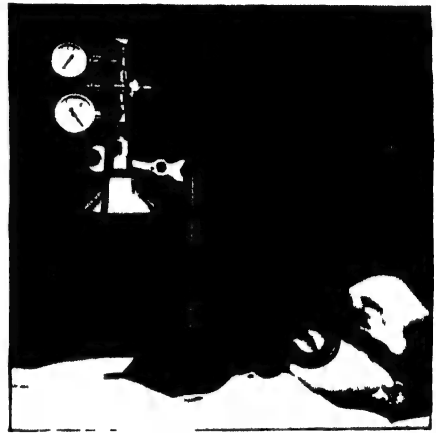


FIG. 63

Administration of oxygen with a B.L.B. mask. (*Surgery of Modern Warfare*)

*Replacement of Fluid Loss.*—Indications for transfusion with blood and plasma and infusions of glucose saline are given earlier in this chapter (p. 151). Repeated blood-pressure readings are taken, the arm air bag being left in position throughout. If the patient is able to drink and vomiting is not persistent, hot drinks should be given liberally.

3. PRE-OPERATIVE WARDS.—When casualties are being received in considerable numbers, one of the most important duties of the surgical controller is to label patients in numerical order, so that they are taken to the theatre at the proper time without further reference to him. He should not begin to operate until this very essential task is finished. From time to time he will visit the resuscitation unit to see which patients have revived sufficiently for operation. Judging by the number and nature of the cases he will give his orders for the number of tables to be manned and for their staffs to be ready for action. Not until he is satisfied that all these essential preliminaries are in train will he take a table. By reason of his experience and skill he will work faster than his juniors and will quickly make up the time spent in organising the night's work.

In the pre-operative ward patients will be prepared as thoroughly as in less arduous times. It is extremely rare that any casualty needs to be operated upon so urgently that he cannot be undressed and washed. The practice of taking men and women to the theatre still clothed and impregnated with the dust and rubble of shattered houses cannot be too strongly deprecated. Those requiring operation, but whose injuries do not demand immediate surgical intervention, should be sent to the general surgical wards, and only that number, which can be dealt with in the first two hours, retained in the pre-operative ward. This special ward is designed for those who need urgent surgical attention, *e.g.*, abdominal lesions, limbs with tourniquets in position, etc. The remainder can be prepared more leisurely in less hectic surroundings.

### **FAT EMBOLISM**

This complication of injury has been regarded as a very rare phenomenon, more often associated with fractures than any other type of trauma. Globules of fat enter the blood stream and are usually trapped in the pulmonary capillaries. Occasionally these droplets occur in such profusion that a state resembling pulmonary embolus results and death may occur.

During the period of intensive bombing of this country attempts were made to establish fat embolism as a frequent cause of shock and as of very common occurrence in battle casualties. This view has not gained acceptance and fat necrosis remains an interesting but rare pathological condition.

A. E. PORRITT.

R. M. HANDFIELD-JONES.

## CHAPTER IX

### ULCERATION AND GANGRENE

#### ULCERATION

**U**LCERATION is the molecular or cellular death of superficial tissues leading to loss of substance, and is due to the action of a traumatic, infective or neoplastic agency. An ulcer, therefore, means the loss of epithelial and the exposure of subepithelial tissues in an area of skin or mucous membrane. The varieties and appearances of ulcers are so numerous and the ulceration is so often merely symptomatic that a complete classification is not feasible here. It is most convenient for the student of general surgery to classify them by their causation.

##### 1. SIMPLE NON-SPECIFIC INFECTIONS (Staphylococcal and Streptococcal).

- (a) Vascular defect, *e.g.*, varicose ulcer, gravitational ulcer.
- (b) Trauma or Pressure, *e.g.*, too tight splinting or plaster bandaging over gouty tophi or retained foreign bodies.
- (c) Thermo-Electro-Chemical.
- (d) Neuropathic, *e.g.*, bedsores, perforating and trophic ulcers.

##### 2. SPECIFIC INFECTIONS.

- (a) Tuberculous—varying types in the skin and all mucous membranes.
- (b) Syphilitic—varying types in all stages.
- (c) Ducrey's soft sore, anthrax, actinomycosis and diphtheria.
- (d) Various intestinal infections; typhoid, dysentery, cholera and stercoral ulceration.

##### 3. NEOPLASTIC, *i.e.*, carcinoma, sarcoma and rodent ulcer.

These conditions may be termed the "exciting causes": the occurrence and behaviour of an ulcer, however, will depend largely on certain "predisposing causes." General debility and wasting from other serious diseases such as diabetes, chronic nephritis, heart disease or septicæmia not only predispose to ulcer formation but also retard its healing.

**The Clinical Appearances** should be carefully studied in order that diagnosis may be facilitated.

1. **POSITION, SHAPE, SIZE AND NUMBER.**—The anatomical site is of some importance, *e.g.*, a varicose ulcer so frequently affects the lower half of the leg, while syphilis affects the upper half. Similarly, the shape may give an indication of the causative organism. Multiple

ulcers scattered over the body suggest a generalised constitutional disease, *e.g.*, syphilis or tubercle, while multiple lesions in a small area suggest seeding out or spread by some pyogenic or parasitic factor.

2. **METHOD AND RATE OF PROGRESS.**—Steadily maintained extension points strongly to new growth, rapid onset and quick healing suggest simple non-specific ulceration, simultaneous healing and extension is characteristic of some specific lesions, and extreme chronicity points to a definite reason for the absence of healing.

3. **THE FLOOR** is usually sunken beneath the healthy surface. It is raised in fungating growths and in healing ulcers when the granulations are exuberant. The type of tissue covering the floor must be noted, *e.g.*, red, vascular and acute granulation tissue, the pale flabby and anæmic granulations of tuberculosis, the fibrous avascular floor of syphilitic ulcers, and the necrotic debris of the new growth.

4. **THE EDGES** reveal much. Sharply defined, red and vascular edges denote extension; rounded, smooth, grey ones point to chronicity; raised, everted and necrotic, they are diagnostic of a squamous-celled carcinoma; the clean cut, "punched out" edge is syphilitic, and the thin, blue, undermined one suggests tuberculosis or other chronic inflammation.

5. **THE SIDES** are, of course, continuous with the floor, but their disposition is important. Some ulcers have slowly shelving walls which pass gradually into the floor, others have sharply sloping walls which meet at an angle without any real floor. Such a "fissured" ulcer is seen in tuberculous disease of the tongue.

6. **THE BASE** is that zone of tissue immediately surrounding and underlying the ulcer. The presence or absence of induration is of importance.

7. **THE DISCHARGE.**—Free pus indicates an active extending ulcer; mixed pus and serum in moderate amounts are to be expected during healing. Scanty sero- or muco-pus is seen in tuberculosis. Healthy granulations bleed readily, but the granulations of tuberculosis and syphilis do not. Certain diseases produce specific cells or constituents in the discharge which are diagnostic, *e.g.*, the golden-yellow granules in actinomycosis.

8. **RELATIONSHIP TO SURROUNDING STRUCTURES.**—Fixation of an ulcer to underlying tissues must mean either a chronic inflammation of long standing or malignancy.

9. **THE LYMPH DRAINAGE ZONE** may be affected in many cases of ulceration. The clinical appearance of the enlarged glands may be of diagnostic value.

### **SIMPLE NON-SPECIFIC ULCERATION**

**The Pathology** of this condition is divided into three stages.

A. **STAGE OF ACTIVE EXTENSION**—The Spreading Ulcer.—The conditions present are exactly similar to those in the wall of an abscess cavity. The surface is covered with dirty greyish disintegrating tissue and no granulation tissue is present, the edges are well-defined and acutely inflamed, the base is thickened and œdematous and pus is being discharged.

**B. STAGE OF PREPARATION FOR HEALING—The Transitional Ulcer.**—The active extension has been arrested and the surface is becoming clean by the shedding of necrotic tissue. No granulation tissue has yet appeared but the surface is now rosy in colour instead of fiery red, and has a glazed appearance as if a thin glistening film had formed over it. The thickening and oedema of the base have disappeared. The rosy film gives place to numerous little red specks, which increase in size till they coalesce and finally the whole area is covered with granulation tissue. In acute cases this stage lasts so short a time that it is hardly recognised. Its imperfect development and its persistence constitute a "chronic ulcer."

**C. STAGE OF REPAIR—The Healing Ulcer.**—The condition now present should no longer be called an ulcer, for it is simply a healthy granulating wound. Its surface is covered with granulations, the edge is actively proliferating and sending in a layer of epithelial cells to cover the surface, and the zone immediately beneath the granulations is changed into scar tissue. Healing is complete.

**Clinical Features**—1. **THE ACUTE ULCER.**—Almost every ulcer has an acute phase at its inception, but the term is applied to those which are characterised by rapid progress and a marked tendency to heal. They often follow slight trauma and are not infrequently multiple. Examples are acute dyspeptic ulcers of the mouth and acute peptic ulcers of the stomach and duodenum. In the mouth and on the skin they are painful and tender, but pass rapidly through the three stages to complete recovery within a few days.

*Treatment* consists in removal of the cause and rest to the part, which should be placed in the position most suitable to overcome venous obstruction. Boracic fomentations or linseed poultices will clean up the surface in the first twenty-four hours, and eusol dressings will help sloughs to separate. After twenty-four to thirty-six hours mild antiseptic dressings, *e.g.*, perchloride of mercury lotion (1 : 1000), flavine or acriflavine in paraffin (1 : 2000), give excellent results. As soon as healing has started, flavine or lotio rubra dressings can be combined to advantage with infra-red or short-wave therapy once a day.

Under such treatment healing is usually complete within a few days but very occasionally a rapid extension of the ulceration occurs involving and destroying a considerable extent and depth of tissue. This constitutes the condition of phagedena, now rarely seen, and then in connection with venereal infections of the external genitals, when the penis may be completely destroyed. Repair is followed by extensive scarring and deformity.

2. **THE CHRONIC ULCER**—Causes of Chronicity.—If an ulcer fails to heal, some good cause must be present and should be sought for before treatment is undertaken. These causes are (a) defective circulation in old people leading to venous stasis and congestion, especially in the legs; (b) venous obstruction, *e.g.*, varicose veins, femoral thrombosis, etc.; (c) lack of proper treatment, especially rest, *e.g.*, a fissure-in-ano which cannot be put at rest until the external sphincter is anæsthetised, divided or stretched; (d) fixation to underlying tissues

(bone, fascia or muscle), which prevents the efficient development of healthy granulations and the drawing together of the edges and floor ; (e) constitutional diseases ; (f) persistence of the original exciting cause ; (g) pressure of oedema on the surrounding vessels, which leads to a rapid extension of the ulcer ; (h) artefact, *i.e.*, the deliberate prevention of healing by the patient, usually if of unsound mind or for the purpose of defrauding employers or insurance companies and eliciting sympathy.

### CERTAIN VARIETIES OF CHRONIC ULCER

**Varicose Ulcer** is very common among elderly women of the poorer classes who have suffered from varicose veins for many years, as a result of which the skin is pigmented and thickened from a chronic infective dermatitis (the so-called varicose eczema, Fig. 64). The actual ulceration is brought on by dirt, rubbing of clothes and neglect. The surface is rough, irregular and glistening, dirty yellow in colour and a few patches of coarse granulations are present.



FIG. 64

Varicose ulcers of left leg ; scars of healed ulcers on right.

The edges are firm and well-defined, the discharge is seropurulent and profuse and the base is densely indurated and adherent to underlying tissues. If the surface of the tibia is in the base, an area of periostitis forms beneath the ulcer, and later a diffuse osteoperiostitis may follow. The ulcer is usually seen in the lower half of the leg and on the antero-internal and antero-external aspects. It may spread gradually round the circumference of the leg and, by obstructing lymphatics and veins, give rise to a condition of pseudo-elephantiasis.

**Eczematous Ulcers** are superficial and have a copious discharge which leads to a spread of the eczema.

**Irritable Ulcers** are seen near the ankle only. They are small, have no connection with varicose veins and are exquisitely tender owing to the exposure of living nerve endings.

**Pressure Ulcers** are commonly caused by the careless application of splints and plaster bandages over prominent bony points such as the heel, the malleoli of the ankle, and the lower end of the radius and ulna. The skin over a rapidly growing innocent growth, over the bone in a conical stump, and over a gouty tophus may ulcerate. Bed-sores may be purely pressure in type, but are usually associated with nerve lesions.

**Neurotrophic Ulcers** are indolent and most difficult to treat. They occur in lesions of the peripheral nerves and of the central nervous system, *e.g.*, tabes dorsalis, transverse myelitis, anterior poliomyelitis and syringomyelia. The ulceration is due to pressure on the desensitised and devitalised skin and therefore occurs over the bony

points of the sacrum, pelvis and lower extremity. The most serious example of this condition is the "bed-sore" over the posterior surface of the sacrum and coccyx. The skin gives way and the ulceration progresses till the bone is laid bare and an infective spinal meningitis ends the scene.

**Thermo-electro-chemical Ulcers** are not common, as these agents cause burns or gangrene rather than local ulcers. In general, they tend to be indolent in spite of a fresh, healthy appearance. Diathermy burns, in particular, take a long time to heal.

**Meleney's Undermining Ulcer** is a rare condition occurring in any wound or in an area of lymphatic glands. It is due to an anaerobic hæmolytic streptococcus, which assumes an aerobic habit on culture within forty-eight hours.

The characteristics of this type of ulcer are these. After an initial stage of local infection extension occurs slowly but persistently below the skin, which at first shows little gross alteration. Later daughter ulcers and peripheral sinuses make their appearance and infection travels along vessels, nerves and fascial planes. The base is covered with greyish, gelatinous, anæmic and shaggy granulations. Clinically there is mild pyrexia and moderate pain.

Treatment consists first in a thorough exposure of all ramifications, pockets and sinuses and second in syringing a suspension of zinc peroxide cream into every part of the wound, which is then lightly packed with gauze soaked in this chemical product (p. 177).

**TREATMENT.**—1. *Prophylactic* treatment, if taken in hand properly, will prevent almost all chronic ulcers. Pressure ulcers should be avoided by care in the application of splints and plaster bandages, by the protection of prominent areas and by careful nursing. Special attention is needed in all very seriously ill patients to preserve the skin over the bony points in the back. The appearance of a bed-sore in a paralysed patient cannot always be avoided, but it is rightly regarded in most cases as a serious reflection on the skill of both doctors and nurses. First, the skin must be kept clean by washing with soap and water three times a day, after which it is carefully dried; secondly, three minutes are spent in *gentle* rubbing of the whole area with a stimulating lotion or a mild antiseptic ointment; thirdly, hardening of the skin is developed by the application of spirit lotion or eau-de-Cologne. If there is incontinence of fæces or urine, the routine must be gone through every time soiling occurs. The patient should lie on a water or air bed, no creases or seams should be allowed in the sheets, and crumbs or other foreign bodies in the bed avoided.

2. *Removal* of the Cause may apply equally to prevention and treatment. Jagged teeth, foreign bodies and all sources of infection are to be removed and all predisposing causes treated, *e.g.*, varicose veins should be injected in the neighbourhood of varicose ulcers.

3. *Treatment* of the Ulcer itself. Many ulcers are due to causes which demand specific treatment. for example, syphilitic ulcers. The treatment of an acute ulcer has been described, and is a very simple matter, whereas the chronic ulcer may be indolent and very unsatis-

factory to treat. The cause of the chronicity should first be investigated, and every effort made to remove it.

*Local Applications.*—The ulcer should first be cleaned up, all sloughs and debris being removed or encouraged to separate by hot boracic fomentations for forty-eight hours followed by eusol dressings for the next few days. When the wound is clean, applications to stimulate the granulation tissue into activity are required. Lotio rubra is applied on several thicknesses of gauze, which are kept moist by frequent renewal; scarlet-red ointment is a powerful stimulant but it must not be allowed to spread over the skin and is applied on a piece of lint accurately cut to fit into the ulcer, the surrounding skin being protected by a mild antiseptic ointment generously applied. Pepper's ointment is another valuable preparation; especially so in bed-sores. Infra-red radiation and short-wave therapy produce healing in apparently hopelessly incurable ulcers even when neuropathic, and are the most powerful therapeutic agents available. They should be given twice daily under skilled supervision. Immobilisation of the affected part and rest in bed will accelerate healing, but it is not always possible to keep patients off their feet. Dickson Wright has shown that varicose ulcers can be induced to heal by firm strapping of the foot, ankle and leg with elastoplast. This is easier to apply and is more satisfactory than the old Unna's paste stocking.

Recently the treatment of wounds and acute inflammations by chemotherapy and immobilisation has been directed to more chronic lesions. Indolent ulcers resisting less radical treatment should be insufflated with sulphanilamide powder and completely immobilised in plaster of Paris.

*Operative Measures.*—Under certain conditions local excision and suture may be the ideal treatment. If an ulcer is easily accessible and has not responded to treatment, and if no condition is present to delay healing in the scar, excision should be considered. Amputation may become advisable because of the large extent of the ulcer, its resistance to treatment, its persistent recurrences, the presence of pseudo-elephantiasis or the onset of carcinoma. Skin grafting will hasten healing, cut short a tedious convalescence and prevent subsequent deformity, provided that the conditions are suitable. In chronic ulceration the conditions are rarely suitable, for the causes of the chronicity are just those which militate against the success of grafting. It will therefore be reserved for those patients in whom the chronicity has been overcome and the surface is in a healthy, healing state.

*Skin Grafting* is the process of transplantation of the whole or part of the thickness of the skin from a healthy area to a granulating surface in order to bring about more rapid epithelialisation of the latter, and to obtain a more pliable scar, which will not contract and produce deformities. The area to be grafted must be clean and healthy and the infection under control. This is estimated by the number of organisms counted in loops of discharge taken from the surface. The wound should be dressed with normal saline for at least seventy-two hours before grafting. There are four methods of grafting:—



1. *Reverdin's Method* is the oldest, and has returned somewhat to favour of recent years. An area of skin is lifted up with the point of a needle, a pair of fine-pointed forceps, or by a hair and a small islet of superficial skin snipped with sharp-pointed scissors. Numerous islets are cut and planted out on the granulating bed.

2. *Thiersch's Method* is of great value and is in most common use.



FIG. 65

A, Large thick razor graft cut from outer side of child's thigh with simple knife-and-board technique

B, Removing the graft Its opacity indicates its thick character

C, Thin razor graft cut from inside of right arm, using simple knife-and-board technique

D, Dividing the graft Note the thinness of the graft

(*Surgery of Modern Warfare*)

Strips of cuticle, 3 in. by 2 in., are cut with a razor from the skin of the thigh so as to include the tips of the papillæ of the true skin (Fig. 65). These are laid on the surface to be grafted in such a way that each overlaps its neighbour and the peripheral grafts overlap the skin edge. Care must be taken to prevent the rolling in of the edges and to express every bubble of gas from beneath the grafts which are maintained in position with a wide-meshed gauze impregnated with medicated vaseline (*e.g.*, tulle gras), and are left undisturbed for ten days.

It is important that the graft be pressed evenly and firmly against

its bed. When the walls of a cavity are to be covered, the graft must be held in place by casts made of paraffin impregnated gauze or dental stent.

3. *Wolfe's Graft* comprises the whole thickness of the skin from which all subcutaneous fat must have been removed. It is sutured to the freshly trimmed skin edge, but its vitality cannot be relied upon and many fail to take.

4. *Pedicle Grafting* is of two types. The fixed base pedicle graft is exemplified by the raising of a flap of skin from the abdominal wall with its base undisturbed. Its free edges are sutured to the margins of the raw area to be grafted on the hand or arm, which is held in position by a plaster bandage. The base is divided ten days later, by which time the graft has obtained a blood supply from its new

bed. The tubular or movable pedicle graft is produced by a many-stage operation, the first of which aims at raising a strip of skin from the deep fascia and suturing its parallel edges together to form a tube of skin containing subcutaneous fat and being attached to normal skin at each end. At the end of a fortnight this pedicle contains a central artery, which permits of a flap of skin being raised at one end of the tube and being swung into position some distance away to repair defects of considerable size.

THE COMPLICATIONS arising in and from chronic ulceration have been dealt with, except the change to a malignant growth. Marjolin's ulcer (Fig. 66) is the name given to a squamous-celled carcinoma



FIG. 66.

Marjolin's ulcer. A squamous-celled carcinoma which has arisen on an old chronic varicose ulcer.

arising in the edge of a chronic ulcer or in a cicatrix. An increase in the induration at any one point in the edge of an ulcer combined with eversion should raise the suspicion of malignancy.

Specific types of ulceration are described in the regional sections of this book.

## GANGRENE

Death of soft tissue in the human body is called "sloughing" and the dead area a "slough." Sloughing of visible pieces of bone is termed "necrosis" and the dead bone is a "sequestrum." Gangrene implies the death *en masse* of a functioning unit of the body such as a toe, a finger, a foot, a hand, a coil of intestine or a testis.

### THE SIGNS OF GANGRENE

These are (1) loss of pulsation in the arteries of the part ; (2) failure of the colour to return to the skin after pressure ; (3) loss of heat, the part being dead cold ; (4) loss of sensation as soon as the nerves are dead ; (5) loss of function, the limb being motionless or the bowel showing no peristalsis ; and (6) changes in colour depending on the conditions present, varying from deep violet mottling to dead white. Before the parts are actually dead there is intense pain in the dying nerves, and after gangrene has occurred pain may be referred to the dead part.

### TYPES OF GANGRENE

In **Threatened Gangrene** the above signs are present in a mild degree and urgent treatment is called for to prevent tissue death occurring.

**Dry Gangrene** develops in the absence of infection, when the final cutting-off of the blood supply has been preceded by a gradually increasing arterial obstruction, as the result of which the veins and lymphatics have become adapted to remove the tissue fluids from the limb without the assistance of the normal *vis a tergo*. The affected part becomes shrivelled, hard, wrinkled and dry ; the disintegrating blood pigments turn it black and the skin assumes a waxy transparency. The dead tissues are separated from the living by an ulcerating "line of demarcation," which is produced by active granulation tissue advancing into the dead tissue from the living margins, at the expense of which the separation is mainly achieved. Each layer of tissue in a limb is not necessarily separated at the same level, the bone in particular surviving to a more distal point, so that the stump after natural separation is likely to be a conical one with the bone protruding beyond the skin. If an extension of the gangrene should occur, it is due to a repetition of the original cause at a higher level in the vessels, and the area affected will increase not by local spread but by graduated leaps. It is always associated with severe pain produced by the dying nerves.

**Moist Gangrene** results from the sudden cessation of the arterial blood supply to a part which remains full of blood, either because the main veins are also obstructed or because the flow in them cannot be maintained in the absence of the normal *vis a tergo*. Two types of moist gangrene are seen.

A. **ASEPTIC MOIST GANGRENE** occurs in the absence of infection when the arterial supply is abruptly cut off without any previous gradual obstruction, and when the death and disintegration of a large mass of soft tissues causes fluid to collect beneath the skin more quickly than it can evaporate. This is sometimes seen in senile gangrene of the lower extremity in which, while dry gangrene affects the foot, aseptic moist gangrene appears in the calf. The parts may show a variety of colours from black, purple, green and yellow to dead white. There is little change in the size, shape and consistency of the limb, and if perfect asepsis is maintained, separation occurs by a zone of

aseptic ulceration and the dead tissue is quietly cast off. In a very few examples evaporation occurs sufficiently rapidly to convert the moist into the dry type of gangrene.

**B. SEPTIC MOIST GANGRENE** is much more common. It results either from the infection of dead tissue in aseptic moist gangrene, and very rarely in dry gangrene, or from the infection of living tissues by certain organisms which cause the death of the part. The onset of gangrene may be encouraged by the lowered vitality of the tissues from other conditions, as diabetes, prolonged illness and neurotrophic lesions. The limb becomes greatly swollen and purple, green and yellow in colour. Blisters containing foul-smelling exudate form in the skin, the muscles are emphysematous from gaseous decomposition, and all soft tissues can easily be lacerated. The surrounding zone of living tissue is invaded by the inflammatory process, the patient rapidly becomes gravely ill and death from septicæmia often follows. If the gangrene spreads it does so by direct extension of the infection into the adjacent tissues. If the patient lives, a broad line of demarcation is formed and separation occurs chiefly at the expense of the living tissues.

### THE CHANGES WHICH FOLLOW GANGRENE

These are due to the reaction between the dead and the living tissues, and the dead will suffer one of three fates :—

1. **Absorption.**—Small areas of dead tissue will be absorbed by healthy granulation tissue containing foreign body giant cells in exactly the same way as the body deals with a catgut suture.

2. **Separation by Aseptic Ulceration.**—The “line of demarcation” is produced by granulation tissue growing from the living margin and digesting the dead tissue. A point is soon reached beyond which the nutrition of the advancing granulation tissue cannot be maintained and a line of ulceration is produced. The blood supply of the bone is more extensive than that of the soft tissues, and the bone separates at a lower level, as a result of which a “conical stump” forms.

3. **Separation by Septic Ulceration.**—The line of demarcation is produced by active suppuration in the living margin, due to its invasion by the infection from the dead tissues. The final line of separation is definitely higher in the limb than the original level of the gangrene.

### GENERAL PRINCIPLES OF TREATMENT

In dry gangrene, every effort must be made to keep the dead tissues dry and aseptic, and in moist aseptic gangrene to keep them aseptic and to encourage and assist them to become dry. The limb is lightly bandaged over a generous swathing of sterilised cotton-wool and the opposite limb exposed to hot dry air for half an hour during each four hours of the day. The general health must be supported and any intercurrent disease appropriately treated. The relief of pain becomes a serious problem, and amputation may be called for because the pain is so severe as to impair the general health and resistance. Natural

separation takes some weeks, and it is undesirable to continue the use of morphia over so long a period, while other drugs may fail to give relief. The details of treatment and the indications for surgical intervention are dealt with under the various types of gangrene. In septic moist gangrene the severe inflammatory reaction rather than the gangrene dictates the course of treatment.

### SPECIAL VARIETIES OF GANGRENE

The classification of gangrene is not easy because more than one factor enters into so many of the varieties. The following is based on etiology and will be found both simple and practical.

#### A. Vascular, *i.e.*, Ischæmic—

##### 1. Primary arterial degeneration :

- |                          |   |                       |
|--------------------------|---|-----------------------|
| (a) Senile . . . . .     | { | Atheroma.             |
|                          |   | Arterio-sclerosis.    |
|                          |   | Monckeberg's disease. |
| (b) Non-senile . . . . . |   | Syphilitic and toxic. |

##### 2. Pressure on the wall of an artery.

##### 3. Thrombosis and thrombo-angiitis obliterans.

##### 4. Embolism.

- |                        |   |                    |
|------------------------|---|--------------------|
| 5. Vasomotor . . . . . | { | Raynaud's disease. |
|                        |   | Ergotism.          |
|                        |   | Carbolic acid.     |

##### 6. Traumatic.

#### B. Infective—

- |                                    |   |                       |
|------------------------------------|---|-----------------------|
| 1. Acute pyogenic . . . . .        | { | Boils and carbuncles. |
|                                    |   | Cancrum oris.         |
|                                    |   | Noma vulvæ.           |
|                                    |   | Phagedæna.            |
|                                    |   | Spreading gangrene.   |
| 2. Gas-forming organisms . . . . . |   | Gas gangrene.         |

#### C. Traumatic (in reality vascular)—

1. Direct.
2. Indirect.

#### D. Thermal, Electrical and Chemical—

1. Burns and scalds.
2. Frost-bite.
3. Lightning.
4. High frequency electrical currents, *e.g.*, diathermy.
5. Escharotics.

#### E. Toxic—

- Diabetic.
- Ergot poisoning.

#### F. Neuropathic.

### VASCULAR GANGRENE

**Senile Gangrene** is the commonest form of gangrene in civil practice, occurring in both sexes after the age of 55 years. It usually affects the toes and feet, but is also met with in the hand, nose and ears. The predisposing causes are: (1) degeneration of the smaller arteries with or without atheroma in the larger trunks; (2) weak heart action and low blood pressure; and (3) lowering of the local nutrition and general resistance by anæmia, nephritis, diabetes and other diseases. The blood supply has therefore been slowly but progressively diminished and gangrene, when it supervenes, is of the dry type. The determining factors are those which result in thrombosis



FIG. 67

Senile dry gangrene of the foot.

either in the peripheral arteries or in the main trunks. Thrombosis of the large vessels may be due to slight injury or to the deposit of clot on a diseased vascular endothelial lining. Cutting a corn, the rubbing of a toe by a new boot, knocking the foot against a chair or table or exposure to cold may be sufficient to precipitate a block of the smaller arteries. The extent of the gangrene obviously depends on the level to which the arterial obstruction extends (Fig. 67).

*Symptoms*.—The patient will have complained of painful cramps in the calf after walking short distances, of numbness and “pins and needles” and a sensation of coldness in the legs. The gangrene starts as an area of redness and inflammation, which later becomes dry and shrivelled. The dying tissues are always the seat of severe pain, but in the early stages the general condition is good, whereas later, exhaustion from the continual pain, lack of sleep and toxic absorption lead to fever, restlessness, wasting and delirium. Finally cardiac, renal or pulmonary complications usher in the end.

*Treatment*.—Threatened gangrene is treated on general principles,

viz., rest, warmth and protection. Periarterial sympathectomy has been advised but is definitely disappointing, and better results may be obtained from lumbar ganglionectomy. When gangrene is present an amputation should be performed, except in the presence of some very definite contraindication. The long drawn-out agony of natural separation cannot be contemplated when amputation does away with the gangrenous area at once. Local amputation near the dead area always fails, as it determines further thrombosis and gangrene spreads in the flaps. Faraboeuf's amputation through the site of election in the leg and the supracondylar amputation above the knee are the methods of choice. Some surgeons advise the latter as a routine, but the former gives better results if the arterial changes are not too advanced. A simple test provides an indication of the level in the limb at which amputation flaps will be well nourished. An Esmarch bandage is applied firmly to the limb from below upwards and a rubber tourniquet is then placed round the thigh just below the groin. The bandage is removed and the limb appears dead white. On the removal of the tourniquet a pink flush returns quickly to a certain level, below which the colour comes back very slowly and imperfectly. This shows the level at which it is safe to amputate.

**Non-senile Gangrene** is rare. It is due to endarteritis obliterans produced by syphilis, typhoid or other toxic states. The femoral artery is thrombosed and the clot may spread to the aorta, in which case gangrene of both legs results.

**Gangrene due to Pressure.**—Large arterial trunks may be pressed upon by tumours, *e.g.*, growths or sacculated aneurysms; the subclavian artery may be compressed by a cervical rib and gangrene of the finger-tips may follow; or the vessels of a limb may be obstructed by too tight bandaging or splinting. This type of gangrene is always dry in type, and its treatment is directed to the removal of the cause.

**Gangrene due to Thrombosis and Thrombo-angiitis Obliterans** will be described under those headings on pp. 278 and 265. Thrombosis is the final determining factor in all cases of gangrene due to arterial disease in either the smaller or the larger vessels. In thrombo-angiitis clotting takes place in both artery and vein, but the gangrene is usually dry.

**Embolic Gangrene** is described on p. 279. It will occur only in those patients who are debilitated from previous illness or who are already suffering from arterial disease, because in healthy people the lodgment of an embolus is exactly analogous to ligature of a vessel and the rapid establishment of the collateral circulation maintains the blood supply of the limb. Gangrene of this type (Fig. 68) is moist and septic, and a foul-smelling mass results. In the early stages (*i.e.*, up to twelve hours) before the onset of actual gangrene, an attempt should be made to save the limb by the removal of the clot from the vessel, an operation known as embolectomy, which has had some encouraging results. After a few hours tissue death will have occurred and an amputation will be required.

**Vasomotor Gangrene.**—RAYNAUD'S DISEASE affects neurotic women between the ages of 20 and 40 years, and is due to an intermittent

spasm of the smaller arteries. The fingers and toes are most commonly involved and the disease is usually bilateral and often symmetrical. There are three stages, viz.: (1) Local syncope of the fingers or toes, which are white, numb and cold; (2) local asphyxia, the parts being congested; and (3) local gangrene of the dry type. The disease may exist for months or years without progressing to the stage of gangrene. Treatment consists in a cervical or lumbar ganglionectomy, or local amputation once gangrene has become established.



FIG. 68

Bilateral gangrene due to embolism in a man of 55 years. The gangrene has reached two-thirds of the way up each leg.

ERGOTISM is unknown in this country at the present time. It produces a dry form of gangrene.

CARBOLIC ACID applied externally as a compress used to be a common cause of gangrene of the fingers, and for this reason it has been entirely given up as a dressing. Gangrene due to this agent should never be seen now.

### INFECTIVE GANGRENE

**Gangrene due to Pyogenic Infections** occurs in boils, carbuncles, cancrum oris, noma vulvæ, phagedena and a special type known as spreading gangrene. Boils and carbuncles are described on p. 231.

CANCERUM ORIS is an acute, infective, gangrenous stomatitis, which affects young children living in insanitary conditions and is fortunately but rarely seen. The children are always weakly and ailing and often recovering from one of the infectious fevers, especially measles. The infection, which is usually a mixture of staphylococcus, streptococcus and the spirillum of Vincent's angina, gains entrance through a breach in the mucous membrane of the cheek or lip. The mouth is already in a foul state with carious teeth and infected gums, and so virulent is the infection and so poor is the resistance of the patient that



the gangrene spreads with alarming rapidity, involves the whole thickness of the cheek and exposes the alveolar margin. There is a foul discharge, much of which is swallowed, and the smell is both nauseous and penetrating. The progress of the infection is marked by rigors and a high temperature, by toxæmia and later by septicæmia. The prognosis is extremely grave.

*Treatment* is prophylactic, that is, in attention to the general health and to dental caries. The steady improvement in conditions of living and the admirable work of the infant welfare organisations has practically eliminated this terrible disease. In the early stages energetic treatment with sulphanilamide and short-wave therapy may succeed in localising the infection. Later all infected tissue must be excised and the raw surfaces cauterised with pure carbolic acid. The resulting deformities will require skin grafting or plastic reconstruction.

NOMA VULVÆ is a similar condition affecting the external genitals of young children, and in spite of its name, can occur in boys. It has also become practically non-existent, but in recent years there have been some examples in fever hospitals among children gravely ill with measles.

PHAGEDENA, or hospital gangrene, was only too common in the pre-antiseptic era. It rarely occurs in this country to-day, and the term is used chiefly in connection with the destructive ulceration of the penis, which occasionally complicates venereal infection.

SPREADING GANGRENE was once synonymous with hospital gangrene but, the latter having become extinct, it is now used to describe a special type of lesion. It is a complication of wounds of the trunk, usually those established for drainage of deep-seated abscesses. In the author's personal experience it has been met with only in empyema wounds and in every case those accompanied by a pleuro-bronchial fistula.

It is described by Frank Meleney as being caused by a symbiosis of an anaerobic non-hæmolytic streptococcus with a hæmolytic staphylococcus aureus: he has named the process "synergistic gangrene." At the edge of a previously healthy wound a thin bright red line suddenly appears and the skin margin becomes everted and raised. This zone, never more than  $\frac{1}{2}$  in. broad, has a surround of pale pink hyperæmia into which it rapidly and persistently advances. As it progresses the tissues left behind become grey white, greatly thickened and indurated. These changes affect the whole depth of both skin and subcutaneous tissues but never go deeper than this. In the absence of treatment the gangrene spreads irresistibly.

Treatment must be immediate and drastic. No matter how extensive it may be, the whole area must be excised with a  $\frac{1}{4}$  in. of normal skin. The wound edge is then undercut for another  $\frac{1}{4}$  in. and the recess thus formed and the whole surface is packed with Meleney's zinc peroxide cream. This must have a high free oxygen content (40 per cent.) and can be obtained in this country only from the firm of Messrs Laporte of Luton, Bedfordshire.

**Gas Gangrene** is described on pp. 35 to 38.

### TRAUMATIC GANGRENE

**Direct Traumatic Gangrene** is the result of an injury which destroys the vessels within the local zone of trauma, when the parts distal to the injury are deprived of their nutrition and die. Such death of tissue is seen in severe crushes in which a limb is injured by a heavy weight, by the moving parts of machinery or by the wheels of vehicles. The tissues are crushed or pulped and the vessels torn, lacerated, crushed or thrombosed. Gangrene is not likely to follow in young healthy people, but in the old and frail a moist type may set in.

The pressure of splints, plasters and bandages may produce local gangrene as has already been described (p. 166), and in the production of a bed-sore direct trauma plays some part.

**Indirect Traumatic Gangrene** is produced by an injury which obstructs the main vessels, and the parts, which die, are at some distance from the vascular lesion. The causes are :

1. Ligature of a main artery, which will cause gangrene only if the parts are already unhealthy from long-standing arterial disease. It is seen only in the lower extremity, in which the toes will be the seat of dry gangrene. This type may be prevented in a certain number of cases by simultaneous ligature of the vein. When gangrene is established, the part should be kept dry and aseptic until a well-marked line of demarcation is present, and then the dead tissue removed by an amputation just above it.

2. Sudden occlusion of both artery and vein will lead to moist gangrene in the majority of people, though in the young and healthy the collateral circulation may develop sufficiently rapidly to restore the nutrition of the limb. Gangrene does not occur if there has been an old long-standing arterial obstruction, because the collateral circulation is already in full service.

Occlusion of both vessels by external means is seen in a strangulated hernia, torsion of the testis, strangulation of the penis by a ligature round the base, or of a finger by a ring which is too tight. The gangrene will be moist or dry according to special circumstances, and amputation or resection will be needed.

3. Subcutaneous rupture of a vessel may lead to gangrene from compression of the neighbouring main vessels by extravasated blood. This should be prevented by ligature of the ruptured vessel and removal of the clot.

### TOXIC GANGRENE

**Diabetic Gangrene.**—Three factors are at work in the production of gangrene in patients with diabetes mellitus : an arterial degeneration, especially in the anterior and posterior tibial vessels, a diabetic neuritis and a condition of the blood favourable to the development of sepsis. Gangrene does not occur in young people with diabetes, and it is probable that the most potent factor is the arterial change of a senile type. It is important to distinguish between true diabetic gangrene and senile gangrene associated with a toxic glycosuria.

The gangrene starts in any point of sepsis which has followed trivial

injury or the cutting of a corn, and it spreads rapidly. It is moist, very foul-smelling and leads to extensive sloughing of the skin. There is so much inflammation in the surrounding living tissues that there is no clear line of demarcation. Pain is usually severe owing to the diabetic neuritis and the gangrene may precipitate an attack of coma. The prognosis is always poor and amputation should be performed at the earliest opportunity.

**Thermochemical Gangrene** has been described on pp. 129 to 141. and **Neuropathic Gangrene** likewise on p. 166.

R. M. HANDFIELD-JONES.

## CHAPTER X

### GENERAL SURGICAL TECHNIQUE

**T**HE student entering the surgical wards to-day for the first time finds it difficult to believe that modern surgery is but seventy years old, and he can have no conception of the "terror" that was surgery before the introduction of the antiseptic and aseptic era. The world owes to Louis Pasteur the discovery that disease was due to microbic invasion, and to Lord Lister the application of that observation to the revolution of surgery and the relief of untold millions of human beings.

#### ANTISEPTIC SURGERY

This premises the presence of organisms in a wound and seeks to destroy them or to prevent their growth by chemical means. Some of these antiseptics are true germicides, but others are capable only of preventing the multiplication of organisms, whilst some potent antiseptics are inapplicable to the human being owing to their toxicity. Antiseptic methods of sterilisation are still employed for certain purposes but in operative technique they have been replaced by asepsis. The following are among the most useful antiseptics in common use to-day.

**Carbolic Acid** belongs to the Listerian epoch ; it is so potent and so destructive of human tissue that its use is restricted to certain specific purposes. Pure carbolic acid is used in minute quantities as a cauterising agent to sterilise septic areas, *e.g.*, the stump of an appendix after its removal ; it is used in solutions of glycerin in gynaecological practice as a paint for the interior of the uterus after curettage, and in very dilute solutions as the basis for gargles and throat sprays. It must never be used in a compress or dressing on the skin for fear of its producing ulceration or gangrene.

**Binioidide of Mercury** is extremely toxic and must never be used in open wounds, but it is a valuable agent for the sterilisation of the patient's skin and the surgeon's hands. It is used either as an aqueous solution (1 : 1000), or in alcohol (1 : 500 parts of 90 per cent. alcohol). It is the universal custom to tint this solution with eosin.

**Iodine** is the most popular of all antiseptics, but it is doubtful if its virtues justify its expense. It is used in 2, 2½ and 5 per cent. solutions in alcohol chiefly in the routine preparation of the skin before operation. It is quite useless unless the fats of the skin have previously been removed by acetone or ether. It is also the inevitable standby in every first-aid post for all wounds.

**Picric Acid** is also used to prepare the skin before operation, as a 2 per cent. solution in alcohol. It has the great disadvantage that its yellow stain persists for several weeks. It used also to be a popular application as a 1 per cent. solution in the treatment of burns.

**Alcohol** is rapidly going out of favour owing to its high cost, and the doubt thrown by bacteriologists upon its antiseptic value. Industrial alcohol remains a useful, if extravagant, method of sterilising needles, scalpels, scissors and skin.

**Dettol** has become firmly established as a useful all-round antiseptic and in war time has practically replaced alcohol. As an aqueous solution or cream it is used for preparing the skin both of the patient and the surgeon, being especially popular with gynaecologists. It has an added virtue in not damaging the hands however lavishly and frequently used.

**Eusol, Dakin's Solution, Chloramine T or Di-Chloramine T.** are members of the hypochlorite group of antiseptics, which act by virtue of their strong oxidising action. They are not very stable in solution or when warm, but have a place in the treatment of infected wounds by continuous irrigation.

**T.C.P.** (Trichlorophenylmethylidosalicyl) is another proprietary preparation greatly in favour, especially among dental surgeons and as a prophylactic against laryngeal and pharyngeal infections. It may be used both for internal and external medication, wounds and mucous surfaces.

**Flavine**—a coal-tar derivative—is used either as an aqueous solution (1 : 1000) or in suspension in paraffin. It is an admirable dressing for open wounds, particularly just after the worst of the infection has passed off and the period of regeneration set in.

**Hydrogen Peroxide** is another agent which acts chiefly by virtue of its oxidising action. It is useful in dealing with anaerobic infections and in the removal of dressings which, caked with dried blood and discharge, are adherent to the wound surfaces.

**Lysol** is both cheap and effective. It is highly poisonous, however, and should not be allowed to come in contact with the skin.

**Formalin** is not suitable as an antiseptic in contact with the tissues, but it has a special place in the sterilisation of such surgical equipment as cannot be boiled, and is used for all gum elastic catheters and bougies, etc. Owing to its irritant nature, these instruments must be washed in a mild sterile solution before use.

**Sulphanilamide** powder has been extensively used in war wounds and its use and value are discussed on p. 38.

### ASEPSIS

Asepsis aims at preventing the entrance of organisms into wounds and so dispensing with the use of antiseptics, all of which are likely to injure living tissues if they are of any real value in the destruction of the infecting organisms.

Pathogenic organisms are introduced into the human body from without, and if everything which is to come in contact with the

patient's tissues has been sterilised before use, the risk of introducing micro-organisms is reduced to a minimum. The most efficient method of killing bacteria and their spores is the use of heat, either by boiling indestructible instruments or by exposing towels, gowns, dressings, etc., to steam. In large hospitals all sterilisation is done in one main central high-pressure plant, in which superheated steam is the active agent. Smaller plants, in which the pressure is lower, are available and are as efficient, though not so rapid in their action. All operation and dressing material is placed in special drums, of which the outer case is double and so perforated and able to slide that the apertures can be made to coincide or close as required. They are loosely packed with dressings and loaded into the steriliser with the openings coinciding to allow full access to the steam. After their removal the outer case is closed and the drums stored for use.

Two essential elements in every operation can never be sterilised except by chemical antiseptics, viz., the patient's skin and the surgeon's hands, but with these exceptions modern surgical technique is based on the principles of asepsis. The technique of individual surgeons must necessarily differ in minor details, but the broad outlines of theatre management are uniformly adopted.

### THE OPERATING THEATRE

The theatre in a large hospital is part of a suite of rooms. These include a surgeon's changing-room and bathroom, one or more rooms for the storage of various requisites and suitably arranged with hot cupboards in which to keep large flasks of sterile saline, a room for needlework and the repair of instruments and gloves, a room reserved for the induction of anæsthesia, and finally the operating theatre itself. This should have three annexes communicating with it by doorless arches, one containing the sterilisers (enclosed in draught cupboards) for the instruments, bowls and saline solutions, a second with hand basins, and drum holders where the surgeon and his assistants "scrub up" and robe, and a third fitted with sinks for the reception, collection and disposal of all dirty towels, dressings, used lotions, etc.

The theatre should be as small as comfort and efficiency permit. It must be equipped with a heating system capable of maintaining a temperature up to 80° F., its floor and walls are to be of polished stone, glazed brick or white tiles and all corners between the walls and floor must be rounded off. The floor should slope to one side towards a shallow gulley for drainage. The only fixtures on the walls should be the electric fittings for light and power, a tube for attachment to the suction apparatus and an X-ray viewing box. If an observation gallery is provided it should run round three sides of the theatre at a height of 7 ft. 6 in. above the floor, be quite narrow, screened breast high, and reached by a staircase outside the theatre. The fourth wall should, if possible, face north and be occupied completely by a window, which should be fitted with an adjustable dark blind. The table is made of metal and is adjustable to all positions required in operations. Tables for instruments, for swabs and lotions and for

the anæsthetist's equipment are constructed of tubular metal framework with glass shelves. Drum holders are of that pattern which allows the lid to be lifted by a foot pedal. Glass shelves for storage of catgut, drainage tubes, scalpels, etc., should be in a small recess off and not actually in the theatre.

Artificial lighting by shadowless electric lamps is the best type of illumination and some arrangement of accumulator and lamps must be installed in case of a general breakdown. In view of the highly explosive nature of ether vapour and of the many recent serious accidents, it is considered undesirable to have any naked light or electric spark in the theatre.

No visitor should be allowed on the floor, which must be reserved for those actually engaged in the operation and for students under instruction. In addition to such sterile clothes as may be necessary, all persons employed on the floor should have special footwear (white canvas shoes, rubber boots or goloshes), which are never allowed to leave the theatre premises.

It is not always possible to work in such ideal surroundings, but the best surgery can still be done in poor conditions if general principles are adhered to. In a private house a large, light and airy room can be easily converted. The carpets and curtains must be taken away, the walls and floors scrubbed and all furniture removed. The room is then well aired and heated, the floor covered with several layers of newspaper and a dust-sheet temporarily nailed in place over them. All that need be provided in the house is an ample supply of boiled water, both hot and cold. The surgeon's theatre sister will bring all the bowls ready sterilised as well as the instruments and dressings.

**Emergency Theatres.**—The foregoing description of an ideal is something to aim at, but not always achieved. The bombing of cities has led to improvisation of emergency theatres, with none of the peacetime luxury equipment. But the work has been as good as in other and happier conditions.

### THE SURGEON AND HIS STAFF

In every case the surgeon should change completely, if possible into white duck trousers, a white short-sleeved shirt and white canvas shoes, or failing this, into grey flannel trousers, white cricket shirt and white shoes. He then proceeds to scrub his hands and forearms up to the elbows under a spray of hot water with a nail brush, which has been sterilised by boiling. This process must last for at least five minutes, during which he pays particular attention to the nails and to every part of the fingers and hand. Any good soap will suffice, and the use of ether or other antiseptic soap is quite unnecessary. He is then clad in a sterile gown, mask and cap. The sleeves end in elastic cuffs which should reach easily with full play to the wrists, and the cap and mask cover the head and face so that only the eyes are unveiled. Finally, a pair of rubber gloves is drawn on to the hands. All his assistants are similarly prepared. Nurses not taking an active part, but acting as "*runners*" for the sister, are clad in sterile gowns with

their heads veiled. The anæsthetist should wear a gown, cap and mask, but these need not be sterile except for operations above the level of the clavicles.

### STERILISATION OF MATERIALS

**A. Instruments** are to-day invariably made of stainless steel. They are boiled in water for at least ten minutes, or if they have previously been used for a septic case, twenty minutes must be allowed. After boiling they are placed on a sterile towel on the instrument table. Any instrument which drops to the floor during the operation must be reboiled if needed again. After use all instruments are thoroughly scrubbed with a stiff brush, special care being given to the serrations of forceps, etc. They are then boiled, dried and replaced in the cabinet.

**B. Rubber Gloves** are more pleasant to wear if dry-sterilised, but this can be done only with special care under low-pressure sterilisation as otherwise the rubber perishes. In case of doubt it is safer that they should be boiled.

**C. Swabs** are made of butter muslin and are made up in several ways, *e.g.*, in small squares of 4, 6 or 8 thicknesses, and sewn together at the edges, or in loosely packed balls. Rolls of gauze and abdominal packs are made of similar material of different shapes and sizes to suit individual requirements. Towels are either white, green or red, and are made of calico. All abdominal packs should have black tapes attached for identification during operation, and small swabs should be put up in packets of six or ten so that they can be easily counted and checked. All these materials are sterilised by high-pressure steam sterilisation. Bowls, trays and dishes are boiled in a special container.

**D. Ligature Materials** are either absorbable or non-absorbable. The latter, except for skin sutures which can be removed, are permanently embedded in the tissues, in which they may act as an irritant or as a nidus for the settlement and development of micro-organisms; but they all have the great advantage of being boilable. The unabsorbable materials include silk, linen thread, silkworm gut, Japanese synthetic gut and horsehair.

**SILK AND LINEN THREAD** of varying sizes are wound loosely on glass spools and sterilised by high-pressure steam heat or by boiling, after which they are stored in glass jars in a solution of biniodide or in spirit. The spools should be boiled immediately before use.

**SILKWORM GUT** is issued in three strengths and some manufacturers stain them with distinctive colours, the strong violet, the medium pink and the fine black. It is sterilised by boiling for at least five minutes immediately before use. A cheaper variety is the synthetic brand, which is always coloured green and which is not quite so strong as the natural variety; it is, however, more pliable and so easier to manage. The very fine black or ophthalmic silkworm gut is the best material for fine work, *e.g.*, in the face or neck. It should invariably be used in preference to horsehair, which no longer merits inclusion among suture materials, being difficult to sterilise and so elastic that it cannot compare with fine silkworm gut.

**CATGUT** is the absorbable ligature and suture material being made



from the submucous layer of the sheep's small intestine. It has the disadvantage of being ruined by boiling, and yet its very origin demands a most highly efficient method of sterilisation. It is probably true to say that no completely safe method will ever be found, but the preparation of catgut has recently been subjected to very stringent regulations by the Ministry of Health, and there are many excellent brands on the market. It is made in a number of thicknesses and by varying methods in preparation is graded as being absorbed by the tissues in ten, twenty or forty days. It is put up for sale in sealed glass tubes, which are immersed in spirit for fifteen minutes before use. Kangaroo tendon is rarely used and then for special purposes such as the reapposition of a fractured olecranon process. It is prepared by similar methods to those used for catgut.

### THE PREPARATION OF THE PATIENT

The **skin** should be shaved the night before operation and carefully washed with soap and hot water. It is dried with a sterile towel, swabbed with acetone and ether successively to remove all moisture and fatty secretions, and then painted with tincture of iodine, picric acid or spirit according to the custom of the surgeon concerned. The skin of some people is hypersensitive to iodine and a blister may result with much unnecessary pain, while picric acid leaves a yellow stain for several weeks. Ninety-five per cent. alcohol is probably the most satisfactory antiseptic to adopt for routine use. The whole area is now covered with a sterile towel and this is held in place by a bandage. The swabbing with alcohol is repeated just before the beginning of the operation.

Except for operations on the colon and rectum drastic **purgation** is needless, and if the patient's bowel action has been normal and regular, an enema on the previous evening is sufficient. Prolonged starvation is also harmful. Food should be withheld for four hours before operation but no longer, and during those four hours patients must be encouraged to eat as much barley sugar as they like. Smoking and alcohol should be absolutely forbidden for twenty-four hours before operation.

A full night's **sleep** is essential, and if the patient is not asleep by eleven o'clock, medinal gr. x should be given. Under no circumstances whatsoever should morphia be used for this purpose. It is also important to put patients' minds at rest, to relieve their anxieties and fears and to assure them of a successful outcome. The use of pre-anæsthetic medication will be discussed in the chapter on Anæsthesia.

### THE OPERATION

The patient, having been placed on the table, is covered entirely with sterile towels so that only the actual operation area is exposed. As soon as the skin incision is made, all bleeding points are picked up with forceps and ligated and the wound edges covered with hot packs, which are fixed in place by towel clips. The details of operative technique cannot be discussed here, but certain general principles are worthy of emphasis. No operation should be undertaken by any

surgeon unless he is capable of meeting with skill and dexterity any complication, however unforeseen, that may occur. The incision should be long enough to give adequate access. All exposed parts not essential to the particular stage of the operation are to be covered with hot, moist packs. Speed is always important, but gentle handling is far more so and it must never be sacrificed to a "flashy" rapidity. Clean cutting and gentle separation mark the good surgeon, and rough tearing the beginner. Bleeding should be reduced to a minimum and, whenever possible, vessels picked up in artery forceps before division.

At the completion of the operation the wound is covered with generous layers of gauze and wool, the whole being firmly bandaged in place. If no drainage has been necessary, the dressing should be changed on the second day, after which the wound is left undisturbed till the stitches are removed on the eighth or tenth day. If a drainage tube should have been inserted, the dressings may become saturated with blood and serous discharge during the first twenty-four hours, in which case they should not be removed, but more layers of wool or cellulose tissue placed over them and an additional bandage applied. At the end of another twenty-four hours the whole dressing must be removed, and from then onwards redressing be done daily. The after dressings are of great importance, and as much care is to be taken of the surgeon's hands and his instruments as at the operation. An undrained wound will give no anxiety, but a drained one can be easily infected with secondary organisms and the resulting mixed infection may have serious effects. The truth of this teaching has recently been emphasised by the Medical Research Council's memorandum on "Hospital Infections." It has been more fully alluded to on p. 128.

Many operation wounds need but little dressing, and patients are more comfortable without bulky bandages, especially in hot weather. After thyroidectomy, for example, the first dressing is removed on the second day, when the wound can be protected with a thin layer of gauze fixed with mastisol—a surgical glue. Clean abdominal incisions may be supported after the sixth day by wide strips of elastoplast strapping.

### POST-OPERATIVE TREATMENT

(On return from the theatre the patient is placed in bed, this having been heated during the operation by a radiant-heat cradle. While under the influence of the anæsthetic he is placed on one side with the knees drawn up and the head kept low in order to prevent any vomitus or secretion trickling down into the trachea. Six ounces of distilled water with gr. xx of aspirin and gr. lx of sodium bromide are run into the rectum. He is wrapped in a blanket, covered with the usual bedclothes and kept warm with hot-water bottles, applied *outside* the blanket. He is not to be left for a moment until he has regained consciousness, when he can be moved into the position demanded by the particular requirements of the operation.

**Shock** will be treated by the methods laid down in Chap. VIII.

**Relief of Pain.**—Few operations, however small, are unaccompanied by some pain, and in all those performed on the abdominal contents,

on bones and joints, on the thorax and the neck, pain will be considerable. No patient should be allowed to suffer pain unnecessarily, and equally no drugs should be given needlessly. Some patients will suffer severe pain stoically without a complaint, while others will behave as if a minor ache were a major disaster. It is one of the more important of the student's early lessons to be able to distinguish the two types. As soon as the effects of the anæsthetic have worn off completely, a hypodermic injection of morphia (gr.  $\frac{1}{4}$ ) will be needed by the majority of patients and a dose of gr.  $\frac{1}{8}$  may be repeated in six hours' time. After this it should not be necessary to use morphia or its derivatives, but reliance should be placed on other less powerful analgesics.

**Feeding.**—There will be no desire for food for the first day or two after operation, and no effort should be made to force food upon an unwilling patient, who can exist quite comfortably on small drinks of water at frequent intervals. Gastric resections, intestinal anastomoses and rectal operations demand procedures special to themselves, but after most operations patients may start to take eggs, milk and fish on the second and third day.

**Bowel Action.**—In ordinary cases, in which there has been no interference with the intestinal canal and there is no fear of peritonitis, a simple enema can be given on the evening of the day following operation, and this may be repeated the next day. On the third evening an aperient is administered and a saline draught in hot water given the next morning. A good action should result, but it must be remembered that after an operation the bowel will not respond to a normally efficacious dose; in fact, in many patients it is kinder to give castor oil in spite of its unpleasant taste. It acts so rapidly that it is not given until the early morning.

**Confinement to Bed.**—A great deal of needless suffering, weakness and muscular atony are due to keeping a patient too long in bed, and to a rigid restriction of movement even in bed. From the second day every patient is to be encouraged to move about in bed, to assist in the bed-making and in the rites of the bed-pan and the blanket-bath. After an uncomplicated operation for appendicitis five days in bed are sufficient, and ten to twelve days are ample for all abdominal operations, except those for the repair of hernia which require eighteen days for their after-care. If the finances of the patient allow, massage of the lower and upper extremities should be given from the third day onwards. If these principles were more universally adopted, there would be fewer examples of that terrible post-operative complication, pulmonary embolism.

**Breathing Exercises and Muscle Drill.**—Recently, instead of treating individual patients, we have instituted a twice-daily drill for all occupants of surgical wards. A member of the physiotherapeutic department is in charge and ward sisters have become keen co-operators. Deep breathing exercises help to banish pulmonary complications and patients quickly enter into the spirit of the thing. Muscle exercises for toes, legs, fingers and hands are also done by drill. The results are extremely encouraging.

R. M. HANDFIELD-JONES.

## CHAPTER XI

### ANÆSTHESIA

#### EXAMINATION OF PATIENT AND ITS BEARING ON THE CHOICE OF ANÆSTHETIC

**T**HE anæsthetist and his patient must not meet as strangers at the operation. It is most desirable that sympathetic relationship and confidence should have been established by a previous visit, especially if the patient is nervous and distrustful. The experienced anæsthetist will never undervalue this preliminary contact, which will enable him to decide the type of anæsthetic required.

Colour, build, adiposity, voice and type of respiration have each an immediate significance for the expert eye, and a rapid estimation of physical fitness can be made by inquiry into the range of the patient's usual bodily activity. Shortness of breath on exertion, inability to take strenuous exercise or to withstand fatigue, suggest, even in the absence of definite signs of disease, the wisdom of postponing operation; or, when this is not possible, such disability may call for special consideration in the operative procedure which is to follow.

In this connection it should be noted that perfect physical condition is not always a guarantee that anæsthesia will be safe. The young athlete in hard training is a case in point, and his anæsthetisation should be approached with the greatest possible care. Collapse, comparable to the sudden faint at the end of a race, is by no means unknown, and is most formidable when it does occur, complicated as it is by the toxic effect of anæsthesia; similarly the robust appearance of the alcoholic may deceive the less wary. Nevertheless, accurate observation and inquiry into matters of physical ability must not supersede adequate physical examination, and it is useful to consider each system of the body in selecting an anæsthetic.

**Central Nervous System.**—There is no organic nervous disease which contraindicates general anæsthesia. Epilepsy is no bar, but it should be remembered that a fit may suddenly occur, most often in the early stages of induction. In mental disease anæsthetics are well tolerated, but it is inadvisable to use the quick-acting anæsthetics—ethyl chloride and nitrous oxide—as they appear liable to cause maniacal outbursts during induction.

The eye should always be examined before an operation to exclude the possible presence of an Argyll-Robertson pupil. Such a precaution has saved the subject of a tabetic crisis from operation, while the insensitiveness of a well-fitting and undiscovered glass eye has been known to lead to premature activity on the part of the surgeon.

**The Digestive System.**—The mouth should be examined for sepsis, but whether septic teeth should be removed before the main operation is a debatable point. The general view appears to be that teeth should only be extracted if sufficient time is available for reasonable oral hygiene to be established. In urgent cases reliance must be placed in antiseptic mouth washes, though teeth which are actually loose should be removed.

Diabetes mellitus and liver insufficiency are the only alimentary conditions which demand the avoidance of a general anæsthetic. Chloroform must never be used, but ethyl chloride, nitrous oxide and oxygen or ether may be exhibited with care. It is always advisable for these patients to undergo a preparatory course of medical treatment, and for a physician to be available in case of post-operative toxæmia. The use of insulin has, of course, greatly minimised the immediate post-operative risk.

**Urinary System.**—The urine of every patient should be examined to exclude albumen, sugar, acetone and diacetic acid. A general anæsthetic does not appear to affect materially the course of nephritis, though the output of albumen may be temporarily increased. When chronic nephritis is present ether and nitrous oxide and oxygen are the anæsthetics of choice. The same precaution should be observed in glycosuria as in established diabetes. Acetonuria, commonly present in malnutrition, after severe vomiting, and in acute infection, contraindicates the use of chloroform.

The presence of acetone and diacetic acid in the urine indicates grave metabolic disturbance and forms a definite contraindication to a general anæsthetic. In children, however, an acute acidosis may accompany some urgent surgical condition, to deal with which a general anæsthetic may be necessary. In such emergencies ethyl chloride, which has been shown to have little or no effect on the blood sugar, is comparatively safe.

**Respiratory System.**—An acute infection of any part of the respiratory tract contraindicates the administration of a general anæsthetic, except in cases of urgent necessity. When this is the case, the choice of anæsthetic can be subject to no exact rules but must be decided as the operation progresses.

In chronic respiratory diseases the secretions should be dried up as far as possible by the judicious use of belladonna or small doses of atropine given for several days. Asthmatic subjects generally give little trouble during anæsthesia, but it should not be forgotten that an asthmatic attack may occur at any stage, and has been known to be fatal.

A definite history of pulmonary tuberculosis is an absolute contraindication to a general anæsthetic, so great is the risk of causing a recrudescence of the disease. Analgesia with preliminary narcotic medication should always be employed. It must be remembered that respiratory infections in general are liable to throw an undue strain on the right ventricle, and that cyanosis in these cases must never be lightly regarded.

**Circulatory System.**—Since all anæsthetics produce a phase of

cardiac depression succeeding preliminary stimulation, it is the duty of the anæsthetist to estimate, as accurately as possible, the capacity of the heart to resist the strain of the proposed operation. A careful inquiry into the patient's normal daily activity combined with a thorough physical examination should form a sound basis for an opinion.

An uncompensated or failing heart absolutely forbids a general anæsthetic, though valvular disease is not a contraindication if compensation is complete. Mitral and aortic incompetence, either separately or together, rarely give trouble if they do not cause inconvenience in *daily* life. On the other hand, mitral stenosis should always be regarded seriously, for the heart is liable to fail suddenly, and an infarct from the left auricle may cause sudden death.

Myocardial degeneration, characterised by a tic-tac rhythm, and the difficult distinction, by auscultation alone, of first and second sounds at the apex is a very serious condition. When general anæsthesia cannot be avoided, the anæsthetist should try to keep the pulse rate as nearly normal as possible by the judicious use of chloroform and ether. Gas alone, or gas and oxygen should be used with the greatest caution.

Cardiac irregularity may be due to so many causes that when there is doubt a cardiologist should be called in. The response of the heart to exertion serves as a fairly reliable indication of its probable behaviour under anæsthesia.

Hyperpiesis is not in itself a contraindication, though cases of cerebral hæmorrhage sometimes occur during anæsthesia, in which the responsibility of the anæsthetic cannot be always disproved. Provided great care is exercised in avoiding cyanosis, these cases do well, though special care with nitrous oxide is advisable. A week's rest in bed before operation with a light diet and saline laxatives is of great advantage, but more drastic measures are to be deprecated if the risks of a serious surgical procedure are not to be increased during early convalescence.

Anæmia, with a 40 per cent. hæmoglobin index, is a contraindication to general anæsthesia. If operation is essential and a general anæsthetic cannot be avoided, a preliminary blood transfusion should be performed and open ether used. With anæmia of any severity, gas and oxygen should be avoided.

### PRE-OPERATIVE MEDICATION

The preparation of the patient for operation has been described elsewhere (p. 185) and pre-operative medication alone need be considered here.

**Atropine**,  $\frac{1}{100}$  gr., given not less than forty-five minutes before operation, is universally accepted as being desirable; the inhibitory action of the vagus nerve on the heart being thereby diminished, respiration stimulated and mucous secretion lessened. In this connection it is well to emphasise that atropine takes forty-five minutes to reach its full effect, and an increased dose does not hasten this

effect but serves only to prolong it and add to the after discomfort of the patient.

### PRELIMINARY NARCOTISATION

This question must be considered from the point of view both of the patient, the anæsthetist and the surgeon. The effect of the narcotics commonly employed is to diminish or abolish apprehension, to prolong the period of post-operative analgesia and reduce post-anæsthetic discomfort. They can assist the anæsthetist by diminishing the amount of anæsthetic which he will need to use and sometimes enable him to employ gas and oxygen, which alone would be insufficient ; while an operation which would be dangerous with a general anæsthetic may be made possible with the use of analgesia alone. This addition to the armamentarium of the anæsthetist is not without its risks and should only be adopted in a particular case after careful discrimination, as the drugs in general use are cardiorespiratory depressants, and their elimination throws an extra strain on the liver and kidneys. They should be avoided in the presence of asthenia, pulmonary disease and impairment of hepatic or renal function, as once they are introduced into the circulation it is difficult, if not impossible, rapidly to counteract their action in the face of emergency. Build, weight, age and habits must be taken into consideration when estimating the dose for each individual patient, and it is unwise to increase this dose if it appears to have failed in its action, because fear and nervous tension may delay its full effect until the addition of the anæsthetic proper. Idiosyncrasy for these drugs is met with and is characterised by extreme cardio-respiratory depression, which will not respond to stimulation ; this latter fact distinguishes the condition from the effect of an overdose in which, unless very big, there is generally some response. (Oramine appears to be the most efficient drug to use in these cases.

In general it may be said that a dose of a narcotic insufficient to produce a hypnotic effect tends to arouse irritability and excitement.

While these remarks apply in some measure to all the pre-operative narcotics, special mention must be made of certain members of the group in common use :

**Morphine** is never administered alone but always in conjunction with atropine or scopolamine. Morphine,  $\frac{1}{8}$  or  $\frac{1}{4}$  gr., with atropine,  $\frac{1}{100}$  gr., is the usual quantity ordered, while the Roche ampoule containing omnopon,  $\frac{1}{4}$  gr., and scopolamine,  $\frac{1}{100}$  gr., is the popular prescription when the amnesic action of the latter is desired. It must be borne in mind that the addition of anæsthesia is likely to cause respiratory depression, and the signs of anæsthesia are markedly affected especially in regard to the size of the pupil, which remains small, and the corneal reflex which is lost generally in the early stages.

**Paraldehyde and Avertin** may be considered together as they both are administered per rectum and their dosage based on body-weight. Paraldehyde is given in the dosage of 1 dr. per stone of body-weight and diluted to ten times its volume with normal saline. The maximum quantity permissible is 8 dr., which should never be exceeded. This drug is less depressing in its effect than avertin.



but it is used almost entirely for children probably because its permeating smell is very objectionable to adults. The dosage of avertin per kilogram of body-weight is shown on a chart supplied with the drug, together with instructions for making the mixture, and requires no further comment here.

It need hardly be said that with both these drugs a purely weight-based dosage is unreliable; the patient's build, physical vigour and temperament need to be brought into the calculation, and fat should not be counted as body-weight in an obese person.

As to technique, the bowel should be washed out within twelve hours of the time of administration. The injection should be started one hour before the time of operation with the patient lying comfortably on the left side. A small rubber catheter, to which is attached a tube and funnel, is inserted into the rectum as far as it will go and one half of the dose decided upon is slowly run in, when the catheter is clamped and left *in situ*. At the end of fifteen minutes any effect that the drug may have had is observed and another quarter dose administered. Fifteen minutes later the patient is again observed, and if there is drowsiness and incoherence no further dose should be given until a further fifteen minutes has elapsed, when a final quarter dose may be administered if necessary. This technique, though tedious and exacting, is worthwhile as it assures that the minimum requisite dose is given, and a marked effect from the first dose should give rise to suspicion that an idiosyncrasy exists. The catheter is left *in situ* from the commencement of the administration until the operation is finished, when it should be unclamped and the bowel washed out. Gas and oxygen or ether may be used as required to produce anaesthesia, but chloroform should be avoided.

The barbiturates in common use are nembutal, evipan sodium and pentothal.

**Nembutal** given intravenously is so dangerous that it has been given up. It is, however, frequently given by mouth to produce narcosis in children, the maximum quantity usually given being 3 gr. one hour before operation. A rough guide to dosage is  $\frac{1}{4}$  gr. per year of age, though physique must be taken into consideration in every case. It must be remembered that this drug is a liver poison, and large doses have been known to result in suppression of urine.

**Evipan Sodium** is a very powerful narcotic with a rapid and profound action which is generally of short duration. These properties enable it to be used for short operations in which muscular relaxation is not required, nitrous oxide would be insufficient, and the disturbance of a more powerful general anaesthetic unwarranted. It must be emphasised that the contraindications to its use are the same as those for narcotics in general, and that elderly people frequently take from twenty-four to forty-eight hours to recover completely from its effects.

Evipan is given intravenously in a 10 per cent. solution, of which 11 c.c. is the maximum safe dose, which should not be exceeded even if it appears to have missed its effect. The patient should be recumbent and an assistant at hand who can prevent the airway becoming



obstructed as consciousness is lost. The injection is made at the rate of about 1 c.c. in fifteen seconds, and the patient is kept engaged in conversation; the amount used when he can no longer talk is noted and the injection stopped for a period of thirty seconds, when, if anæsthesia has not supervened, a further similar quantity may be injected, and this should be considered the safe full dose. While the dose may thus be estimated with some accuracy, it is impossible to estimate the length of time that anæsthesia will last, for which reason gas and oxygen or ether should always be at hand for use if necessary. Furthermore, it must be remembered that, although the patient appears to be perfectly normal after recovery, he must not be considered responsible for his actions for some hours. Evipan given until consciousness is lost may be used as a preliminary to inhalation anæsthesia with gas and oxygen or ether.

**Pentothal** is similar in action to evipan. It is, however, a more powerful drug, and anæsthesia generally supervenes when the patient ceases to talk. The respiration becomes very depressed, and muscular relaxation is usually obtained for a short period. Recovery of consciousness is very rapid and follows quickly on the resumption of normal respiration. Extravenous administration is liable to cause destruction of tissue, and should be treated at once with hot fomentations.

It may be said of both evipan and pentothal that their continuous administration and the use of preliminary hypnotics to prolong their action should be considered as dangerous and belonging to the province of the expert anæsthetist.

The symptoms of an overdose of any of these narcotics are those of paralysis of the medulla, and should be treated by warmth, artificial respiration and the judicious use of 5 per cent. CO<sub>2</sub> and oxygen accompanied by the intravenous injection of 5 c.c. of coramine. If these measures fail, the withdrawal of cerebrospinal fluid through a cisternal puncture may be efficacious. It may be noted that pentothal sometimes causes severe coughing or sneezing and this fact should be taken into consideration when the choice of anæsthetic is being made.

### GENERAL CONSIDERATIONS IN THE ADMINISTRATION OF AN ANÆSTHETIC

The anæsthetist's success in gaining his patient's confidence is put to the test when the time for inducing anæsthesia arrives. He may indeed congratulate himself if the patient afterwards should have no memory of discomfort nor any disagreeable recollection of the passage into unconsciousness. Silence should be secured and the light should be shaded but still sufficient. The patient lies in a comfortable and relaxed position, protected as far as possible from external stimulus. He must not be allowed to grip anything except his own hands, and should be touched as little as may be. When a gas mask is used it must be applied with the lightest pressure, while the open mask can be held away from the face or simply rested on the

face-pad. All sense of restraint should be avoided so that the conditions of anæsthesia may resemble those of natural sleep. The patient should be told to breathe as if he were quietly awaiting sleep in his own bed, and sleep should be the theme of the anæsthetist's remarks. In this way the action of suggestion is used to reinforce the effects of the anæsthetic drug.

Provided that the anæsthetist has obtained and knows how to hold his patient's confidence, precautions such as these will almost always lead to a smooth induction with complete absence of excitement and struggling. It should be remembered that the sense of hearing is the last to be abolished and the first to return during recovery.

No anæsthetic should be administered except in the presence of a third person, inattention to this rule having led to serious damage being done by a violent and unconscious patient during the second stage of anæsthesia. Erotic dreams are not uncommon, especially during the administration of gas in the removal of teeth, and these have been sufficiently vivid to lead to legal action for assault against the administrator.

Anæsthesia having been established, the patient's head should be turned to one or other side to prevent any secretion that may collect in the mouth from flooding the larynx, the jaw being gently supported and an ear kept exposed. The lobe of the ear, having a large capillary blood supply, blanches when momentarily squeezed, the return to normal colour being almost instantaneous. Under the stress of operation the time taken for the colour to return becomes longer, and constitutes a very early indication of circulatory embarrassment. The ear, it may be said, is second only to the lips as an index of the oxygenation of the blood.

In this connection it may be remarked that bright arterial blood in the wound with cyanosis of the scalp, ear and lips denotes poor capillary circulation and myocardial inefficiency.

### THE SIGNS OF ANÆSTHESIA

Anæsthesia is divided into four stages for convenience of description, each of which has its own definite signs, though the passage from one stage to another is not clearly distinct.

**The First Stage** extends from the commencement of induction to loss of consciousness. In this stage :

1. The respiratory and cardiac centres are stimulated.
2. Superficial and deep reflexes are present.
3. The pupil of the eye gradually dilates as consciousness is lost.

During this stage any movement made by the patient is voluntary and may be purposeful. Forcible restraint should never be attempted, for it is disastrous to a quiet induction and may lead to complete loss of the patient's self-control ; the simple request to lie quiet is all that is needed. As loss of consciousness is approached, the hearing is

amplified and appears to be lost only when unconsciousness is reached. When unconsciousness is complete the respiration becomes audible and assumes the type in which inspiration is through the nose and expiration is oral.

**The Second Stage** extends from the loss of consciousness to complete anæsthesia. During this phase :

1. The respiration may be irregular and breath may be held.
2. The pulse is rapid, becoming slower as anæsthesia progresses.
3. The reflexes become more and more sluggish until eventually they are abolished, that of the cornea being the last to go.
4. The pupil of the eye gradually contracts but remains active to light, and the eyeball is frequently seen to be moving.

In this stage involuntary and purposeless movements may be made which require restraint and general excitement may be prominent. The breath may be held and the musculature of the whole body go into spasm, but this is rarely the case if the first stage has been properly conducted. Should it occur, however, the mask should be removed from the face, little else remaining to be done until the  $\text{CO}_2$  in the blood is sufficiently increased for the spasm to relax. To attempt to open the jaw is liable to lead to injury and is futile, since the muscles of respiration share the general tonic spasm. As soon as spasm ceases oxygen should be administered, and it is inadvisable to exhibit the anæsthetic until respiration has returned to normal.

The corneal reflex is obtained by gently lifting the upper lid of the eye with one finger and very lightly brushing the corneal conjunctiva with another finger of the same hand. It is not generally understood that the conjunctiva covering the cornea is much more delicate and sensitive than that covering the sclerotic coat of the eye, and ignorance of this fact is liable to lead to mistakes in judging the stage of anæsthesia reached. Too frequent stimulation of the cornea is liable to render it insensitive.

**The Third Stage**, that of surgical anæsthesia, is characterised by regular, rhythmic respiration :

1. The pulse approaches more nearly to the normal rate.
2. All reflexes are completely abolished.
3. The pupil is dilated and may show either a sluggish response to light or none at all.
4. The eyeball is fixed and the eye is often open.
5. The conjunctiva is wet.

When once this stage has been reached the anæsthetist should concentrate his attention on the respiratory rhythm, every patient in the condition of surgical anæsthesia settling down to a definite rhythm which is individual and seldom the same in any two cases. As the operation proceeds, any alteration in this respiratory rhythm must be noted and its cause ascertained at once ; if this were always done many of the alarms of anæsthesia would be avoided.

ALTERATION IN RESPIRATORY RHYTHM may be caused by :

1. *Obstruction of the Air-way*.—The jaw muscles being relaxed, the jaw tends to fall back and with it the base of the tongue. This impinges on the posterior pharyngeal wall, and some degree of obstruction is occasioned. It may be overcome by holding the jaw forward, so that the lower teeth are in front of and overlapping the upper. Should this fail a Hewitt's air-way (Fig. 69) must be inserted into the mouth, care being taken that the tongue is not rolled back, and that the end of the tube is between the base of the tongue and the pharyngeal wall. Obstruction may also be caused by an accumulation of mucus or saliva in the pharynx ; an attempt to relieve it by lowering

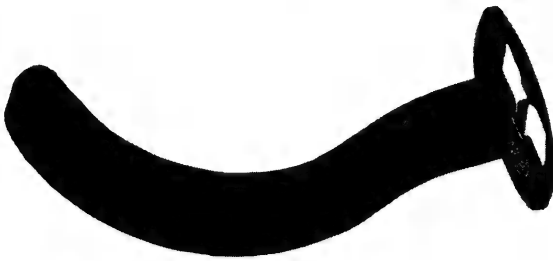


FIG. 69

An airway. (Charles King, Ltd.)

the head should first be made, but if it fails the mouth should be opened, the tongue drawn forward by a clip and the pharynx sponged out with great care and gentleness. It should be noted that the edentulous often obstruct the air-way by sucking in their cheeks, in which case a simple gag large enough to keep the lips apart is generally all that is needed. It sometimes happens that inspiration may become suddenly and completely obstructed, although respiratory movements are in evidence. This emergency, generally caused by a plug of mucus completely occluding the larynx, is characterised by the suddenness and quietness with which it occurs. Lowering the head and squeezing the chest at the end of an inspiratory movement may dislodge the plug, but if not, the larynx should be cleared by direct laryngoscopy or, as a final measure, tracheotomy may be performed.

2. *Apnœa*.—This is caused by over-oxygenation of the blood and a reduction of its  $\text{CO}_2$  content, which entails the absence of the normal stimulation to the respiratory centre. Apnœa is therefore most frequent after excessive respiratory effort, which may be partly due to the anæsthetic but is more often due to the deep breathing encouraged by the inexperienced anæsthetist. It is most likely to occur early in the third stage but is rarely observed when a closed method of administration is used. The signs of this condition are hardly mistakeable ; the patient shows a bright red colour, and though the air-way is unobstructed respiration becomes more and more shallow until it appears to cease. If the onset of these signs be recognised soon enough, the administration of additional oxygen will frequently stop them ; but if this fails, the administration of  $\text{CO}_2$  with the oxygen will rapidly cause a return to normal respiration. The condition tends to right itself as the tension of  $\text{CO}_2$  in the blood rises, but it is unwise to wait for this since considerable strain is put on the heart during the interval. Rhythmic squeezing of the chest will generally

start respiration quite rapidly. If cardiac inefficiency is suspected CO<sub>2</sub> and oxygen should be exhibited with the greatest care lest the sudden stimulation cause heart-failure. Anæsthesia will have considerably lightened when normal conditions have returned, so that it may be necessary to stop the operation until a suitable degree of anæsthesia is re-established.

3. The respiratory rhythm will be altered if anæsthesia becomes *too light*; this will be shown by the return of the corneal reflex, activity of the pupil in response to light and possibly by movement of the eyeball.

4. Alteration will occur if anæsthesia becomes *too deep*; in which case absence of the corneal reflex, a dilated and fixed pupil, a fixed eyeball, the conjunctiva either dry or drying, and failing respiration and pulse are the danger signals.

Manipulations by the surgeon such as stretching the anal sphincter, traction on an abdominal viscus, or the delivery of a tumour, etc., almost always affect the respiratory rhythm.

**The Fourth Stage** is caused by an overdose of anæsthetic, and its symptoms are associated with paralysis of the medulla. Pulse and respiration fail, the eye is fixed and insensitive to light and touch, the conjunctiva is dry and the intra-ocular tension decreased. The patient is pale and cold and beads of sweat appear on the nose and forehead.

*The Treatment* of this serious condition consists in rapidly eliminating the anæsthetic from the body and taking vigorous steps to keep the vital centres active. Operation and anæsthetic should at once be stopped and the patient put into a modified Trendelenburg position. Oxygen should be administered and artificial respiration by Sylvester's method begun, while hot packs are applied to the precordium. If the emergency is recognised early enough, these measures generally suffice to restore respiration and circulation; but if not, an injection of adrenalin and strychnine into the substance of the heart may cause sufficient response to overcome the lethal action of the anæsthetic. Cardiac massage may be undertaken without delay if the abdomen is already open, but if not the epigastrium should be rapidly opened as the last resort. This fourth stage of anæsthesia must be distinguished from the condition which is called surgical shock. The nature and duration of the operation, the quantity of anæsthetic administered, the ocular signs which indicate that anæsthesia is not unduly deep, and the wet conjunctiva are the main points of distinction.

The above description of the signs of anæsthesia applies when atropine alone has been administered before the operation. When an hypnotic has also been given its general effect is to decrease the activity of the reflexes and to cause the pupil of the eye to remain smaller.

It should be remembered that the stage of anæsthesia is determined by the consideration of a series of signs, of which no single one is by itself reliable; thus, the corneal reflex is peculiarly undependable in young children, while in the aged it is unwise to be guided by the size and activity of the pupil.

### THE CHOICE OF ANÆSTHETIC AS INDICATED BY THE SITE OF OPERATION

Although the general principles in the choice of the anæsthetic apply to every part of the body, certain special considerations are likely to arise in certain operations.

**Operations on the Brain** are best performed under avertin and local analgesia, which allow all the time needed for these lengthy procedures without any of the disadvantages of a general inhalation anæsthesia.

The cerebellum, on the other hand, should always be approached under intratracheal anæsthesia. The patient must be supported by sand-bags under each shoulder and the pelvis, so that the movements of the chest are not impeded. Gas and oxygen is the method of choice reinforced with ether if necessary.

**Operations on the Eye** are usually performed under cocaine instillation, but if a general anæsthetic is required chloroform is ideal in achieving a motionless eyeball, a minimum of hæmorrhage and an absence of post-anæsthetic vomiting.

Pentothal or evipan may be used with advantage, but a preliminary injection of omnopon and scopolamine should always be given to reduce to a minimum the risk of coughing or sneezing during the operation.

**Operations involving the Mouth and Upper Air Passages.**—The surgeon demands a clear field and the prevention of the aspiration of blood, mucus or a foreign body into the trachea.

In clean cases an endotracheal tube with packing off of the pharynx with soft gauze will meet these requirements. In the presence of sepsis or malignant disease a laryngotomy performed after anæsthesia is established has a strong claim to being the method of choice, as the risk of carrying morbid matter into the respiratory system by the passage of a tube through the affected area is eliminated; the pharynx can be packed off with absolute certainty, and anæsthesia maintained with equal ease and safety. The laryngotomy tube may be removed directly the pharyngeal and laryngeal reflexes are active.

**Operations on the Thyroid Gland** are liable to give anxiety because of thyrotoxicosis and possible myocardial degeneration. If there is definite tracheal obstruction the anæsthetist should be prepared to pass an intratracheal tube at any time. The toxic patients are best dealt with by preliminary narcosis and by blocking the cervical plexus and the line of the skin incision with novocain. Avertin or omnopon and scopolamine are useful preliminaries.

**Prostatectomy** is best performed under a low spinal analgesia, though many patients do very well with a simple ether anæsthesia.

**Abdominal Operations** may be performed under any method of anæsthesia, the choice usually being dictated by the skill of the anæsthetist available. Spinal analgesia for abdominal operations often implies that the standard of anæsthetic administration is not high.

**Intestinal Obstruction** requires special consideration. There is no condition in which the expert anæsthetist can be of such great assistance to the surgeon. Vomiting is liable to occur at any stage, vast quantities of fluid being regurgitated without any movement on the part of the patient (see p. 633). A stomach tube should be passed as soon as the stage of unconsciousness is reached and the head kept low. Spinal analgesia is of great service in acute obstruction unless the diagnosis has been delayed and the patient is very toxic, when a local infiltration of the abdominal wall and gas and oxygen will suffice.

### ETHER

Ether is administered by the following methods : (1) Inhalation—open or closed ; and (2) Insufflation.

**The Inhalation Method.**—Ether vapour has a most unpleasant smell and is very irritating to the respiratory tract ; moreover, when it is used for induction the process is tedious both to patient and anæsthetist. It is therefore seldom administered without the employment of a preliminary narcotic or without the prior use of a less objectionable anæsthetic.

In the *open method* the patient's face is covered by gamgee tissue with an aperture for mouth and nostrils and a Schimmelbusch mask (Fig. 70), covered with six or eight layers of gauze, is allowed to rest on the face-pad. Ether is dropped slowly and regularly upon the centre

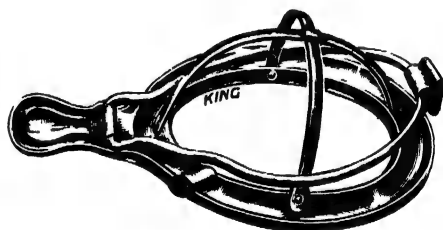


FIG. 70

Schimmelbusch mask. (Charles King, Ltd.)

of the mask until unconsciousness is reached. Thereafter speed can be increased and the drops distributed all over the mask.

The anæsthetics which are most commonly used before open ether are ethyl chloride or a mixture of chloroform and ether. If the former is employed, surgical anæsthesia should be complete before the ether is rapidly and lavishly exhibited ; otherwise it sometimes happens that laryngeal spasm develops when the ether follows. In this case consciousness returns before ether anæsthesia is established.

When a mixture of chloroform and ether is used for induction, anæsthesia should be carried only to the beginning of the second stage before transferring to ether alone.

With the *closed method* a Clover's inhaler (Fig. 71) is used, preliminary unconsciousness being obtained by gas and oxygen, or by a mixture of chloroform and ether. Gas can be administered with the ether container in position, and this is a safe and pleasant method, though considerable practice is required before smooth anæsthesia can be



regularly secured. The mistake most frequently made is that of increasing the strength of the ether vapour too rapidly, which

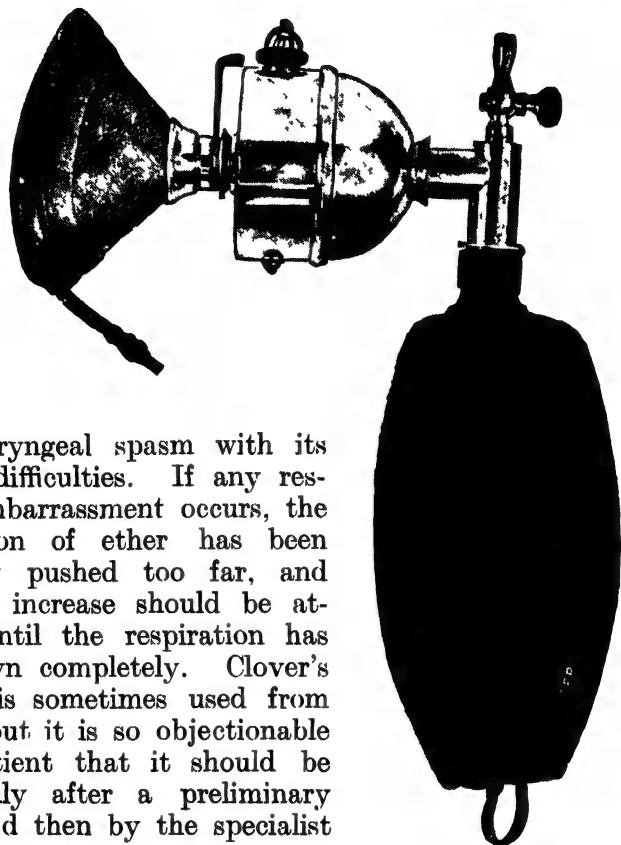


FIG. 71  
Clover's ether  
apparatus.  
(Charles King,  
Ltd.)

initiates laryngeal spasm with its attendant difficulties. If any respiratory embarrassment occurs, the concentration of ether has been temporarily pushed too far, and no further increase should be attempted until the respiration has settled down completely. Clover's apparatus is sometimes used from the start, but it is so objectionable to the patient that it should be applied only after a preliminary narcotic and then by the specialist anaesthetist.

**The Insufflation Method**, by which is meant the administration of the anaesthetic through a tube passed into the trachea, obviously overcomes one of the difficulties of anaesthesia, the maintenance of a clear air-way. It involves instrumentation of the throat, and therefore causes some trauma which is not always negligible. Throat sensitiveness varies greatly, and it is by no means uncommon for soreness of this passage to contribute a great deal to any post-operative discomfort which may be present. When once the tube is in the trachea, any anaesthetic can be administered, either on a mask or under pressure. A number of apparatuses for pressure administration are on the market; all of them contain a safety-valve, which may be regulated to blow off at all pressures above the maximum of 30 mm. Hg. The method is specially useful in operations on the upper air passages, where there is a danger that the mouth and pharynx may be flooded with blood.

### CHLOROFORM

Chloroform is universally recognised as the most dangerous of the anaesthetics; a reputation which is attributable both to its toxicity and to its dangers when administered by the inexperienced.



It remains, however, the anæsthetic of choice in certain conditions :

1. In some forms of respiratory disease ; emphysema, chronic bronchitis, etc.
2. In hyperpiesis and arterial degeneration.
3. When the cautery is used and when diathermy is applied in the region of the air-passages.
4. In cases in which post-anæsthetic vomiting or hæmorrhage would prove serious, as notably in some operations on the eye.

Chloroform should be avoided :

- (a) In septic conditions ; always in acute sepsis and in chronic sepsis when there is marked toxæmia.
- (b) In diabetes mellitus and glycosuria.
- (c) In liver insufficiency.
- (d) In chronic alcoholism, especially when this occurs in persons of the plethoric type.
- (e) In children.

**The Preparation of the Patient** for chloroform anæsthesia does not differ from that described elsewhere except that no previous narcotic should be given. Atropine, however, should invariably be employed, as it diminishes the irritability of the vagus which seems to be singularly susceptible to chloroform.

Chloroform must never be administered by a closed method but on an open mask. The following technique, when carefully carried out, has proved successful.

The face should be covered by a gamgee pad with the exception of the nose and mouth, and a Schimmelbusch mask covered with three or four layers of gauze should be held a finger's-breadth away to admit plenty of air. The anæsthetic should be allowed to drip slowly and regularly on the centre of the mask, but not over its whole surface. When unconsciousness has been reached, the mask may be placed on the pad resting on an oxygen tube, which is always kept in operation. Steady administration of the chloroform is continued until surgical anæsthesia is reached, when just sufficient drops should be put on the mask from time to time to maintain the anæsthesia required. Oxygen should be given throughout because the slightest degree of cyanosis is liable to be followed very rapidly by signs of respiratory and cardiac failure. No surgical procedure should be attempted under chloroform until full surgical anæsthesia has been reached, as many fatalities have occurred when minor operations have been attempted under light anæsthesia.

**The Signs of Anæsthesia** call for no special mention except that the pupil remains somewhat small. A pupil 2 mm. in diameter with an absent corneal reflex would indicate a depth of anæsthesia which would be signified by an aperture of 4 mm. or even 5 mm. if ether were being used. When once surgical anæsthesia has been induced, unremitting attention must be paid to the respiratory rhythm and the size of the pupil, and the cause of any alteration in either must be ascertained at once.

The stage of excitement is the most dangerous period of the induction, as struggling or holding the breath is liable to be followed by one long deep breath. When these symptoms occur it is advisable to remove the mask until regular respiration is re-established, for chloroform is so rapidly absorbed that it is quite possible to inhale a lethal dose in one deep inspiration.

**Post-operative Complications.**—1. Vomiting is less frequent than after other anæsthetics, with the exception of gas and oxygen, but varies considerably, and if severe, is generally more prolonged than that following ether.

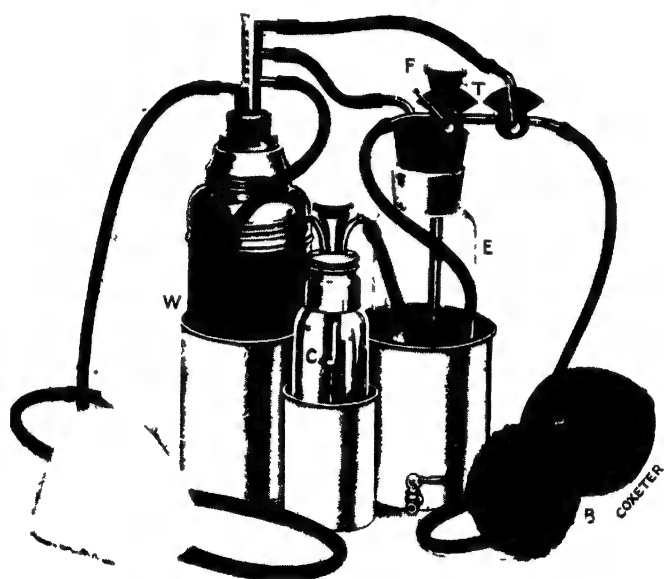


FIG. 72

Shipway's warmed ether and chloroform apparatus  
(Charles King, Ltd.)

2. Pulmonary complications appear to be less frequent than after ether, but, when they do occur, are liable to be more severe. This may in some measure be accounted for by the resistance of the patient being lowered by the more toxic effect of chloroform.

3. Delayed chloroform poisoning is rare; it presents the picture of acute acidosis and should be treated as such. It occurs generally about seventy-two hours after operation as a sequel to severe vomiting; and is characterised by restlessness lapsing into coma, the urine being loaded with acetone and diacetic acid. The pre-operative and post-operative administration of glucose has played a considerable part in rendering this a rare condition.

Chloroform is sometimes administered by means of a Junker's inhaler or a Shipway's warm vapour apparatus (Fig. 72), the principle of which is that air or oxygen is blown through chloroform and the vapour is led to the patient through a tube. The vapour may be delivered under a mask or by a tube placed in the nose or mouth.

### ETHYL CHLORIDE

The administration of ethyl chloride is beset with danger unless its rapidity of action is fully realised, for surgical anæsthesia can be obtained in as little as forty-five seconds, and further application will rapidly lead to over-dosage. This action however, is compensated by rapid elimination and the return of consciousness unless the dangerous stage has progressed too far.

It is of great value as a preliminary to ether narcosis, especially in children, but should not be followed up by chloroform; it causes a rapid fall of blood pressure and chloroform is liable to produce a further fall which may cause anxiety.

Ethyl chloride may be administered by the open or closed method.

1. **The Open Method** is perhaps the safer and is the more satisfactory if ether is being used later. A gamgee pad covers the face with the exception of the nose and mouth, and a small mouth prop may be placed between the teeth; a Schimmelbusch mask with six or eight layers of gauze is lightly held on the face pad. The patient is instructed to breathe quietly, and ethyl chloride is sprayed gently over the whole mask; after a few breaths respiration becomes deep, and this generally heralds loss of consciousness and the commencement of excitement. During this stage the teeth may sometimes be clenched and the breath held, but is uncommon if the strength of vapour has not been increased too quickly, though complete absence of excitement is rare. The respiration becomes regular, the muscles relax, the corneal reflex is generally absent, and the pupil dilates. After this the mask should be removed, and the operation quickly started. Relaxation for a varying period up to, and occasionally exceeding, forty-five seconds may be expected, and in a favourable case unconsciousness may last as long as three minutes.

When ether is to be used subsequently it should be administered with a lavish hand directly regular respiration has been produced by ethyl chloride; but not so quickly that laryngeal spasm is set up, for consciousness is then likely to be regained before ether anæsthesia has been established.

2. **The Closed Method.**—A measured quantity of ethyl chloride is introduced into a rubber bag, in and out of which the patient respires until anæsthesia is produced. One of the best ways of employing the method is by means of a Loosely's inhaler, the principle of which is that a valve can be regulated so that air is inspired and expired into the bag; by shifting the valve to and fro breathing into the bag is secured. An ordinary gas mask can be fitted to the apparatus, but care should be taken that it is suitable for the face and that no leakage takes place round its edges.

Opinion differs as to the dosage that should be used with this method; and certainly experience allows a broader margin than would be safe without it. The open method is probably the better under five years of age; from five to fourteen years of age 4 to 5 c.c. appears to be a good average dose, though this depends to some extent on the build and physique of the child. From fourteen to twenty

years 7 c.c. may be given, while for adults 10 c.c. is usual. However, to realise its rapidity of action, to recognise the signs of anæsthesia and to withdraw the drug when these are present is perhaps the best insurance against accident, so that the quantity originally used is, within reason, of secondary importance.

### ADMINISTRATION OF NITROUS OXIDE GAS

Nitrous oxide has long been recognised as the safest anæsthetic for short operations in which loss of reflexes is not required. In these cases it is usually given with an admixture of air, as the addition of oxygen appears to be more likely to cause a feeling of faintness which is sometimes delayed as long as thirty minutes before becoming apparent. The aim of the administration should be to avoid extreme cyanosis and to get as great a concentration of nitrous oxide in the blood as possible. This object may be attained in the following way: The gas bag is partially filled and the mask placed over the patient's nose and mouth with the valves open to the air. Quiet regular breathing should be insisted on and no gas administered until this has been obtained. The gas is then admitted during expiration, and the patient is allowed to take one breath of gas and one breath of air, then two breaths of gas and one breath of air, until six breaths of gas and one breath of air is being given. This proportion should be maintained until the respiration becomes deep, and then pure nitrous oxide should be given and the mask removed at the first sign of stertor. In this way anæsthesia lasting for thirty to forty-five seconds may be obtained and is used for short dental extractions. Gas and air may be administered continuously for some minutes either through a nasal mask, if required for dentistry, or by means of the ordinary face mask for operations elsewhere. The technique described may be used, but stertorous breathing must be avoided by regulating the amount of air admitted. The danger of administering gas and air in the presence of a full stomach cannot be over-emphasised, and it is best avoided in cases of myocardial degeneration.

### NITROUS OXIDE AND OXYGEN IN MAJOR SURGERY

Nitrous oxide is used for prolonged operations with an admixture of oxygen instead of air. Its low toxicity and absence of effect on blood pressure are very desirable, but when used by itself it produces amnesia rather than abolition of pain. At any rate it does not abolish reflex response to the most painful stimuli, and therefore it needs to be supplemented with narcotics or some form of analgesic to keep the patient reasonably quiet. Operations on the limbs, for instance, can be performed with little difficulty, but this is not the case when abdominal section is concerned.

Relaxation of the abdominal wall can rarely be obtained with gas and oxygen alone. Spinal analgesia will relax the abdominal muscles and is probably better without the addition of gas and oxygen if the patient is of suitable mentality, but it cannot always be used with

safety. Paravertebral block analgesia is attended by fewer complications but is not so efficient and, unfortunately, fails in a considerable percentage of cases because of the difficulty of hitting off every nerve; for if one or two are missed it is sufficient to render the analgesia almost useless. But if neither of these techniques is adopted, preliminary narcosis is almost essential, and local analgesia indispensable. In this case manipulation is hampered and operation prolonged in all but the simplest cases.

In fact, its well-earned reputation in short administrations does not apply when its use is prolonged, for collapse, both during and after operation, is by no means unknown, while severe fatigue is a common sequel. Gas and oxygen is inclined to increase hæmorrhage, and should not be used without consideration in operations where heavy loss of blood must be avoided.

The modern gas and oxygen apparatus is so contrived that the gases can be laden with ether or chloroform vapour or with both (Figs. 73 and 74), while  $\text{CO}_2$  can be administered if it is found necessary to overcome any respiratory depression caused by pre-medication. Thus in the event of difficulties the method can be altered and any mixture may be given by the simple process of turning taps. It might be invidious to speculate how often these drugs, quite unostentatiously administered, have sustained the credit of gas and oxygen.

**The Preparation of the Patient** does not differ in any respect from that described elsewhere. It is the rule to use narcotics in the preliminary medication, atropine, scopolamine and morphia being probably the most popular, though avertin has to some extent replaced them. Atropine is considered by some to cause restlessness during anæsthesia, but if it is omitted and reliance placed on scopolamine and morphia,  $\text{CO}_2$  should always be kept at hand to stimulate respiration, should it become unduly depressed. The same precaution should be taken when the basal narcotics are exhibited.

It will be gathered that gas and oxygen can be used for almost any operation to which the human body is subjected, but only at the

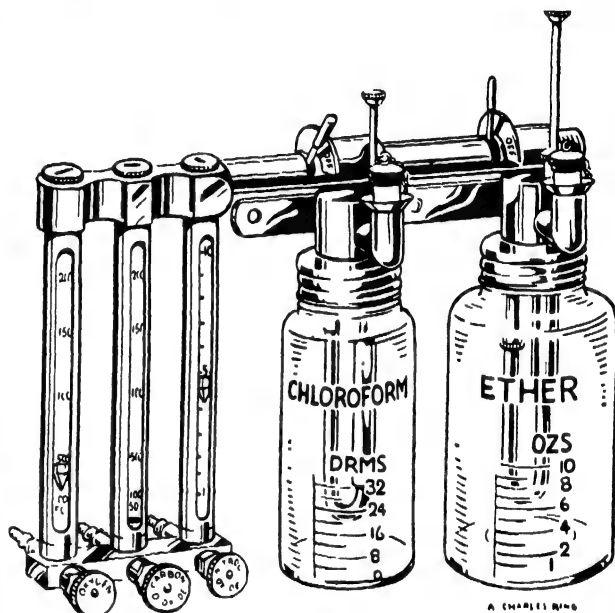


FIG. 73

Flow-meter for controlling the quantities of nitrous oxide, oxygen and carbon dioxide, together with chloroform and ether bottles. (*Charles King, Ltd.*)

expense of time and with the necessary conjunction of drugs which are difficult to control. It should be employed with the greatest precaution, if at all, in hyperpiesis, anæmia, alcoholism, respiratory

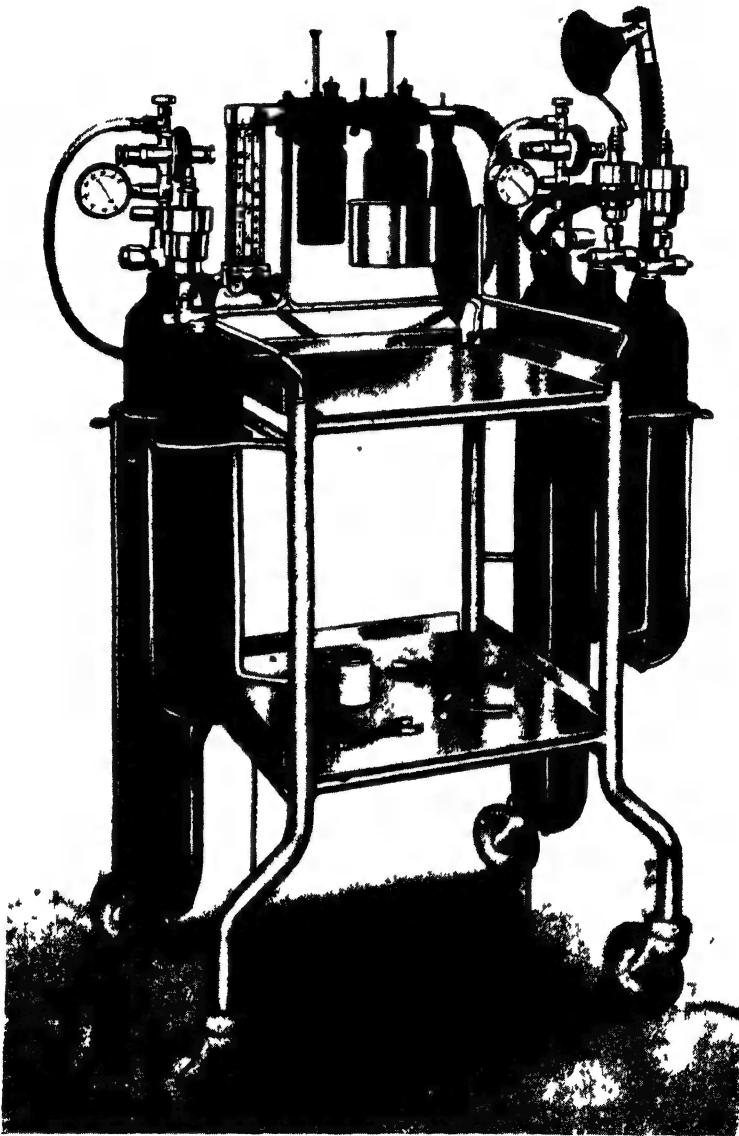


FIG. 74

A modern anæsthetic trolley complete with cylinders, flow-meter, anæsthetic bottles, bag and face piece. (*Charles King, Ltd.*)

embarrassment, cardiac diseases where a slight degree of anoxæmia would be dangerous, and in such conditions as cellulitis of the neck, wherein engorgement of the tissues threatens respiratory obstruction.

**The Sign of Anæsthesia** is regular respiration. A moving eyeball

is common, but the conjunctival reflex is generally absent. Other reflexes being seldom absent though they may be diminished.

**The Administration of Gas and Oxygen** is difficult and requires considerable experience before success is attained. Perhaps the most important point is that the patient should be saturated with nitrous oxide before the operation commences. This takes time, and five, or even ten, minutes spent in achieving it are not wasted. The following technique is generally successful; first, the bag is filled with oxygen and the patient allowed to take several breaths before the nitrous oxide is gradually introduced. The oxygen is then turned off and the nitrous oxide permitted to flow gently into the bag until the patient is slightly cyanosed, after which a small proportion of oxygen, just enough to remove cyanosis, is allowed to mix with the gas. Re-breathing takes place during the whole of this procedure, the mistake most frequently made being to admit too much oxygen after the cyanotic stage has been reached. The modern apparatus is equipped with sight-feeds for the gases, by means of which the proportions administered are seen. When anæsthesia has been established the gas-bag valves may be brought into operation and the gases given in the proportions found most suitable. This method is wasteful of gas and appears to have no great advantage over re-breathing, provided the precaution of emptying the bag from time to time to get rid of the accumulation of expired  $\text{CO}_2$  is taken.

**Post-operative Complications.**—The prolonged administration of gas and oxygen is followed by all the post-operative complications common to the other anæsthetics, but there is little doubt that if discretion be used in selecting cases the risk of their occurrence is reduced to a minimum. Serious vomiting, for instance, is rare. It is difficult to compare the frequency of chest complications; all that can be said is that these do occur, but so many potential causative factors are involved in all post-operative respiratory troubles that it is difficult to decide how far the anæsthetic is responsible.

**Cyclopropane.**— $\text{C}_3\text{H}_6$  has come into prominence as an anæsthetic agent during the last decade and is a very valuable addition to the armamentarium of the specialist anæsthetist. This gas, which is administered with oxygen with a soda lime  $\text{CO}_2$  absorber in a closed circuit, is expensive and the apparatus required both costly and complicated as it is essential that the proportions of cyclopropane and oxygen be accurately known.

The action of this anæsthetic is rapid and profound especially on the respiratory system which becomes more and more depressed as anæsthesia deepens; indeed automatic respiration may be deliberately abolished and replaced by respiration controlled by intermittent pressure on the re-breathing bag: the facility with which this manœuvre can be accomplished renders this an ideal anæsthetic for thoracic surgery.

Cyclopropane, however, may be used in any branch of surgery in which complete muscular relaxation is not essential, slightly increased hæmorrhage not a deterring factor and the cautery is not being used, but no attempt should be made to administer this gas without expert



supervision until proficiency in estimating the depth of anæsthesia is acquired.

### CARE OF THE PATIENT AFTER ANÆSTHESIA

A patient under the influence of an anæsthetic must not be left unattended for a moment until consciousness is well re-established, while the invigilation should continue for at least twelve hours if one of the basal hypnotics has been used as an adjuvant. The room should be warm and draughtless, the bed heated ; but all hot-water bottles must be removed and not replaced until the patient is fully conscious. It should be remembered that the heat-regulating centre is always disturbed to some extent by anæsthesia, and failure to recognise this, both at the time of operation and subsequently, contributes to the incidence of chest complications.

On return to bed the unconscious patient should be turned on his side, the knees bent up, and the head comfortably supported on the edge of a pillow so that the mouth is turned towards the bed ; this position is natural and tends to keep the air-way clear by allowing the tongue to fall forward. Any secretions that may collect in the mouth run out without hindrance and the strain of vomiting is reduced. Of course, the type of operation must be taken into consideration, but it is seldom that this sort of position cannot be assumed.

Vomiting is the most immediate and troublesome sequel to anæsthesia. It frequently occurs while the patient is unconscious and, unfortunately, does not always cease when consciousness returns. Four contributory factors are to be considered in post-operative vomiting ; the patient, the anæsthetist, the anæsthetic and the operation.

Lack of rest, starvation and purgation before operation are factors which render the patient more susceptible to post-operative vomiting, while apprehension and lack of confidence in the anæsthetist play their part. Those who suffer from train sickness and sea sickness are generally very sick after anæsthesia.

The anæsthetist's neglect of detail renders vomiting more common ; thus the too rapid concentration of vapour in the early stages leads to mucus secretion in the mouth, and the swallowing of it laden with vapour may cause gastritis even to the point of hæmatemesis. Inattention to a clear air-way and to slight degrees of cyanosis, the administration of too much anæsthetic and the fault of allowing anæsthesia, once established, to become alternately light and deep all lead to the same end.

Gas and oxygen is least likely to cause vomiting, though, when it does, the condition is generally prolonged and severe ; chloroform and ether vomiting, though more frequent, is less likely to be severe and, in fact, may cease before consciousness returns.

Abdominal operations, whether performed under anæsthesia or analgesia, are frequently followed by vomiting, particularly when there is manipulation of the uterine adnexa, traction on the testicle or manual examination of the abdominal contents. Vomiting of altered blood after operations on the nose and mouth is common, and often lasts until oozing has ceased.



Post-operative vomiting, therefore, depends on many factors, and its incidence is less frequent when these are duly considered. Circumoral pallor and respiratory alteration are the most constant forerunners of vomiting and are followed by retching. Finally, the breath may be held, the jaws clenched and the lips tightly pursed. This latter condition need cause no alarm for often the insertion of the index finger and thumb between the lips and then round the upper jaw, so that the cheeks are kept separated from the teeth, will start respiration and keep the air-way clear. More energetic measures with gags, tongue clips, etc., should only be resorted to in emergency, since the teeth may be injured thereby.

Whatever the cause of continued vomiting may be, treatment consists in first ensuring that there is no anæsthetic-laden mucus collected in the stomach. Half a pint of warm water with a drachm of sodium bicarbonate is introduced into the stomach and is generally returned in the course of a few minutes. Vomiting may then cease, but if not, one minim doses of iodine in a little water may be given hourly. Orange juice, lemon juice, weak tea, sips of champagne, or even wafer bread and butter if it can be taken. Should all these measures fail nothing further should be given, but the stomach should be washed out through a Jutte's tube, which is introduced through the nose and is easier to swallow than the usual stomach tube. This can be left in position without much discomfort to the patient, and his stomach kept completely empty. Failure to control vomiting by this method should suggest the possibility of intestinal obstruction or acidosis. It should be remembered, finally, that post-operative vomiting may become hysterical and thus require psychological rather than physical treatment.

**Post-operative Pulmonary Complications.**—These generally commence in forty-eight to seventy-two hours after operation; the appearance of symptoms earlier than forty-eight hours suggest that infection has been previous to operation. The part played by the anæsthetic in causing lung complications is difficult to assess; they may occur with any general anæsthetic and are specially liable to do so after abdominal operations; this is also true of operations performed under analgesia, though the incidence of complications is generally considered to be less frequent. The maintenance of an even temperature in the sick room, freedom from draughts, and the adoption of Fowler's position are of inestimable value in warding off lung trouble.

Broncho-pneumonia, massive collapse of the lung, and pulmonary embolism are the conditions most likely to be met. The signs, symptoms and treatment of broncho-pneumonia are too well known to need description here. Massive collapse of the lung, however, and pulmonary embolism need differential diagnosis, as their onset is generally similar, but the former is but a risk to life while the latter is frequently fatal. In massive collapse the heart is always displaced towards the affected side, while there is no displacement with embolism; shock in embolism is generally much more pronounced than in massive collapse; the sputum in the latter is greenish and never blood-stained, while, if the patient survives, the sputum of

pulmonary embolism generally becomes blood-stained very soon. The treatment of these conditions is not within the province of the anaesthetist.

**Post-operative Conjunctivitis** is generally attributed to the anaesthetic, and all that need be said is that care and delicate handling of the conjunctiva by the anaesthetist when he elicits the corneal reflex and attention to keeping the eye closed during the whole of the operation prevent the condition occurring.

### ANALGESIA

The production of analgesia is so vast a subject that a detailed description of the methods used is outside the scope of this article ; indeed, no more than a general consideration can be attempted.

The first essential is an accurate knowledge of the anatomy of the nervous system, especially of the distribution of the sensory nerves, without which the success to be expected in the more elaborate undertakings is small.

Nothing short of complete absence of pain is to be sought, for partial success is likely to be worse than failure owing to the lapse of confidence it causes. The promise that all sensation will be abolished should never be made, since often the sense of touch or pressure is retained and may be misconstrued as pain.

There are but few operations which cannot be performed under analgesia, but the various methods should not be used without discrimination ; persons of unstable disposition, even in spite of narcotics, may often endure mental anguish that leaves an indelible impression on their minds.

The introduction of an analgesic causes trauma to the tissues, and their resistance at the site of injection is lowered ; therefore the method should not be used in the presence of sepsis or in conditions in which the blood stream is infected. The minimum quantity of the solution should be injected, and the strictest aseptic technique practised ; syringes and needles must not be boiled in water containing bicarbonate of soda, as the minutest quantity of this substance in the analgesic solution is likely to render it ineffective, as does spirit.

A great number of different substances are used for the production of analgesia, each having different characteristics and different degrees of toxicity ; the commonest are synthetically prepared and are derived from cocaine.

The use of *cocaine* is now limited almost entirely to direct application to mucous surfaces—the eye, nose, throat, larynx, etc. Its local action is very powerful, and if absorbed in any quantity into the circulation can cause in the susceptible giddiness, faintness, vomiting, epileptiform convulsions and even coma and death. Such untoward symptoms appear to be due to rapidity of absorption ; the addition of adrenalin 1 : 10,000 to the solution tending to prevent this by causing ischæmia at the point of application. The strongest solution commonly used is 10 per cent., and this only for surface application in nose and throat work ; great care should be taken that none is

swallowed as absorption from the stomach is rapid. A 5 per cent. solution is the usual strength used in operations on the eye and for intratracheal injection, etc. Cocaine should never be used in the urethra, the mucous membrane of which seems to be singularly permeable. The treatment of poisoning consists in lowering the patient's head, giving cardiac stimulants and, in extreme cases, artificial respiration.

*Novocain* is still, perhaps, the most popular of the cocaine derivatives used for producing analgesia; it is of low toxicity, is not damaging to the tissues, and is effective in concentration as small as 0.5 per cent. Its action is not so prolonged as that of some other preparations, especially when infiltration is used, but the addition of adrenalin, 1 : 10,000, is sufficient to prolong analgesia.

**The Methods of producing Analgesia** are surface application, infiltration, nerve block, intrathecal and extrathecal injection.

The first, as has been said, is limited to mucous surfaces.

Infiltration or the saturation of the tissues is limited to surface surgery.

In *regional* or nerve-block analgesia the nerve trunks are affected by injecting the tissues round them. This requires a precise knowledge of anatomy and, if a large number of nerves have to be blocked, is very irksome to the patient. When this method is used the needle must be introduced and the syringe attached afterwards, as otherwise there is danger of injecting solution into a blood vessel.

*Intrathecal* injection implies the introduction of the analgesic within the theca of the spinal cord. The cord ends at the upper border of the second lumbar vertebra, and the injection should be made below this level. *Novocain* seems most suitable for this procedure, 4 c.c. of a 5 per cent. isotonic solution being the maximum dose in general use. The level to which the analgesia will ascend appears to be determined within five minutes, after which it is safe to put the patient in the Trendelenburg position. The one cause for anxiety is fall of blood pressure, which can be mitigated by the intramuscular injection of 1 c.c. of ephedrine; but a modified Trendelenburg position is the best way of avoiding serious symptoms.

The most common complication is headache, which is generally very severe, continuous, intractable and associated with diplopia. This may even last for six weeks and sometimes may be cured by the withdrawal of cerebrospinal fluid. Transient incontinence of urine and faeces sometimes occur either alone or with paraplegia; the latter condition has been known to be permanent. As a general rule it may be said that operations above the umbilicus should rarely be attempted, and the method should be avoided when blood pressure is low.

*Extradural Analgesia* is obtained by traversing the membrane of the sacral hiatus between the bifid spinous process of the fifth sacral vertebra and the tip of the coccyx. The membrane having been pierced, the direction of the needle is altered to the plane of the sacral canal and introduced for about 2 in.; its arrival in the canal can be ascertained by the ease with which it can be moved from side to side and by the ready flow of solution. *Novocain* in 2 per cent. solution

pulmonary embolism generally becomes blood-stained very soon. The treatment of these conditions is not within the province of the anæsthetist.

**Post-operative Conjunctivitis** is generally attributed to the anæsthetic, and all that need be said is that care and delicate handling of the conjunctiva by the anæsthetist when he elicits the corneal reflex and attention to keeping the eye closed during the whole of the operation prevent the condition occurring.

### ANALGESIA

The production of analgesia is so vast a subject that a detailed description of the methods used is outside the scope of this article ; indeed, no more than a general consideration can be attempted.

The first essential is an accurate knowledge of the anatomy of the nervous system, especially of the distribution of the sensory nerves, without which the success to be expected in the more elaborate undertakings is small.

Nothing short of complete absence of pain is to be sought, for partial success is likely to be worse than failure owing to the lapse of confidence it causes. The promise that all sensation will be abolished should never be made, since often the sense of touch or pressure is retained and may be misconstrued as pain.

There are but few operations which cannot be performed under analgesia, but the various methods should not be used without discrimination ; persons of unstable disposition, even in spite of narcotics, may often endure mental anguish that leaves an indelible impression on their minds.

The introduction of an analgesic causes trauma to the tissues, and their resistance at the site of injection is lowered ; therefore the method should not be used in the presence of sepsis or in conditions in which the blood stream is infected. The minimum quantity of the solution should be injected, and the strictest aseptic technique practised ; syringes and needles must not be boiled in water containing bicarbonate of soda, as the minutest quantity of this substance in the analgesic solution is likely to render it ineffective, as does spirit.

A great number of different substances are used for the production of analgesia. each having different characteristics and different degrees of toxicity ; the commonest are synthetically prepared and are derived from cocaine.

The use of *cocaine* is now limited almost entirely to direct application to mucous surfaces—the eye, nose, throat, larynx, etc. Its local action is very powerful, and if absorbed in any quantity into the circulation can cause in the susceptible giddiness, faintness, vomiting, epileptiform convulsions and even coma and death. Such untoward symptoms appear to be due to rapidity of absorption ; the addition of adrenalin 1 : 10,000 to the solution tending to prevent this by causing ischæmia at the point of application. The strongest solution commonly used is 10 per cent., and this only for surface application in nose and throat work ; great care should be taken that none is

swallowed as absorption from the stomach is rapid. A 5 per cent. solution is the usual strength used in operations on the eye and for intratracheal injection, etc. Cocaine should never be used in the urethra, the mucous membrane of which seems to be singularly permeable. The treatment of poisoning consists in lowering the patient's head, giving cardiac stimulants and, in extreme cases, artificial respiration.

*Novocain* is still, perhaps, the most popular of the cocaine derivatives used for producing analgesia; it is of low toxicity, is not damaging to the tissues, and is effective in concentration as small as 0.5 per cent. Its action is not so prolonged as that of some other preparations, especially when infiltration is used, but the addition of adrenalin, 1 : 10,000, is sufficient to prolong analgesia.

**The Methods of producing Analgesia** are surface application, infiltration, nerve block, intrathecal and extrathecal injection.

The first, as has been said, is limited to mucous surfaces.

Infiltration or the saturation of the tissues is limited to surface surgery.

In *regional* or nerve-block analgesia the nerve trunks are affected by injecting the tissues round them. This requires a precise knowledge of anatomy and, if a large number of nerves have to be blocked, is very irksome to the patient. When this method is used the needle must be introduced and the syringe attached afterwards, as otherwise there is danger of injecting solution into a blood vessel.

*Intrathecal* injection implies the introduction of the analgesic within the theca of the spinal cord. The cord ends at the upper border of the second lumbar vertebra, and the injection should be made below this level. *Novocain* seems most suitable for this procedure, 4 c.c. of a 5 per cent. isotonic solution being the maximum dose in general use. The level to which the analgesia will ascend appears to be determined within five minutes, after which it is safe to put the patient in the Trendelenburg position. The one cause for anxiety is fall of blood pressure, which can be mitigated by the intramuscular injection of 1 c.c. of ephedrine; but a modified Trendelenburg position is the best way of avoiding serious symptoms.

The most common complication is headache, which is generally very severe, continuous, intractable and associated with diplopia. This may even last for six weeks and sometimes may be cured by the withdrawal of cerebrospinal fluid. Transient incontinence of urine and faeces sometimes occur either alone or with paraplegia; the latter condition has been known to be permanent. As a general rule it may be said that operations above the umbilicus should rarely be attempted, and the method should be avoided when blood pressure is low.

*Extradural Analgesia* is obtained by traversing the membrane of the sacral hiatus between the bifid spinous process of the fifth sacral vertebra and the tip of the coccyx. The membrane having been pierced, the direction of the needle is altered to the plane of the sacral canal and introduced for about 2 in.; its arrival in the canal can be ascertained by the ease with which it can be moved from side to side and by the ready flow of solution. *Novocain* in 2 per cent. solution

with 1 : 10,000 adrenalin is used and the minimum quantity injected is 50 c.c., which may be increased to double if the analgesia be required above the umbilicus. Experiments seem to show that the nerve-trunks alone are affected, the dura mater acting as a barrier to further progress. The method is not popular owing to the uncertainty of the analgesia produced, but it appears to be free from the injurious effects that sometimes accompany intrathecal injections. The full effects of the analgesic are generally not obtained under thirty minutes.

A. W. MATTHEW.

## CHAPTER XII

### PHYSIOTHERAPY AND RADIOTHERAPY

#### PHYSIOTHERAPY

**T**HE surgeon's art does not come to an end, when the last stitch has been introduced and the dressing applied after an operation, or when the displaced ends of a fractured bone are replaced in anatomical alignment. In many surgical diseases the operation—important as it may be—is but the beginning of a course of treatment, which is designed to restore the function of the local operation zone and the patient's general bodily activity to normal. Many physical agencies have been brought to the surgeon's aid in his attempt to restore health after removal of diseased tissue or after the repair of damaged parts.

**The Prevention of Deformity** will be emphasised in many chapters of this book, and is an outstanding consideration in the treatment of fractures, nerve injury and lesions of the central nervous system. A few examples only will be given here to impress upon the reader the utmost importance of this aspect of surgical work. When a peripheral motor nerve is injured, the muscles supplied by it become paralysed and atrophied, while the unopposed group gradually contract and pull the joints, on which they act, into the position of the full range of their movement, unless the limb is adequately splinted to prevent this deformity. Such a condition is very difficult to correct, owing to the fibrosis of the contracted muscles and to the inability of the paralysed muscles to regain their function, even when the regenerated nerve has reached them; yet it is so easily prevented by splinting in such a position that the paralysed muscles are relaxed and their opponents are stretched.

Many injuries, *e.g.*, fractures and dislocations and many diseases, *e.g.*, infections of joints, require immobilisation, as the result of which a fear will always exist that stiffness or fixation of joints may occur. Where it is possible the parts must be placed in the position, in which their function is at its best in spite of the possible handicap of adhesions. The *position of function* of the hand (Fig. 75) is an excellent example, and another is the "position of election" for ankylosis in a joint, the exact position in each joint having been carefully worked out to ensure the maximum function of the limb.

After operations *position* is often of the utmost importance. After the radical removal of a carcinoma of the breast, the movements of the shoulder are well-nigh perfect in spite of the mutilation of the pectoral group of muscles. If, however, the arm were kept close to

the body during convalescence, a very limited range of movement would result, but by placing the arm on a pillow abducted to  $75^{\circ}$  from the body a full range of movement can be obtained.

The prevention of deformity is nowhere better exemplified than in the treatment of burns in the region of joints. Scar tissue inevitably contracts unless prevented by careful splinting and severe flexion of a joint may follow a burn. In such patients, after the initial stage has passed, treatment must include careful splinting in a position of extension until all risk of contraction has been overcome.



FIG. 75

The position of function.

**Restoration of Function.**—It is a sad commentary upon our scale of values that it has required a second world war, an attack upon the civilian voter, to persuade the Government that it is not enough to tend the wounded but that every injured person needs to be restored—as far as possible—to full wage-earning capacity. Again in the past no influence could persuade insurance companies dealing with workmen's compensation that a man after treatment needed a period of graduated work to get him back to full strength for

active duty. In connection with infections and injuries of the hand I have been preaching the overwhelming importance of restoration of function for many years. Total war has at last convinced authority that "rehabilitation" is an essential part of their responsibility. The following description of physical agencies at our disposal will be better appreciated if this final objective is kept in mind. Further, let it be understood that our task is easier in every condition if diagnosis has been promptly and correctly made and efficient treatment has been made available at the earliest possible moment.

**Massage** has as its objects (1) to stimulate the circulation of an injured or immobilised part by acceleration of the flow of venous blood, and so increasing the supply of fresh arterial blood; (2) to assist in the absorption of inflammatory exudates by again increasing the venous and lymphatic drainage; (3) to maintain the tone of the muscles, preventing their atrophy and keeping them in training so that they can resume work quickly when the disability has been cured; (4) to prevent the formation of adhesions in and around joints, and to help to remove articular effusions; (5) when given to the whole body to act as one of the most powerful means of inducing sleep in patients suffering from insomnia, due to nervous strain or breakdown; and (6) greatly to increase the tone of the internal organs, thus assisting in the cure of constipation, etc.

Massage has become a complex subject and has been divided into several types. *Effleurage* is the simple stroking movement with the flat of the hand, which has been dusted with talcum powder. The



stroke from the periphery towards the heart is strong and the return is light. The strokes are at first light and gentle and become increasingly strong and deep. This drives blood and lymph onwards and encourages the entry of new blood, which is laden with antibodies. *Petrissage* consists of a series of kneading movements with the hands, starting at the periphery and working upwards to the root of the limb. This is designed to promote the absorption of exudates and to improve the circulation. *Tapotement* denotes the rapid repetition of sharp blows with the ulnar border of the fingers and palm alternatively with each hand. These blows tend to stimulate the vessels of the area and so to increase the blood supply.

**Movements and Exercises.**—PASSIVE MOVEMENT is carried out by the masseuse and not by the patient. Joints are moved very gently and the range of movement increased as rapidly as possible. It should start at the earliest moment, must produce no pain, and must not interfere with the diseased or injured part. It is customary to carry out the movements after the muscles and joints concerned have been massaged. It aims at keeping moving parts supple and free from adhesions, so that, when active movements are resumed, everything works smoothly.

ACTIVE MOVEMENTS must replace passive as soon as possible and this entails confidence and mental effort on the part of the patient. Care must be taken to prevent weak muscles from attempting too severe a task, and their work must be carefully graduated. As an example, an internal derangement of the knee-joint may require considerable immobilisation, with the result that the quadriceps muscle is weak and wasted. In treatment the limb is placed fully extended on a couch and the patient is instructed to "tighten" the muscles of the thigh; later, the muscles are able to extend the knee when the limb is supported on the side, and finally, sufficient strength has been regained for extension to be produced in the normal way.

Such active movements are sufficient for minor maladies and trivial muscular weakness, but after a serious nerve injury or following anterior poliomyelitis, the muscles demand more complicated and co-ordinated movements than can be produced by word of command. Exercise by machines has become a recognised feature of re-education and a modern fully-equipped department will have a large number of mechanical contrivances, some simple, others highly complicated, but all designed to exercise a definite group of muscles and to produce a co-ordinate series of movements. In these machines an adjustment of the moving parts is provided, so that the effort required can be made increasingly more arduous as the muscles regain their power.

**Heat** produces a vasodilatation of the surface vessels, and this leads to an increased flow of arterial blood, to which process the name "active hyperæmia" is given. Heat may be applied in many ways.

**MOIST HEAT.**—The hot fomentation is the best-known method of applying moist heat. It is prepared by cutting out four layers of lint to a size well overlapping the inflamed area. These are placed in a

calico wringer and immersed in boiling water for one minute. The water is completely wrung out of the lint, which is applied to the skin and covered with a layer of waterproof material and wool and bandaged in position. Its disadvantages are that it loses its heat so quickly and that it tends to spread infection to the previously normal skin. A linseed poultice is a better vehicle of moist heat, but it has been largely replaced by "antiphlogistine." Heat can be retained in these dressings by the application over them of electrically heated pads.

For many purposes hot baths give better results than hot dressings, and if the part can be easily immersed, this method should always be adopted. Special baths are made for the leg, the arm and the hand, and the solutions used are iodine (one drachm of the tincture to the pint of water), normal saline, eusol or hydrogen peroxide. The temperature should be about 110° F., at which it should be maintained for thirty minutes, while the limb is immersed in the bath.

Moist heat is used in the treatment of septic wounds and surface sepsis, such as boils and carbuncles, for which fomentations and poultices are of real value during the first forty-eight hours. If the pus has been given adequate drainage and all tension has been relieved, it is never necessary to continue with these dressings for longer than two days. Fomentations are often used for too long a time with the result that the skin becomes white and sodden and small pustules are seeded out on previously healthy skin. Baths are often useful for many days in cases of severe sepsis, but dry dressings should be used in the intervals. Baths have an entirely different application, which is outside the scope of the surgeon and will be found in textbooks of medicine, as for example, in the spa treatment of rheumatic and arthritic disorders.

DRY HEAT is immeasurably superior in every case in which there is no septic wound or surface sepsis, and even in these dry heat should be substituted for moist as soon as the sepsis has subsided and the wound is cleanly granulating. It may be obtained from several sources. Radiant heat baths are of many types and sizes, some being large enough to include the whole trunk and thighs, and others designed for a small area such as the hand (see Figs. 60 and 117). These baths are supplied with electric lamps, coupled with a number of switches so that the temperature can be regulated. Another source of heat extensively used to-day is from the infra-red end of the spectrum. Small portable applicators may be obtained for use in private houses and for small areas, while larger installations are available for the treatment of the body as a whole.

These methods of treatment produce their effects on the surface only, and in deep-seated lesions it is desirable to obtain heating of the more deeply placed tissues. This is possible by the use of "diathermy" and "short-wave therapy" (see below).

Dry heat is used for many purposes in treatment. One of the chief factors in the causation and maintenance of shock is the loss of body heat, and radiant heat baths are the best means of counteracting surgical shock. Operating theatres are kept well heated for this

reason, and the bed is warmed by radiant heat cradles for the reception of the patient after operation. Many lives were saved during the first Great War by the use of hot resuscitation chambers in the advanced and main dressing stations and casualty clearing stations; resuscitation rooms are prominent features of casualty reception hospitals in bombed cities in this war. Dry heat is also of great value in the treatment of septic wounds and in a great variety of inflammatory lesions, such as fibrositis, lumbago, rheumatism, rheumatoid and osteo-arthritis, neuralgia and neuritis. Especially important is it in assisting the recovery of movement after injury or infection, as for example, in the restoration of function in the fingers and hand.

ACTUAL HEAT by direct contact is used in surgery for many purposes and is applied by means of a "cautery." The *actual cautery* denotes the use of iron rods of varying shapes and sizes heated to a dull red heat. It is rarely applied in this form to-day owing to the facility

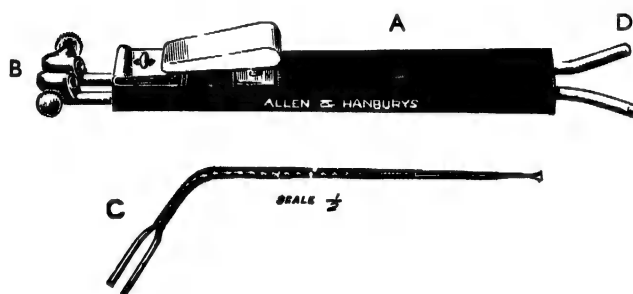


FIG. 76

An electric cautery. (Allen & Hanbury's)

- A, is the insulated handle with its contact key;  
B, the clamps to which the cautery points C are fixed; and  
D, points for the attachment to the electrical supply.

with which cautery points can be heated electrically. As a means of producing counter-irritation, the actual cautery is utilised to inflict a number of parallel seared lines on the skin over a painful area, *e.g.*, in the back, especially when all other methods have failed and a strong neurasthenic element is present. The *electric cautery* (Fig. 76) consists of a platinum wire loop of varying shape and size mounted in an insulated handle and connected to an electric battery or to the main supply via a rheostat. The platinum point can be brought to any degree of heat by regulating the current. It has completely replaced the actual and Pacquelin cautery for many purposes. Hæmorrhage from mucous surfaces and the depths of wounds may be arrested by sealing the vessels with a dull red heat. Hypertrophied mucous membranes, *e.g.*, in the nose, in minor degrees of prolapse of the rectum and in caruncles of the female urethra may be reduced by linear cauterisation, and hæmangiomata and small tumours of the skin and mucous membranes, such as papillomata and polypi, can be removed by cauterisation. The so-called diathermy cautery will be described below.

### ELECTRICAL METHODS

**The Galvanic Current** is a constant current derived from a battery. It is used in conjunction with the Faradic current in testing the reactions of muscles and nerves, and in treatment it has a mildly beneficial action by stimulating the surface vessels. It is chiefly used in the method of treatment known as "ionisation." If two metal electrodes are covered in lint which is soaked in various solutions and they are applied to the skin, the passage of a galvanic current will drive the positive and negative ions into the skin from the opposing electrodes. This phenomenon is made use of in the treatment of rheumatic or traumatic lesions, zinc, iodine or salicylate ions being driven into the skin from the positive or negative electrodes.

**The Faradic Current** is an alternating current produced by an induction coil. It is used in conjunction with the galvanic in testing muscles and nerves (Chap. XLIII). In treatment it is invaluable in the stimulation and re-education of wasted muscles.

**The Sinusoidal Current** is also an alternating current. The patient or one of his limbs is placed in a bath of warm water and the current is passed through it. This is recommended for cases of paralysis and chronic nerve pain.

**Diathermy** is a term which has been rather loosely applied to cover a range of electrical manifestations. Essentially, "diathermy" is a high frequency alternating current with a very rapid rate of sustained oscillation. It is used in four separate ways, medical diathermy, surgical electro-coagulation, electro-desiccation and surgical cutting.

In **MEDICAL DIATHERMY** the two electrodes are of considerable surface area and the current passes from one to the other through the soft tissues of the patient which lie between them. Heat is produced in these tissues, but it has been shown that the current flows chiefly round a limb in the plane of the subcutaneous or deep fascia, and little passes directly through the muscles in a direct line from one electrode to the other. The heat produced, therefore, is largely confined to those tissues lying close to the electrodes. Medical diathermy is used in the treatment of a large number of diseases, *e.g.*, gonococcal lesions in the vagina, urethra, cervix and tubes of women, in the epididymis, prostate and vesicles of men, and in rheumatic manifestations and arthritis of varying types.

**SURGICAL DIATHERMY** covers three distinct fields of usefulness.

*Electro-coagulation* is produced when one electrode is reduced to a very small area such as a metal button or a needle point. The current is concentrated at the point so that the cells are coagulated and killed by the heat. This method is used in the treatment of benign and malignant growths, vascular naevi and lupus vulgaris. It has also a real place in the treatment of rodent ulcer, especially after other forms of therapy have failed.

*Electro-desiccation* is produced by a somewhat different type of high-frequency current and is used for the destruction of warts, true papillomata of the skin, painful corns and small naevi. *Acusection* or

surgical cutting is derived from a current of very high frequency capable of producing an arc of intense heat at the electrode. A wire loop or needle is used and all soft tissues are cleanly and instantaneously cut by the moving electrode. This method is used by some surgeons in extensive dissections, *e.g.*, the radical removal of the breast. Small blood vessels and lymphatics are sealed by the arc and malignant tissues can be removed by it. It is this type of current which has had such brilliant results in the operation of transurethral prostatic resection.

**SHORT-WAVE THERAPY**, introduced by Professor Schliephacke, is based on the action of high frequency currents with oscillations of 10 to 100 million cycles per second. Whereas medical diathermy heats the tissues nearest to the electrodes, short wave therapy is said to produce heat in the depths of the tissues, which are traversed directly from one electrode to another. It is now being used for acute, subacute and chronic inflammatory diseases, such as nasal sinusitis, boils and carbuncles, certain eye lesions, axillary and cervical adenitis, certain bone and joint diseases, sciatica and lumbago, neuritis and neuralgia and for stimulating the more rapid healing of wounds.

**Ultra-violet Light** is produced by the passage of current through mercury vapour tubes. It is used for many purposes, especially in those deficiency diseases in which absence of sunlight and fresh air play a part. It is also used for its general tonic action and confers increased resistance to infectious ailments. It acts by producing calciferol in the deeper layers of the skin.

### CORRECTION OF DEFORMITY

Should a deformity have occurred or movements of joints be restricted, complete function cannot be restored until the former has been corrected and the latter made free. A complete account of such treatment is obviously impossible here but some of the methods can be indicated.

The first essential to success is that each patient must be inspired by an overmastering enthusiasm to get well as quickly as he or she can. The competitive spirit can be a splendid stimulus and for this reason patients with similar lesions should always be treated side by side in full view of each other. Later machines are of value in amusing people as well as treating stiff joints and weak muscles. Often no elaborate or expensive apparatus is required and much ingenuity can be expended in designing simple home-made "gadgets"; many patients, mechanically minded, will suggest their own improvements. Pulleys, weights, string, etc., form the basis of such machines. Figs. 77 and 78 show various types of exercisers. Figs. 77, 78 and 79 are examples of special splints designed to overcome stiff and deformed fingers; they serve here merely to illustrate the type of splint which can be adapted to many joints. These appliances are designed to produce their effects by persistent traction obtained by strong elastic bands. This method has many advantages over forcible manipulation.



FIG. 7

A digitorium or dummy practice keyboard. An adjustment gives variation in the tension of the springs.



FIG. 78

Home-made exerciser for fingers and thumb



FIG. 79

A simple palmar cock-up splint giving "position of function." On right it has been adapted to pulling stiff fingers into flexion at the metacarpo-phalangeal joints. (Kanavel.)



FIG. 80

A dorsal cock-up splint producing flexion of the fingers, opposition of the thumb and dorsi-flexion of the wrist. (*Kanavel.*)

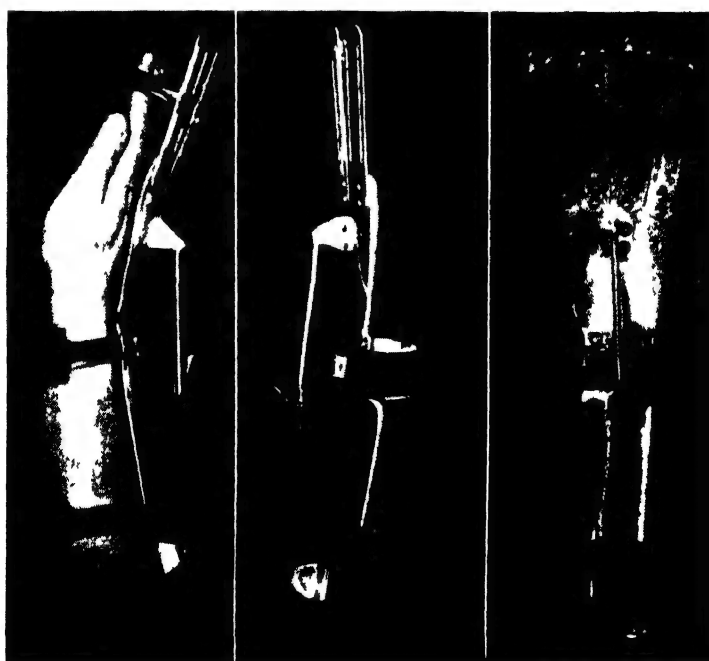


FIG. 81

Splint for obtaining full extension of the fingers and dorsi-flexion of the wrist (*Kanavel.*)

## X-RAYS

X-rays are a specialised type of ether waves which have the property of penetrating many substances impenetrable to human sight. The heavy metals and their salts are impervious to X-rays, and generally speaking, the higher the atomic weight, the greater the resistance to these rays. It would be out of place to discuss the physics of X-rays and their production, but certain details in the technique of radiography must be described.

X-rays penetrate the various tissues of the human body to varying degrees. The bony skeleton containing calcium salts is resistant and throws a sharp picture on a photographic film, and by a specialised technique indistinct outlines of soft organs such as the kidneys can be obtained. In clinical surgery, X-rays are employed for two purposes, viz., diagnosis and treatment.

### X-RAYS IN DIAGNOSIS

X-rays are utilised in diagnosis by exposing radio-sensitive film, and by the use of specially prepared screens to visualize the rays. Barium platino-cyanide and calcium tungstate have the property of fluorescence when exposed to X-rays, and this is utilised in the manufacture of the fluorescent screen, made by coating a firm piece of cardboard with one of these solutions and mounting it in a frame. Screening is an essential part in the technique of the radiography of the heart, lungs, and gastro-intestinal tract, the localisation of foreign bodies and the reduction of fractures.

X-ray films are made in exactly the same way as ordinary photographic film, silver salts having the same reaction to both visible light and X-rays. The film is enclosed in black paper which is, of course, penetrated by the X-rays. The fluorescence of calcium tungstate is used to increase the definition of the image and to decrease the time of exposure. Intensifying screens are made of fine art cardboard coated with an emulsion of calcium tungstate. The film, which is prepared on both sides, is removed from its coverings in the dark room and mounted in a special "cassette" with an intensifying screen applied to each surface. The cassette when closed is light-tight and its surface presented to the rays consists of a thin sheet of aluminium. The exposure is divided by ten by this procedure and thereby instantaneous photography is possible, which is of great value in taking radiograms of moving structures, *e.g.*, the heart.

Definition of the image can be further improved by the elimination of all secondary radiations, and this is achieved by the use of the Potter-Bucky diaphragm, which embodies a wide strip of canvas to which are applied alternating strips of lead-foil and wood. This apparatus is interposed between the patient and the film, and the canvas strip is in slow motion during the exposure.

Pictures must be taken of any subject in two planes at right angles to each other, usually direct anteroposterior and true lateral. If this should not be feasible, stereoscopic films are taken. Two separate





FIG. 82

A lateral radiogram of a normal skull showing the  
pituitary fossa

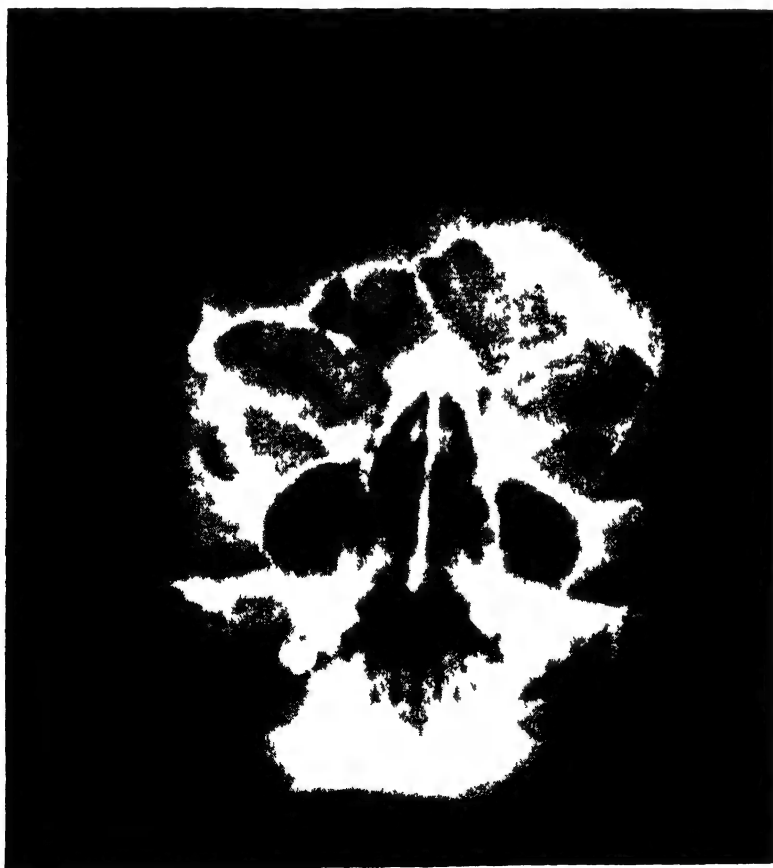


FIG 83

An anteroposterior radiogram of a normal skull showing the  
nasal sinuses.

exposures are made; in each case the films and the patient occupy identical positions, but the tube is moved 6 cm. between the two exposures. The films are viewed in a special apparatus, which enables one picture to be seen by the right eye and the other by the left eye, and the component parts of the area in the photographs stand out in their true relationship.

**The Skull.**—In injury or disease of the vault or base (Figs. 82 and 83) anteroposterior and lateral views are required, and if any doubt exists a stereoscopic pair of films may help to dispel it. The air sinuses require a special technique. The head is hyper-extended with the film placed in front, just touching the tip of the nose and the front of the chin. This will show all the sinuses except the sphenoidal. Each pair of frontal and ethmoidal sinuses and maxillary antra can be compared side by side. The teeth are photographed on small films in waterproof envelopes, which are placed against the buccal surface of the teeth and held in position by the patient.

**The Spinal Column.**—The standard anteroposterior and lateral view technique is applied throughout the whole column with one or two exceptions. To obtain views of the atlas and axis the patient has the mouth propped open with a dental gag and the rays pass through the mouth. Lateral views of the upper six dorsal vertebræ are not very satisfactory owing to the shoulder girdles, and a stereoscopic pair of films may be needed. **The bones and joints of the arms and legs** provide no departure for standard technique. Fractures, dislocations, subluxations and disease of the bone are well seen, but displacements of soft parts, such as intra-articular cartilages, are not shown.

**The Thorax** is most advantageously examined by the use of the fluorescent screen and by photography. The expert radiologist can obtain much information from screening; the range of movement of the diaphragm on each side, the nature and regularity of the heart movements, the displacement of the heart from its normal position and the movement of each side of the chest can be closely studied. Photographs are taken with intensifying screens, and without the Potter-Bucky diaphragm, so that exposures of one-fifth to one-tenth of a second are possible. The patient is examined in the erect position or seated on a chair. The "tomograph" is referred to in Chap. XXIV.

The level of air or fluid in the pleural cavity will be shown, while evidence of tuberculous disease or neoplasm of the lungs can be identified. Further detailed examination of the lungs is obtained by the injection of lipiodol into the trachea (Fig. 84). The posterior mediastinum is best seen in photographs taken in the left oblique position; the size of the aortic arch and the presence of tumours or enlarged glands can be demonstrated (see illustrations in Chap. XXIV).

**The Gastro-intestinal Tract** is not visible unless filled with an opaque solution. Barium sulphate is an insoluble salt and is opaque to X-rays. It is put up in several different preparations by various firms. It may be mixed with milk, hot chocolate, or Horlick's malted milk and made into a thick creamy consistency. Screening in skilled hands gives more reliable and more extensive information than photography.

The *Œsophagus* is examined while the patient is standing erect in the left oblique position, the barium meal being watched in its progress down the *œsophagus* (Fig. 85). If any doubt exists, a small piece of cotton-wool is well soaked in the meal and swallowed. Its progress is slower than the liquid and it can be watched more easily and will be held up by slight strictures which would prove no obstacle to liquids. Photographs are taken by the double intensifying screen technique without the diaphragm. The *stomach* is examined by visual



FIG. 84

Photograph of the chest, illustrating the use of lipiodol injection into the bronchial tree.

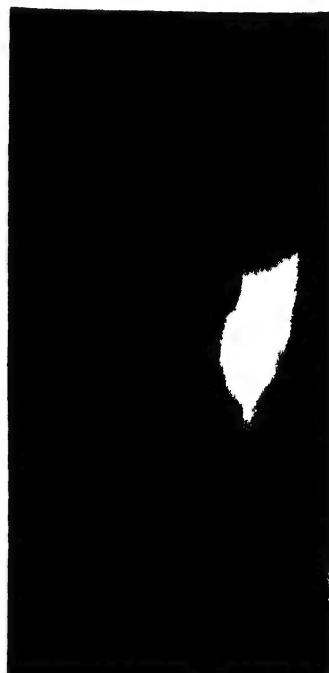


FIG. 85

A lateral view of a barium swallow held up in the *œsophagus* by a carcinomatous stricture.

screening during its filling with the meal. Peristalsis is watched and compared with normal, and the general shape, size and position of the organ noticed. Massage with the gloved hand is then carried out to move the barium about so that the curvatures, the pyloric antrum, the pylorus and the first part of the duodenum can be examined. Photographs will be taken at definite intervals and the time noted at which the stomach is completely free from barium, and should normally occur within four hours.

*The Duodenum.*—The first part of the duodenum is physiologically closely allied to the stomach, and can be easily demonstrated and examined by visual screening and photography. The other parts of the duodenum are not easy to investigate, as the meal is passed very rapidly into the jejunum. The first part is known radiologically as the Duodenal Cap, being shaped somewhat like a triangular hat.

*The Small Intestine* yields few results of value from barium-meal examination. Abnormalities of position may be seen and occasionally the appearance suggests the presence of chronic intestinal obstruction. The meal should have reached the ileocæcal valve in two and a half to three hours, and be entirely passed into the colon in six to eight hours.

*The Appendix* can be visualised in many patients, but its non-appearance is no evidence of disease. It is examined by screening and palpation during a barium meal after the small intestine has emptied itself into the colon. It should be clearly understood that a diagnosis of chronic or subacute appendicitis is never justifiable on X-ray evidence alone.

*The Opaque Enema* is used for the investigation of the rectum and large intestine. The patient is to be prepared in every respect as if he were to be operated on for rectal disease. A suspension of barium sulphate in water is slowly injected by an enema syringe or merely by gravity from a douche can. The patient lies on the back with the tube below and the screen above and the passage of the enema is carefully noted. Photographs are taken by the double intensifying screen technique with the Potter-Bucky diaphragm. If diverticulitis is suspected, a final photograph must be taken after the opaque enema has been washed out during the following day. The little pockets of barium in the diverticula will be left behind and give a typical picture (see Chap. XXIX).

**The Biliary Tract.**—Gall-stones do not show on a direct X-ray photograph, except in a small proportion. Recent advances in technique have made it possible to visualise the gall-bladder, a process known as Cholecystography. It cannot be said that the method is yet perfect, but it does add much to the armamentarium of the diagnostician. Sodium tetra-iodophenolphthalein is opaque to X-rays, is secreted by the liver cells and appears in the bile. It may be administered by the mouth or intravenously, the latter method giving definitely superior pictures, but it is accompanied by toxic symptoms in so many patients that the oral route has become the routine method. The patient, having been carefully prepared for forty-eight hours, is kept on a fat-free diet during the whole of the day before examination, and no food is given after a light supper at 7 P.M. with which is taken the keratin-coated capsule of the drug. Twelve to fourteen hours later photographs are taken and then a fatty meal is given; further photographs are taken two and four hours later. The gall bladder should be seen filled and distended at the first examinations, and partially contracted and filled with concentrated bile later.

A normal gall-bladder will show a regular pyriform shadow; if the cystic duct is obstructed no shadow will appear; and if the gall-bladder is full of stones, a curious honeycombed type of shadow may be seen (see Chap. XXXIII).

It must be clearly realised that only positive findings are to be relied upon, and that the absence of a shadow is not sufficient evidence of a diseased gall-bladder.

**The Urinary Tract.**—The close anatomical relationship of the colon to the kidneys and ureters makes a careful preparation of

the patient an essential preliminary to all urinary radiography, for the outlines of the kidney can be completely obliterated by gas in the colon. The urinary tract, *i.e.*, kidneys, ureters and bladder is examined by photographs, the patient lying supine with the tube above and the film below. During the exposure the patient is instructed to hold the breath. Double intensifying screens and the Potter-Bucky diaphragm are used. A lateral view must also be taken so that the relationship of radio-opaque shadows to the vertebral column can be defined, this being of importance in the differential diagnosis of renal and biliary calculi.

*Intravenous Urography* permits the visualisation of both renal pelves, ureters and the bladder with an opaque fluid. Twenty cubic centimetres of uroselectan B are injected into a vein in the antecubital

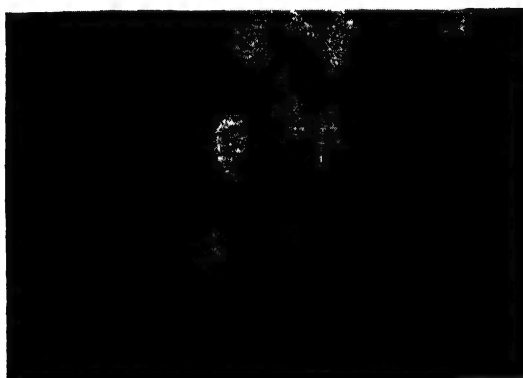


FIG. 86

The appearances produced during an intravenous urography in a normal patient.

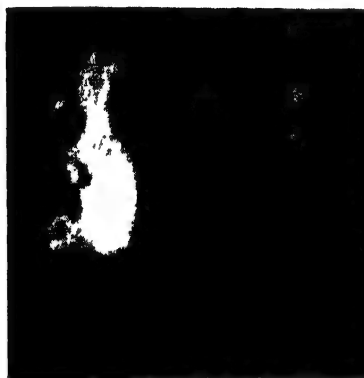


FIG. 87

An instrumental pyelography of the right kidney in the same patient.

A study of these two photographs shows the differences in density and detail produced by the two methods.

fossa, and the dye should make its appearance in the urine within 4 minutes. Photographs are taken at 3, 8, 12, 25 and 40 minutes (Fig. 86).

*Instrumental Pyelography* is somewhat more complicated in that a cystoscopy must be done in order to introduce into the ureter a catheter, through which a 20 per cent. solution of sodium iodide is injected into the renal pelvis. The patient must be fully conscious when the injection is made, and the fluid must not be introduced at a pressure higher than 15 mm. of mercury, lest the kidney be damaged (Figs. 87 and 88).

Intravenous urography has become the routine method, as it is so simple to perform and gives very satisfactory results. The density of the shadow is not great and in stout subjects points of fine detail are not shown, and the method is not suitable for any patient whose renal function is seriously impaired. Instrumental pyelography results in a much denser shadow, and is reserved, therefore, for patients in whom the intravenous urogram has left a doubt in the minds of the surgeon and radiologist and those in whom the intravenous route is contraindicated.

The ureters can be visualised by intravenous and instrumental pyelography and by the passage of an opaque bougie through a cystoscope. These methods are essential in the recognition of a small shadow suspected of being a ureteric calculus.

The bladder is examined by photographs, which will reveal the presence of radio-opaque calculi. The bladder itself may be visualised by filling it with 20 per cent. sodium iodide solution, and photographs should be taken in the anteroposterior, lateral and right and left oblique positions. A cystogram will show the presence of diverticula.



FIG. 88

Another example of an ascending pyelogram showing a filling defect in the centre of the pelvis due to an air bubble.



FIG. 89

An anteroposterior view illustrating the arrest of lipiodol in the spinal theca by a neoplasm.

**The Central Nervous System.**—Intracranial disease can occasionally be diagnosed by special radiographic methods. Ventriculography is a method whereby a needle is introduced into one lateral ventricle, cerebrospinal fluid withdrawn and replaced by sterilised air. Radiograms show any deformation of the ventricular shadows. The pineal gland usually contains areas of calcification, and by special technique it can be shown in anteroposterior and lateral pictures. Its shadow should be strictly in the middle line, and its displacement may give an indication of the presence and location of a cerebral tumour.

**The Spinal Cord** may be investigated and the level of tumours decided by the injection of lipiodol, an oily inert substance containing iodine, 1 c.c. of which is injected into the cisterna magna. Being of a higher specific gravity than cerebrospinal fluid, it sinks to the bottom of the dural space in the sacral region of normal persons, whereas it will be held up if this space is narrowed or obliterated by growth or inflammation (Fig. 89). If this should occur, a second injection is

made into the lower part of the dural canal in the region of the third lumbar vertebra and the patient tilted head downwards. The resulting pictures give the level and the extent of the obstruction.

**Foreign Bodies** may be localised in several ways. Careful screening should give an indication of their position, and photographs taken in two planes exactly at right angles to each other give more certain information. Exact localisation can be done by certain complicated methods, which need not be described here.

**Arteriography.**—By special technique it is possible to take photographs of the arteries of the upper and lower limbs by injecting uroselectan into the axillary and common femoral arteries. The method is of some value in the assessment of the degree of arterial degeneration in certain diseases in which the circulation of the limb is poor. It is also used in the localisation of brain tumours and aneurysms.

### X-RAY TREATMENT

**Surface Therapy.**—X-rays are capable of producing severe burns of the skin if the dosage is excessive. X-ray dosage is controlled by testing the strength of the rays and by the time of exposure which will produce an erythematous blush of the skin. This is the "erythema" dose, and it is estimated by the Sabouraud technique. Small paper discs coated with barium platino-cyanide are exposed to the X-rays, which gradually change the green tint to a brown colour. By comparison with standard tints the dosage can be correctly determined. Surface lesions, such as ringworm, acne, lupus, squamous-celled carcinoma of the skin (Fig. 90), rodent ulcer and secondary carcinomatous glands, if close beneath the skin, react very favourably. The rays are filtered by aluminium screens and full erythema doses are given.

**Deep Therapy.**—So much of the power of the rays is lost in traversing the skin that only small doses reach deep-seated tumours.

Deeply penetrating short-wave X-rays are used for deep therapy, the rays being focussed in the deep-seated growth through a succession of different areas of skin, known as the "portals of entry." One skin portal is given a full erythema dose, and then on alternate days other portals are used, until the full dose has reached the growth. Recently a delicate dosimeter has been introduced, the use of which



FIG. 90

A photograph illustrating the cure of an extensive squamous-celled carcinoma of the cheek after radium and X-ray therapy.

allows an exact calculation of the dosage required. All types of deep-seated growth can be attacked by this technique, but in certain situations there is a real danger of damaging normal structures so seriously that deep therapy is considered unjustifiable. Deep therapy is essentially an expert's work ; full details can be obtained only from textbooks of Radiology.

## RADIUM

The physics of radium activity is outside the scope of this book ; suffice it to say that radium salts are strongly radio-active, and are almost inexhaustible, since they lose their potency at the rate of  $\frac{1}{2500}$ th of their weight each year. Radium may be administered in the form of needle or plaque containers. Needles are made of platino-iridium, inside which radium sulphate is carried in very small glass tubes in quantities of 0.5, 1 or 2 mg. The needles are inserted into the growth and have eyes which are threaded with silk or wire to permit their removal. Metal plaques are but rarely used, and external therapy is obtained by planting needles in a paraffin-wax mould applied to the skin. In the radium bomb a large quantity of radium (4 to 5 grm.) is concentrated in one metal container, and by special technique the bomb can be made to direct rays to deep-seated tumours in a manner somewhat comparable to a deep X-ray therapy tube. Fortunately the enormous expense precludes its use except in the hands of an expert.

Radium therapy should not be undertaken by anyone except after a course of instruction in its use. Its haphazard employment by unskilled workers cannot be too strongly deprecated. Imperfect dosage can do irreparable harm, and malignant growths can be stimulated into intense activity instead of being destroyed.

In the treatment of intrabuccal carcinomata and of cancer of the cervix uteri, radium therapy may be said to have replaced surgical excision, but in no other part of the body is this true. Radium is the handmaiden of surgery, not its supplanter. No operable growth is ever to be treated with radium alone, but must be removed surgically and radium used to irradiate the field of operation and the outlying areas of possible involvement.

Radium is of great value in the treatment of uterine disorders ; it will produce temporarily or permanently the artificial menopause, and many gynæcologists invariably prepare their patients for hysterectomy for carcinoma by preliminary irradiation.

Skin diseases, both inflammatory and malignant, respond well to radium, and growths of the larynx and pharynx have given encouraging results from bomb therapy.

Sarcoma and carcinoma show very varying degrees of response to the rays, but generally speaking, the more rapidly growing and the less differentiated type of cell responds most readily.

It must once again be emphasised that radium should be used only by those specially trained in its use.

R. M. HANDFIELD-JONES.



# CHAPTER XIII

## DISEASES OF THE SKIN

### INFECTIONS OF THE SKIN

**I**NFECTIONS of the skin are of pyogenic, specific or parasitic origin, and may be classified as follows :—

<i>Pyogenic</i>	<i>Parasitic</i>
Boils.	Ringworm.
Carbuncles.	Scabies.
Cellulitis.	Tinea Cruris, etc., etc.
Erysipelas.	Favus.
Impetigo Contagiosa.	
<i>Specific</i>	
Tuberculous . . . . .	(a) Bazin's disease.
	(b) Ulceration.
	(c) Lupus Vulgaris.
	(d) Verruca Necrogenica.
Syphilitic . . . . .	(a) Primary.
	(b) Secondary.
	(c) Tertiary.
Gout.	
Erythema Nodosum.	
Lupus Erythematosus.	
Erythema Pernio (Chilblain).	
Molluscum Contagiosum.	
Leprosy and other tropical diseases.	

### PYOGENIC INFECTIONS

**A Boil or Furuncle** is a localised infection of the skin due to staphylococcal invasion of a hair follicle or sebaceous gland, leading to suppuration and local gangrene. Occasionally, the inflammation stops short of suppuration and resolution occurs, the condition being then known as a "blind boil." A fully-developed or "ripe boil" consists of a central slough (the dead follicle or gland) surrounded by pus and a wall of active granulation tissue.

A boil may occur in any part of the skin supplied with hairs, most often in those areas subjected to rubbing or pressure, *e.g.*, the back of the neck in men where the collar rubs, the axilla and the face. It begins as a small red thickening which is tender rather than painful, and

from the centre of which a hair may be seen to protrude. It slowly increases in size, becoming conical in shape, dusky red in colour, painful and extremely tender. At the apex of the swelling a grey-white spot appears, around which the skin gets thinner until it bursts and pus exudes. Within three to five days the slough separates as a "core" which is extruded, after which the swelling subsides and the cavity granulates rapidly. The surrounding skin needs protection during treatment as satellite boils are likely to appear.

*Treatment.*—The blind boil should be left alone as interference may cause suppuration where none would have occurred. Many boils may be aborted and others brought more rapidly to a head by the application of a leech to the prominent centre, and by the intramuscular injection of manganese butyrate (1 c.c. every third day). As soon as pus is formed a small incision is made and the area treated three times a day with Bier's suction glass. The surrounding skin is carefully cleansed, swabbed with absolute alcohol and, before applying hot fomentations, smeared with a mild mercurial ointment to avoid the formation of satellite boils. When the slough has separated a small dry dressing will suffice. The practice of applying pure carbolic acid on the pointed end of a match or probe can do nothing but increase the amount of necrosis and should never be employed; and a similarly popular practice of squeezing the boil cannot be condemned too strongly as it may be the determining factor in the production of a staphylococcal septicæmia.

**Furunculosis or Recurrent Boils.**—This is an indication of lowered resistance on the part of the patient, who should be examined thoroughly to ascertain the cause. Early chronic interstitial nephritis, diabetes or other serious organic disease may be present, but the common predisposing factor is debility due to overwork, nervous exhaustion and lack of a holiday. The tendency to recurrence may be most persistent and treatment will often prove difficult as patients are adamant in their refusal to give up their work and go away for a real holiday. The condition is deserving of more serious attention than it usually commands, as such dangerous diseases as acute osteomyelitis in children, and perinephric abscess and septicæmia in adults are known to have their origins in a simple boil.

*Treatment* consists in the local attention to each boil as it occurs, and general therapy to improve the patient's condition. The combination of sun and salt water cannot be surpassed and, if possible, the patient should be sent to the seaside. Failing this local infra-red and general ultra-violet radiation should be employed.

**A Carbuncle** starts as a staphylococcal or rarely as a streptococcal infection of a hair follicle and sebaceous gland or of a sweat gland; but, whereas in a boil the process remains localised, in a carbuncle it spreads more deeply. When pus ruptures out of a hair follicle it enters a "columna adiposa," along which it follows the path of least resistance in the subcutaneous fat until it reaches the loose connective tissue meshwork beneath the skin. The infection is then able to spread centrifugally and pus tracks up adjacent columnæ adiposæ and so reaches the surface at many points. The central area becomes necrotic

and pus and cellular debris ooze from it, but as the opening is insufficient for free drainage, the infection continues to spread at the periphery beneath the skin, and an untreated example will spread over a large surface area. A well-developed carbuncle shows four separate zones (Fig. 89); in the centre is the necrotic area, around which is a punctate zone in which beads of pus have reached the surface along the columns of fat; surrounding this is a purple zone of subcutaneous pus, and lastly an area of inflammatory induration. Carbuncles are commonly seen on the back of the neck and are sometimes associated with diabetes, and for this reason the urine must always be examined. Incorrect diagnosis and imperfect treatment may lead to such an extensive spread of the infection that prolonged illness and death may follow. If the pathology is understood, the need for drastic treatment will be readily appreciated.

*Treatment.* — There are two methods available, the expectant and the operative, and many surgeons to-day practise the former. The patient is put to bed and the usual general eliminative treatment adopted. The carbuncle is treated with short-wave therapy twice daily, antiphlogistine poultices and sulphathiazole by mouth. When drainage is established a dressing of glycerin and magnesium sulphate is substituted.

If in spite of expectant treatment the carbuncle is extending its limits, and drainage is not sufficient, a crucial incision is made (Fig. 91). The ends of each incision **MUST** extend into normal skin. The four flaps thus formed are picked up with toothed forceps and are dissected off the underlying deep fascia; in this way all the infected "columnæ" are divided and the subcutaneous tissues completely drained. The necrotic debris is removed with a sharp spoon, and gauze soaked in eusol is lightly packed in under the flaps and a large dressing applied. The gauze is removed in forty-eight hours time and the wound dressed with paraffin and flavine. Rapid healing will be stimulated by infra-red radiation and skin grafting is rarely needed. If the patient is a diabetic, appropriate treatment will be directed to that condition.

**FACIAL CARBUNCLES.**—The danger area of the face is bounded by a line drawn from the angle of the mouth to the external orbital process and thence upwards to the hair line. Sepsis of any sort, but especially carbuncles, in this area is fraught with great danger owing to the possible spread of infection by the angular vein into the cavernous sinus. For this reason facial carbuncles are never incised but intensive palliative treatment is adopted. Ligature of the angular vein is quite futile.

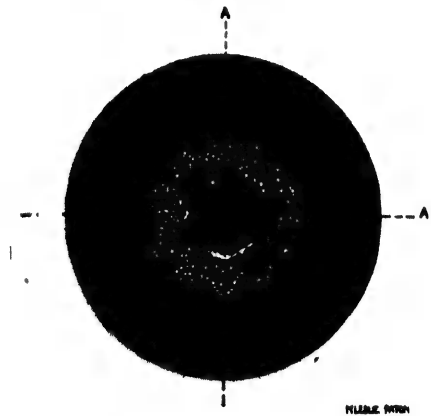


FIG. 91

Drawing showing the areas of a carbuncle.

A A dotted lines indicate the extent of the incisions.

Cellulitis and erysipelas have already been described (see Chap. III).

**Impetigo Contagiosa** is of streptococcal origin and is most commonly seen on the face in children. The cocci gain entrance through an abrasion and form intradermic vesicles filled with a serous exudate, which coagulates when the vesicles rupture, a yellow crust being formed. The exudation continues at the periphery and the lesion increases in size until contiguous patches meet and coalesce. The infection tends to spread rapidly over the whole face and down on to the neck.

*Treatment.*—The crusts must be gently removed by soaking them either in warm sweet oil, hydrogen peroxide (3 volumes) or lysol (0.5 per cent.), twice daily, after which the underlying lesions are covered with a mild mercury ointment, *e.g.*, Ung. Hydrarg. Nit. dil. or Ung.

Hydrarg. Ammon. dil. Great care must be taken to prevent the spread of the infection and in men shaving must be prohibited until the lesions are healed.



FIG. 92

A large gouty tophus of the index finger.

### SPECIFIC INFECTIONS

**Gouty Tophi.**—Deposits of sodium bi-urate are typical manifestations of chronic gout. They may occur in the subcutaneous tissues of the fingers and hand (Fig. 92) as well as in joints, bursæ and the cartilages of the ear.

**Tuberculous Infections of the Skin.** **BAZIN'S DISEASE**, or erythema induratum, affects girls and young women whose circulation is poor. The characteristic nodules appear on the postero-external aspect of the legs, but are also rarely met with in the upper limb and on

the face. The condition starts as a small nodule beneath the surface of the skin, which gradually becomes thickened and purple in colour. The nodules are firmly fixed in the skin, have ill-defined edges, but move freely over the underlying tissues. Their discoloration does not disappear on pressure. Some of the nodules break down exuding a thin serous discharge, and later form typical tuberculous ulcers, while others become spontaneously absorbed. The lesions are usually bilateral and are much aggravated by cold weather.

*Treatment* is disappointing, as relapses are so frequent. Rest in bed, intravenous injections of neosalvarsan and radiotherapy all improve the local condition, and curettage of ulcerated nodules will

accelerate convalescence. General treatment will be directed towards the underlying tuberculosis.

**TUBERCULOUS ULCERS** occur as a result of (1) breaking down of a Bazin's nodule ; (2) extension from a tuberculous bone, joint, tendon sheath or bursa ; (3) extension from a tuberculous gland ; (4) spread of infection at muco-cutaneous junctions ; and (5) infection of a skin wound by bacilli from the surface.

*Treatment* usually needs to be directed towards the underlying lesion, but, wherever possible, the ulcer should be excised and the wound sutured.

**LUPUS VULGARIS.**—The initial lesion is an intradermic nodule varying in size from a pin's head to a small pea. It is soft, semi-translucent and brownish yellow in colour, and on pressure with a glass slide the typical apple-jelly appearance is seen. A cluster of nodules coalesce to form a lupus patch, which spreads by extension at the periphery by the further formation of nodules. The older or central area tends to heal by scar tissue in which new lesions may occur. The infection reaches the skin by lymphatic spread from adjacent mucous membranes or more rarely may be blood-borne or implanted from the surface. It attacks both skin and mucous membranes and is usually found on the face around the nose and mouth, and on the neck, but it also occurs on the trunk and on the palm of the hand and sole of the foot. It affects children and adults and is a slowly progressive chronic condition which is of little danger to life, though locally destructive of tissue. A squamous-celled carcinoma is apt to arise in old lupus scars.

*Treatment* of a single patch consists in excision and suture or grafting if necessary. This is rarely practicable and the best results are obtained from heliotherapy combined with local exposure to X-rays, the Finsen quartz lamp or concentrated ultra-violet light. The lesions of the mucous membranes must also be treated if a complete and lasting cure is to be achieved. Patients should be kept under observation for many years, even after apparent cure.

**VERRUCA NECROGENICA** (Butcher's Wart or Anatomical Tubercle) is due to the direct implantation of tubercle bacilli into the skin, and so is usually seen on the fingers, hand and forearm. It begins as a papule which extends to form a warty granulating mass on an indurated base, surrounded by a red or purple zone of inflammation.

*Treatment* is excision.

**Syphilitic Diseases** of the skin are met with in all stages of the disease, and are described in Chap. V, pp. 67 and 69.

**Erythema Nodosum** is generally regarded as being of rheumatic origin, but it seldom occurs in typical cases of that disease. The lesions consist of raised indurated nodules, oval in shape and of considerable size, which are at first red and later purple in colour. They are seen on the legs, often over the subcutaneous surface of the tibia, and on the extensor aspect of the feet. The nodules are painful, tender and hot, and there may be a mild pyrexia. The condition may be mistaken for a deep abscess, cellulitis or even acute osteomyelitis of the tibia, but the distribution and clear demarcation of the nodules,

and the fact that they are often multiple and bilateral should point to the correct diagnosis.

*Treatment* consists in rest and local applications of hot dressings or evaporating lotions.

**Lupus Erythematosus** is characterised by oval or round patches of erythema, covered by lamellated scales, on the under surface of which are tiny horny plugs which project into the openings of the hair follicles. The lesions tend to heal in the centre, leaving a thin atrophic scar, while telangiectases are seen in the active areas. The scaly patches affect chiefly the malar regions of the face and the bridge of the nose, producing the so-called "butterfly erythema" from the shape of the lesion. The disease may cross the muco-cutaneous junctions of the lips and nares and attack the nasal and buccal mucous membranes. It affects both sexes between the ages of twenty and fifty years, is chronic and difficult to eradicate. It is believed, especially by the continental pathologists, to be associated with tuberculosis, but the evidence is inconclusive. The patches are slightly tender and give rise to a mild burning sensation.

*Treatment*.—No local applications seem to have any effect and radiotherapy is disappointing, but of recent years excellent results are reported from the use of sanocrysin, which is given in intravenous doses of 0.1 grm. weekly for twelve weeks.

**Erythema Pernio** (chilblain) is an erythematous condition of the fingers, toes, hands and feet, due to venous stasis occurring in young people in cold weather. Chilblains appear as raised purple swellings which are tender and irritable and whose surface is often broken by rubbing. They may be largely prevented by the use of thick stockings and gloves, and by active exercise. If there is any suspicion of a chilblain starting, it may be successfully prevented by the use of this paint night and morning :—

R

Tinct. iodi . . . . .	3i
Pulv. ac. tannic . . . . .	3ii
Collodion flex. . . . .	ad 3i
Mft. pigmentum.	Apply with camel's hair brush.

Calcium lactate with or without parathyroid extract is widely recommended but is most disappointing.

**Molluscum Contagiosum** is a condition of unknown etiology in which multiple small rounded nodules occur in the skin. They are raised, umbilicated and semitranslucent, and consist of solid columns of squamous epithelium, the central cells of which undergo hyaline degeneration, while others nearer the surface show an atypical keratinisation which leads to the formation of the so-called "molluscum bodies." The disease affects the skin of the face, neck, hands and scrotum.

*Treatment* consists in scraping or excision.

**Leprosy** has already been described (p. 41).

**Parasitic Diseases** of the skin have little interest to the surgeon and they will be found described in textbooks of medicine.

**Indelible Pencil Lesions** of the skin of the fingers is becoming more widely recognised in this country, though their incidence is higher in America. A small point of pencil lead containing an aniline dye having been implanted, its slow absorption leads to a chronic progressive necrotic process with either (1) a discharging sinus, (2) a small profusely granulating wound, or (3) a necrotic ulcer (Fig. 93). Treatment consists in radical excision.

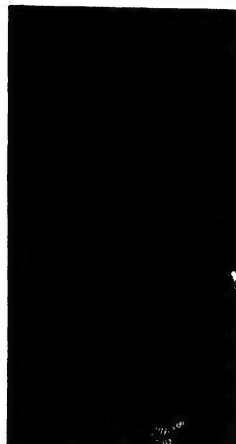


Fig. 93  
Indelible pencil lesion.

## GROWTHS AND CYSTS OF THE SKIN

**A Callosity** is an area of hypertrophy of the skin with marked thickening of the horny layer due to prolonged pressure. It is commonly seen in the palms of the hands of manual workers, the soles of the feet in town dwellers and on the heads, shoulders and backs of certain workmen, *e.g.*, Covent Garden, Smithfield and other porters of like occupation.

**A Corn** differs from a callosity in having a central hard core which grows downwards causing pressure atrophy of the papillæ of the corium. In this way a conical depression is formed around which the cuticle hypertrophies, and a hard avascular area projects from the surface. As a result the causative pressure is intensified and a vicious circle leads to a gradual increase in the size of the corn. They are very commonly seen on the toes, where they are due to the pressure of ill-fitting shoes. Two varieties are described—hard and soft corns.

**THE HARD CORN** is found on the outer surface of the little toe, the inner surface of the big toe and over the extensor surface of the proximal interphalangeal joints of the second, third and fourth toes (especially in conjunction with hammer toes). It forms a small raised conical swelling with a brown depressed centre and is exquisitely tender on pressure, unless carefully tended. Infection sometimes spreads in beneath the corn and suppuration occurs with swelling and great pain. This may lead to sloughing and spontaneous cure of the corn, but, on the other hand, the pus may track deeply and infect the subjacent bones or joints.

**THE SOFT CORN** occurs in the moist skin of the web between the toes and the hypertrophied epithelium becomes white and sodden.

*Treatment* consists in removing the source of pressure by obtaining properly fitting shoes. The corn can be kept painless by careful and regular paring away of the projecting hypertrophied skin without producing bleeding. An attempt, often unsuccessful, to destroy the conical core can be made by painting the area daily with the following :—

R	Ac. salicylic . . . . .	gr. lx
	Extr. cannabis indic. . . . .	gr. xxv
	Collodion flex. . . . .	ad ʒi

Or	R	Ac. salicyl . . . . .	gr. xxv
		Ung. resorcin (2 per cent.) . . . . .	ad ʒi

Soft corns respond most readily to resorcin ointment (2 per cent.). It is rarely advisable to excise corns as the scar tends to become thickened and tender, but intractable soft corns may be treated in this way. Large hard corns on the outer side of the little toe, which cannot be kept painless by paring, are most suitably dealt with by amputating the little toe at the metatarso-phalangeal joint.

**Papillomata** occur as two types—(a) warts and (b) true new growths. A **wart** is probably never truly neoplastic, but due to a virus infection. One or more may occur on the fingers and hands of young people, varying in size from a pin's head to an inch in

diameter and having a rough surface apparently composed of three or four horny centres. Multiple warts are also seen on the penis and vulva as large, soft and vascular masses in association with gonorrhœa (p. 63). Many paints and pastes are advised in the treatment of warts, but the simplest and most effective method of getting rid of them is by the use of carbon dioxide snow or diathermy.

**TRUE PAPILOMATA** are either pedunculated or sessile, their surface being either smooth, villous or nodular. Some of them are pigmented. The skin around them is normal and the lymphatic glands draining the area are not affected. These tumours may safely be left alone, provided the patient consents to regular supervision, but if they are subjected to pressure or friction, or if they

have shown recent signs of active growth, they should be excised, and this especially holds true of the pigmented type. Large tumours can be treated by X-rays, radium or diathermy, or by excision and skin grafting.

**Angiomata** occur as capillary and cavernous hæmangiomata, and are described on p. 286.

**Dermoid Cysts** are congenital in origin and occur only in certain situations, corresponding to lines of fusion of embryonic cutaneous surfaces. They are seen commonly in the midline and at the outer angle of the orbits, usually being clinically present at birth. They grow slowly and have a loose attachment to the surrounding structures but not to the skin itself. They should be excised.

**AN IMPLANTATION DERMOID** (Fig. 94) is traumatic in origin, a small



FIG. 94

An implantation dermoid cyst of the hand.



island of squamous cells being carried into the subcutaneous tissues by some blunt instrument, *e.g.*, the blunt end of a needle (into the fingers of seamstresses). The cells continue to grow, forming a cyst lined by squamous epithelium and secreting keratin. The fingers, hands and wrists are the areas usually affected. The cyst should be excised.

**Squamous-celled Carcinoma** of the skin (Fig. 95) may be seen in any part of the body and is frequently associated with prolonged chronic irritation, of which the following examples may be quoted : cancer of the scrotal skin in chimney sweeps, cancers in paraffin, tar and X-ray workers, and those developing in long-standing, imperfectly healed scars, in lupus patches, in chronic ulcers and sinuses, and in certain syphilitic lesions such as leukoplakia. Clinically the growth



FIG. 95

Squamous-celled carcinoma of the hand.

may first appear as a raised, indurated warty nodule which gradually spreads in the skin and into the underlying tissues (Fig. 96). Eventually the surface epithelium breaks down and a typical ulcer results, with its raised everted edges and its indurated base. In some cases the growth remains papillomatous in type with but slight surface ulceration, whilst others, *e.g.*, on the lips, develop into a densely fibrous ulcer with little or no overgrowth. The lymph glands ultimately become involved, being hard and fixed, but there is no marked enlargement unless a heavy secondary infection is present.



FIG. 96

Squamous-celled carcinoma of the skin of the lower eyelid.

*Treatment* consists in excision of the growth and its lymphatic field, with appropriate plastic or grafting methods to close the wound if necessary.

**Rodent Ulcer (Basal-celled Carcinoma)** occurs in both sexes after the age of 40 years, and the majority are seen on that part of the face above a line joining the angle of the mouth to the external auditory meatus, particularly upon the lateral surfaces of the nose, near the inner canthus of the eye and on the cheek below the lower lid (Figs. 97 and 98). It is thought to arise from either the basal layers of the epidermis, the sebaceous or sweat glands or the hair follicles, and it spreads by infiltration in the skin and into the underlying tissues. It appears first as a small, raised, hard nodule and many weeks or months may elapse before ulceration occurs. The ulcerating rodent is apt to be mistaken for a squamous-celled carcinoma, but its edges are not so markedly everted and they are surrounded by a narrow zone of raised and thickened skin which is infiltrated but not yet ulcerated by the growth ; and, in addition, areas of attempted repair may be

seen in the centre or in the edge of the ulcerating area. The slow progress of the lesion, its long history and the complete absence of



FIG. 97

Rodent ulcer just beneath the inner canthus.

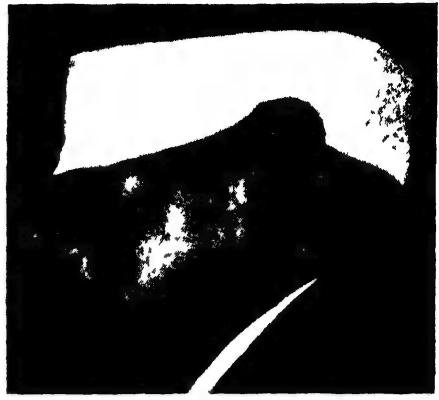


FIG. 98

Rodent ulcer on the malar region in front of the ear showing pre-ulcer stage.

lymph gland involvement distinguish it from the squamous-celled tumour. In its later clinical course a rodent ulcer tends to fall into one of two groups, the superficial and the penetrating. The superficial rodent is of very slow growth, spreads chiefly in the skin and subcutaneous tissues, and does not infiltrate the deeper structures; the penetrating type not only destroys the skin but slowly and inevitably erodes the underlying tissues, including bone, in such a manner that the most terrible deformities result in neglected cases before death occurs from intercurrent disease.



FIG. 99

Large melanoma in the middle of the back. Patient alive and well, married, with two children, after removal twelve years ago.

*Treatment.*—In spite of its slow growth a rodent tumour should be removed at the earliest opportunity, because eradication of the penetrating variety is difficult, and if incomplete recurrence of the growth is of more rapid and virulent a type. This characteristic led surgeons at the beginning of the modern surgical era to apply to rodent ulcers the label “*Noli me tangere.*” The recognition of the pre-ulcerative stage is therefore of the utmost importance. Removal

by complete excision performed by the high-frequency cutting current is the method of choice in every operable case, followed by suture or grafting. Radium has had excellent results in the early superficial types. For the inoperable penetrating ulcers X-rays and radium should be tried, but they both may result in an increase in the rate of growth of the deeper parts of the tumour. Eventually, nothing remains except the prevention of sepsis as far as possible.

**Melanoma.**—*A.* BENIGN MELANOMA, or Pigmented Mole, is one of the commonest congenital abnormalities of the skin, consisting of a small soft fleshy tumour which is either flat, slightly raised or



FIG. 100

Malignant melanoma on the side of the foot.

pedunculated, brown in colour and occasionally covered with hair. These tumours usually continue unchanged throughout life, but occasionally one may suddenly take on rapid growth and become malignant. The benign melanomata need no treatment, unless in an area constantly exposed to pressure or friction (*e.g.*, on the shoulders beneath the strap of the braces), or possibly for cosmetic reasons, when they should be excised.

*B.* THE MALIGNANT MELANOMA (Melanotic Sarcoma or Carcinoma) (Chap. VI, p. 110).—These tumours may arise in the choroid coat of the eye, in any part of the skin (Fig. 99), and even in the mucous membranes of squamous type, *e.g.*, the mouth. The dorsum and outer side of the instep of the foot (Fig. 100), and the skin around the toe-nails are the commonest situations. These growths spread both by lymphatics (Fig. 101) and blood stream, and are characterised by widespread dissemination in the nearest lymph-gland area and throughout the visceral and skeletal systems. The primary tumour may remain small, yet almost every organ may be affected and in these cases the urine will contain melanin.



FIG. 101

Mass of secondary melanotic glands from the groin of the patient seen in Fig. 100.

*Treatment* consists in local excision of the primary growth with its lymphatic field. Many cases, however, will be inoperable owing to blood stream metastases.

## THE SEBACEOUS GLANDS

**Sebaceous Cysts** result from blockage of the duct of a sebaceous gland by dirt or inflammation, and are commonly seen (Fig. 102). on the scalp, face, neck, back and scrotum. They are frequently multiple, especially on the scalp (multiple wens), and may reach a large size. The cysts are attached to the skin and the orifice of the duct can usually be identified. They may undergo the following complications :—



FIG. 102

Sebaceous cyst behind the ear.

1. They may become infected, in which case they form a localised encysted abscess cavity; should they grow to a very large size, ulceration of the skin due to pressure atrophy may follow.
2. They may rupture subcutaneously or externally.
3. They may form a "sebaceous horn" from slow protrusion of the contents which become inspissated and finally keratinised.
4. They may become malignant, being then known as "Cock's Peculiar Tumour."

*Treatment.*—The cysts must be removed completely or they will recur. If the orifice of the duct is included in a small elliptical incision, they are easily dissected out. Infected cysts should be opened and curetted with a sharp spoon and allowed to granulate from the bottom.

**Adenomata of the Sebaceous and Sweat Glands** occur in the scalp as slowly growing firm reddish tumours which may ulcerate. They should be excised. Another cystic adenoma rarely found in the scalp is known as the Epithelioma Adenoides (cysticum).

**Rhinophyma** is a hypertrophic condition of the skin of the nose in elderly men due to blocking of the sebaceous ducts. This is followed by a low-grade infection with fibrosis and thickening of the skin. A large bulbous and pendulous mass results (Figs. 103 and 104), covered by purple skin with the typical *peau d'orange* appearance.

*Treatment* consists in carefully removing slices in the hypertrophied skin until the nose has regained its normal shape, after which the raw area is rapidly covered over by newly formed epithelium from the cut ends of the ducts.



FIG. 103

Rhinophyma. A man known to many generations of students at St Mary's Hospital.



FIG. 104

The rhinophyma after removal from the patient seen in Fig. 103.

## VASCULAR AND NEUROPATHIC AFFECTIONS OF THE SKIN

**Erythema ab Igne** is a condition of blotchy pigmentation of the skin in front of the legs due to prolonged exposure to heat, and has become more common since the more general use of gas fires. No treatment is possible.

**Trophic Ulcers** occur in the skin owing to malnutrition and defective sensation in lesions of the central nervous system (lower motor neurone type), *e.g.*, transverse myelitis, tabes dorsalis, syringomyelia and infantile paralysis, and in injuries and diseases of the peripheral nerves. They may follow slight trauma, *e.g.*, abrasions or bruises, as a result of which the surface epithelium is shed and a shallow ulcer with irregular edges and anæmic sluggish granulations develops. It usually proves resistant to treatment, but stimulating lotions and the infra-red lamp should bring about healing, provided the ulcer is carefully protected.

**Perforating Ulcers** are also of trophic origin, occurring most commonly in tabes dorsalis on the soles of the feet and on the borders of the great and little toes. The condition usually starts with a hæmorrhagic effusion beneath a callosity under the ball of the big toe. The thickened skin is shed and an indolent ulcer is formed which penetrates into the underlying tissues and finally erodes the metatarsophalangeal joint and bones. A perforating ulcer has a long narrow sinus with no sign of repair, white sodden epithelium is heaped up around the opening, and there is a slight discharge. The condition is *painless* throughout.

*Treatment* should be directed to the underlying cause; local scrapings or applications are useless and an amputation may be necessary.

**A Bed-sore** is a particular type of trophic ulcer occurring in

paralysed or bed-ridden patients in areas subjected to continual pressure; *e.g.*, the sacral region of the back. An ulcer forms very rapidly and may penetrate to the bone; it is clean, punched out, purple in colour, devoid of granulation tissue and continues to progress as long as the pressure continues.

*Treatment* is essentially preventive, but even the most careful nursing will not always succeed in avoiding it. However, the use of air rings, air or water beds, careful and assiduous inunctions and massage should prevent them. When they do occur, all pressure must be removed, absolute asepsis maintained and the cavity treated with stimulating lotions, Bier's suction glasses and the infra-red lamp. They are best dressed with firm elastoplast strapping.

### AFFECTIONS OF THE NAILS

The growth and appearance of the nails depends upon their beds being normal and healthy. A nail that becomes separated from its bed will die and be cast off, but if the bed is healthy a new nail will grow to perfection. Injuries to the fingers sometimes result in hæmorrhage beneath the nail, which is subsequently lost. In these cases the nail should be maintained in position as long as possible in order to protect the bed and prevent pain and deformity of the new nail.

Infections of the nail and nail-bed (paronychia) will be dealt with in Chap. XIV.

**Onychogryphosis** is a condition of overgrowth of the nail of the big toe due to chronic infection following lack of care and cleanliness. The nail grows forward, becoming thickened, ridged and curved, and eventually comes to resemble a ram's horn.

*Treatment* consists in removal of the nail and complete destruction of the nail-bed and root.

**Ingrowing Toe-nail** also occurs commonly in the big toe and is due to two factors, pressure from tight shoes and careless cutting of the nail. Toe nails should be cut with a straight and not a curved edge, and they should project beyond the margin of the cuticle. Ingrowing toe-nails develop because the sharp edge is forced into the cuticle and the resulting ulcer forms abundant granulation tissue which overlaps the nail.

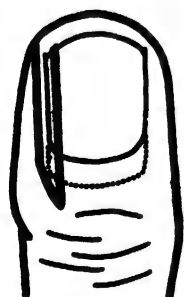


FIG. 105.

Incisions for the removal of an ingrowing toe-nail.

*Treatment* in the early stages consists in trimming the nail, curetting the granulations, iodine baths and hot fomentations for forty-eight hours. In the later stages, Watson Cheyne's operation gives the best results. A wedge of tissue is removed consisting of a strip of nail, its underlying nail-bed and the contiguous skin-fold. The skin flap is sutured alongside the cut edge of the nail and no raw area is left. It is rarely, if ever, necessary to remove the whole nail (Fig. 105).

R. M. HANDFIELD-JONES.

## CHAPTER XIV

### INFECTIONS OF THE FINGERS AND HAND

**A** *NATOMY*.—The **distal segment** of a finger consists of the distal phalanx, the attachments of the flexor profundus and the extensor tendons and the fibro-fatty pulp in front of the phalanx, all of which are contained within the skin, part of this being of specialised development forming the nail and its bed. A transverse section of the distal segment reveals the presence of a number of fibrous septa (usually fourteen) which pass from the periosteum to the skin and which extend the whole length of the segment. The end of the finger is thereby divided into a number of compartments which are filled with fat and in which the sweat glands lie. A longitudinal section shows that the fibro-fatty pulp extends to the level of the insertion of the flexor tendon, and that the fibrous septa are attached to the deep fascia which covers the tendon. The pulp is therefore a closed sac. The branch of the digital artery which supplies the epiphyseal end of the distal phalanx is given off in the middle segment of the finger and reaches the epiphysis without entering the pulp. This explains the immunity of this part of the bone, which is able to re-form the shaft after necrosis. The dense lymphatic plexus in the space communicates freely with the vessels in the periosteum, to which infection may be carried in the early stages of a septic finger. The nail-bed is a specialised derivative of the skin, which forms and nourishes the nail. The exposed part of the nail comprises three-quarters of its extent, the remainder being concealed beneath the skin.

The **tendon sheaths** of the index, middle and ring fingers have no connection with the synovial bursæ beneath the anterior annular ligament. They extend from the level of the distal interphalangeal joint to a point in the palm one finger's breadth proximal to the web between the fingers. The deep fascia surrounds the sheaths and is attached to the sides of the phalanges, in this way forming a fibro-osseous tunnel lined with synovial membrane. This maintains the tendons in contact with the phalanges during movements of the fingers. The sheath of the little finger has a similar arrangement in its digital section, but in the palm it is continuous with the ulnar bursa, which spreads laterally to form a sac reaching to the base of the middle metacarpal and extends under the annular ligament on to the anterior surface of the pronator quadratus in the forearm. The tendons of the index, middle and ring fingers enter this bursa just distal to the annular ligament, so that the deep and superficial flexor tendons of all the fingers are contained in the ulnar bursa at the wrist. The sheath of flexor longus pollicis begins at the level of the interphalangeal joint and extends upwards through the muscles of the thenar eminence. It is continued under the annular ligament to reach the anterior surface of the pronator quadratus. At the wrist it is called the radial bursa, but actually it is simply one continuous tendon sheath. There is not supposed to be any communication between the two bursæ, but it is present in so many hands that it may be considered a normal variation. There is also a wide variation

in the anatomy of the sheaths, *e.g.*, the ulnar and radial bursæ may have no connection with the sheaths of the little finger and thumb, and the ulnar bursa may consist of two, three or four separate tubes beneath the anterior annular ligament. The commonly accepted arrangement is shown superimposed on an X-ray photograph which illustrates their extent (Fig. 110).

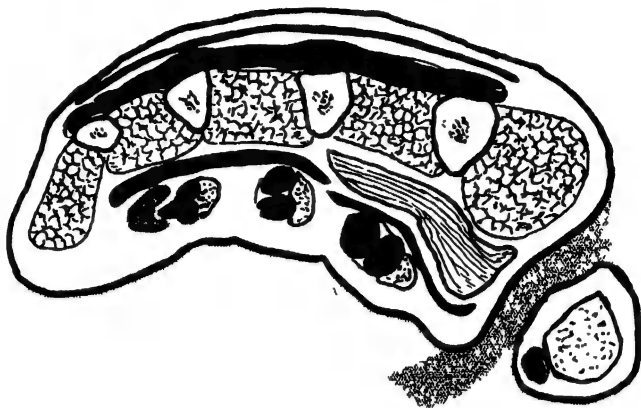


FIG. 106

Transverse section of hand showing in the palm middle palmar space in red, thenar space in blue, synovial sheaths in green; on the dorsum subaponeurotic space in red, subcutaneous space in blue.

The **fascial spaces** of surgical importance are the Middle Palmar and the Thenar spaces. Their extent and relations are as follows (Fig. 106):—

#### A. THE MIDDLE PALMAR SPACE.

Anteriorly, it is covered by skin, palmar fascia, superficial palmar arch, the flexor tendons of the little, ring and middle fingers, and a fibrous sheet immediately behind the tendons.

Posteriorly, a fibrous sheet separates it from the deep palmar arch and the interossei muscles.

Ulnar side, a fibrous membrane separates it from the hypothenar muscles.

Radial side, a strong fibrous septum, which is attached to the median ridge on the shaft of the middle metacarpal, divides it from the thenar space.

Distally, it extends to the level of the distal flexion crease in the palm, but sends three prolongations along the canals of the little, ring and middle lumbricals.

Proximally, it extends to the level of the carpo-metacarpal joints and sends a fine prolongation upwards behind the tendons to reach the pronator quadratus.

#### B. THE THENAR SPACE.

Anteriorly, it is covered by skin, deep fascia, superficial thenar muscles, flexor tendons of the thumb and index finger and occasionally of the middle finger.

Posteriorly, the fascia covering the adductor transversus pollicis muscle.

Ulnar side, the fibrous septum between it and the middle palmar space.

Radial side, it extends to the metacarpal of the thumb.

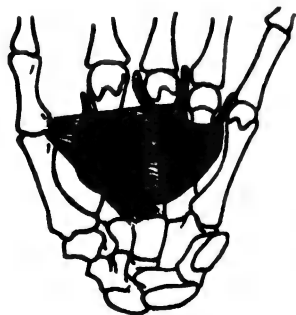


FIG. 107

Diagram showing the extent of the middle palmar and the thenar spaces. The extensions along the lumbrical canals will be noted.



Distally, it reaches the level of the radial end of the middle flexion crease, with an extension along the index lumbrical canal.

Proximally, it reaches the level of the carpo-metacarpal joint. The spaces are separated from each other by the firm septum already alluded to, but at its proximal extremity this septum becomes so thin that pus can track from one space to the other, although anatomically there is no communication between them. Their extent is shown in Fig. 107.

The **lymphatics** of the hand are divided into a deep and a superficial group. The deep group run with the veins and is of less surgical importance than the superficial. The skin and subcutaneous tissues of the palmar surface of the palm, fingers and thumb are supplied with a dense network of lymphatic capillaries. The dorsal aspect of the distal and middle compartments of the fingers also have this dense plexus, but the dorsum of the hand and proximal segments of the fingers are less well drained. At the wrist this capillary plexus is resolved into a series of lymph vessels which pass up the forearm and arm. In the middle of the forearm between twenty-four and thirty can be demonstrated running parallel to each other. The majority pass direct to the axilla, but those which drain the little and ring fingers usually enter the epitrochlear gland in front of the internal condyle of the humerus.

### THE PROPHYLACTIC TREATMENT OF HAND INFECTIONS

Injuries to the fingers and hand are frequent in industrial workers, but permanent disability is due to the infection which follows and not to the injury, as the following figures show :—

1. Seventy-five per cent. of disabilities following hand injuries are due to infection.
2. Seventy-five per cent. of severe deformities of the hand are due to infection.
3. Between 7 and 9 per cent. of all cases with a total incapacity claim are due to hand infections.
4. Sixty-five per cent. of all hand injuries which are awarded compensation are due to infection following trivial trauma.

These figures prove that the importance of prophylactic treatment cannot be over-estimated, for apart from permanent incapacity, the number of working hours wasted by those in whom a complete cure is obtained represents a serious financial loss to both employer and employee. The serious significance of trivial injury is emphasised by these statistics. Methods of prevention therefore fall into two groups, viz., the abolition as far as possible of all causes of minor injury and the prompt sterilisation of such injuries as do occur. The first is the concern of the masters and men, but also provides a problem for the medical worker in the field of industrial medicine. The treatment of the minor scratch, cut or prick is a simple matter. The facilities that are provided by industrial firms for first-aid treatment will depend on the size and efficiency of the works ; and these will not be fully utilised unless the men are taught to present themselves for all injuries, however slight. The medical profession faces its own peculiar dangers in post-mortem rooms and operating theatres. The

steps to be taken in all these cases are as follows: (1) Cessation of work; (2) encouragement of bleeding; (3) cleansing of the wound; (4) sterile dressing; and (5) immobilisation of the arm.

1. **Cessation of Work** may appear unnecessary in slight injury, but the loss of part of a day's work is preferable to the weeks spent in recovering from an infection of the hand.

2. **Encouragement of Bleeding.**—The immediate concern of the subject of the injury and of the onlookers is to stop the bleeding. This instinct must not be followed. Bleeding—especially the slow welling-up from the depths of a wound—will wash out infecting organisms more adequately than anything else. If the bleeding has stopped, the wound should be encouraged to bleed by holding it under a stream of hot water or by bandaging the arm so that venous congestion is obtained, and the bleeding allowed to continue for two minutes.

3. **Cleansing of the Wound.**—If the hand is clean at the time of injury it should be thoroughly washed; but if it is very dirty, as it must necessarily be in the case of many manual occupations, more harm than good is done by washing. The injured area is placed in a bath of an alcoholic tincture of iodine for five minutes, the edges of the wound being separated to allow free access to iodine.

4. **A Sterile Dressing** is applied, and great care is taken to ensure that the bandage is not so tight as to impede the flow of blood.

5. **Immobilisation of the Arm** in a sling till bedtime and a long night's rest complete this prophylactic technique. If the least swelling, throbbing or pain occur at the site of injury a surgical opinion should be obtained without delay.

## NOMENCLATURE OF INFECTIONS IN THE HAND

The term "whitlow" will be entirely discarded, because it has been used to cover a number of conditions of varying complexity and severity without reference to their anatomical site or pathological significance. A subcuticular whitlow is merely a purulent blister on any part of the hand; a subcutaneous whitlow is an infection in the subcutaneous tissues of the hand; a thecal whitlow is an acute tenosynovitis, and a subperiosteal whitlow is an osteomyelitis of the phalanx. The term "whitlow" too often conveys the impression of a trivial infection, and no infection in the hand can be considered trivial, leading, as it may, to death or a useless hand. The poor standard of treatment of these infections is due to a lack of exact appreciation of their anatomy and pathology, and to this the use of the term "whitlow" has definitely contributed.

## INFECTIONS OF THE DISTAL SEGMENT OF THE FINGERS

**Distal Pulp Infections.**—These usually follow the most trivial injuries, such as pricks by a pin, needle, wood splinter or rose thorn, or by needles or spicules of bone in surgical operations and post-mortem examinations; but in a certain number of cases the infecting

organisms gain entrance through the ducts of the sweat glands without injury. The infection is usually staphylococcal in origin and starts in the fat of one of the compartments of the distal segment. Owing to the peculiar anatomy of the closed pocket a rapid rise in tension occurs, and if this is not relieved the soft tissues of the finger pulp become necrotic and the infection spreads through all the compartments and, further, the periosteum is involved and an osteomyelitis of the phalanx results. Delayed treatment may also lead to acute tenosynovitis and fascial space abscesses in the palm. Symptoms arise within a few hours of the injury, the patient becoming aware of a soreness or pricking sensation in the end of the finger. This rapidly progresses to a severe throbbing pain, which prevents either sleep or rest. In the early stages the site of injury becomes swollen and tender, and within twelve to twenty-four hours the whole of the distal compartment is tensely swollen and very tender. Later this tenseness gives place to induration and eventually an area of fluctuation appears. The middle and proximal segments and even the dorsum of the hand may become swollen, but there is no tenderness in these areas. The temperature is raised to 100° or 101° F. and the patient is tired from pain and lack of sleep, but there are no signs of constitutional involvement.

The only error in *diagnosis* is provided by acute lymphangitis, but in this condition there is tenderness throughout the swollen area, red lines of lymphangitis are seen in the forearm, and there is never the same induration in the distal pulp.

*Treatment* consists in early incision. If this is withheld till fluctuation is evident, the patient has been compelled to suffer needless pain and to run the risk of necrosis of the phalanx or of the more serious complications such as tenosynovitis or space abscesses in the palm. In the early stages, when the swelling and tenderness are localised, an incision over the point of maximum tenderness will suffice, but it is unusual for the patient to come for advice until the whole distal pulp is involved. The anatomical arrangement of the closed pocket is such that a median incision on the flexor surface can open one compartment only. This leads to inadequate drainage and leaves a scar on the tactile part of the finger. A lateral incision opens the pocket more satisfactorily, and if one is made on each lateral surface through-and-through drainage is obtained. In advanced cases the pocket should be laid open by uniting the lateral incisions by a cut over the tip of the finger (Fig. 108).

**Paronychia** is a staphylococcal infection around the edge of the nail, arising from an infected "hangnail." At first there is a red, swollen and tender area localised to one side of the cuticle, but many patients allow this to spread until there is a raised red and puffy collar around the nail. There is a general lack of appreciation of the pathology of this condition. The pus is not between the skin and

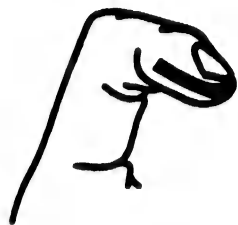


FIG. 108

The incision for distal pulp infection. In severe cases this will be extended right round the finger tip.

the nail-root but between the nail-root and the nail-bed. If the acute stage is inadequately drained a persistently recurring chronic paronychia results.

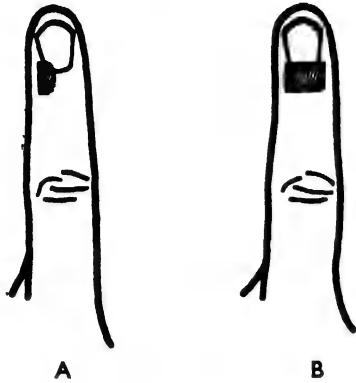


FIG. 109

Incisions for paronychia.

A is for early localised types, and B the more usually employed method.

*Treatment* in the early stages consists in an incision in the skin at the site of the infection to expose the nail-root on that side and a small segment is removed (Fig. 109, A). When the infection has spread around the nail, two parallel cuts are made so that a flap of skin can be dissected off the nail-root (Fig. 109, B). One blade of a pair of fine-pointed scissors is pushed beneath the nail, which is so divided that the nail-root is lifted off its bed and removed, while the exposed nail remains firmly in position on its matrix. A strip of rubber tissue is laid across the infected area and the flap replaced. The drain

is removed in thirty-six hours and the incisions heal rapidly. As the new nail grows a narrow slot of matrix is exposed, and in this way the long and tedious convalescence after a complete nail removal is avoided.

### ACUTE SUPPURATIVE TENOSYNOVITIS

The tendon sheaths in the fingers and hand (Fig. 110) become infected in two ways. They may be opened in severe crushes or lacerations and infected by direct implantation, or they are secondarily involved in conditions such as distal pulp infections or acute lymphangitis, in each of which the organisms are carried to the sheath by the lymphatics. The synovial membrane becomes acutely inflamed and the sheath is distended with pus. If the tension is not relieved by incision the pus ruptures into the fascial spaces of the palm or into the deep planes of the forearm, the synovial membrane being destroyed and the vitality of the tendon imperilled. As a result the function of the hand is seriously diminished either by the loss of the tendon or by its adhesion to the sheath. In many cases the infection is streptococcal in origin, and during dressings of the wound it is important to avoid introducing staphylococci as well.

The *symptoms* and *signs* are :

1. Throbbing pain in the affected area.
2. Symmetrical enlargement of the whole finger.



FIG. 110

A drawing illustrating the arrangements of the tendon sheaths in the fingers and hand. The sheaths are shown superimposed on the shadows of the bones.

3. Exquisite tenderness over the course of the sheath.
4. Great pain on full extension of the finger.
5. Moderate flexion at all joints.

The clinical picture will include the primary infection to which the tenosynovitis is secondary, the onset being marked by a spread of the local signs and a marked deterioration in the general condition of the patient. The swelling is not confined to the affected finger, but spreads on to the dorsum of the hand and into the proximal segments of the neighbouring fingers, and in neglected cases it will also affect the forearm. There is some tenderness over all the swollen area, but along the course of the sheath even gentle pressure cannot be tolerated. The finger at rest is held in semiflexion at all joints, and if an attempt is made slowly and gently to straighten it the patient submits with some anxiety until full extension is approached, and then the pain is so great that no further manipulation is permitted. This picture applies to all five digits, but in the thumb and little finger the infection will probably extend into the bursæ. In this event the area of exquisite tenderness corresponds to the surface marking of the bursa concerned, and the hand becomes greatly swollen, especially on the dorsum. A raised temperature, rapid pulse rate and rigors indicate the severity of the infection.

*Treatment of the Index, Middle and Ring Finger Sheaths.*—An incision is made along the lateral border of the finger—never in the middle line—opposite the middle or the proximal phalanx. The choice depends on the situation of the primary lesion and the presence of secondary complications, *e.g.*, infection in the lumbrical canals. At first the sheath is opened in the chosen segment, and pressure over the unexposed portions will then afford an indication of the severity of the infection. Except in very early cases it will be necessary to extend the skin incision over the previously untouched proximal or middle phalanx and to open the sheath opposite it, but the segment over the joint must be left intact. If the palmar extension is full of pus, the incision is prolonged for  $\frac{3}{4}$  in. into the palm (Fig. 111).

A similar procedure is adopted in the *little finger*, and when the sheath is opened pressure on the ulnar bursa indicates the extent of its invasion. If drainage is necessary, a director is passed into the bursa from the finger incision and its groove turned forwards and towards the ulnar border of the palm. The scalpel is then pushed boldly along the groove as far as the anterior annular ligament and the bursa laid open without injury to other structures. The sheath of the *flexor longus pollicis* provides a different problem in that it must be exposed by careful dissection to preserve intact the thenar muscles,



FIG. 111

A composite diagram showing the incisions for tenosynovitis in the hand. The full details are given in the text.

and the incision must stop one thumb's-breadth from the lower border of the anterior annular ligament. This ensures the safety of the branch of the median nerve which supplies the thenar muscles, and if the long flexor tendon is lost the short flexor remains. The skin incision follows the palmar margin of the short flexor muscle (Fig. 111).

The best position for the fingers after these incisions is semi-flexion at all joints, but if the sheath has been opened in its whole length this position may encourage prolapse of the tendons, and this must be prevented by keeping the fingers extended for the first seven days.

### ABSCESSSES IN THE PALM

The **Collar-stud Abscess** is situated in the distal part of the palm at the base of the fingers. The skin is greatly hypertrophied in this area in all manual workers, and in the professional or leisured

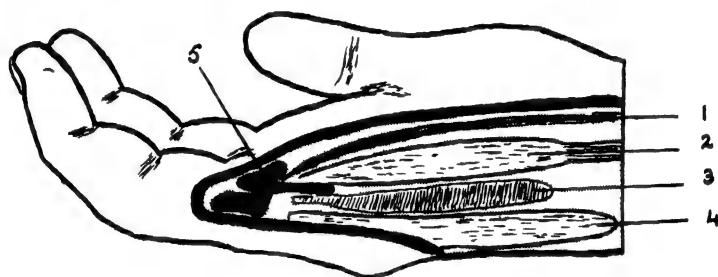


FIG. 112

Section of the hand showing "collar-stud" abscess: (1) palmar fascia; (2) flexor tendons with lumbrical; (3) middle palmar space; (4) interosseous muscle; (5) abscess.

classes unaccustomed rough work will raise blisters, which will be followed by thickening of the skin. If a staphylococcal infection is introduced beneath this thickened skin, pus cannot penetrate to the surface with ease and tracks through the digital divisions of the palmar fascia to reach the web between the fingers. A bilocular abscess is thus formed, one loculus lying beneath the skin and the other deep to the palmar fascia (Fig. 112). This "collar-stud" abscess is a minor infection but becomes important because the pus can so readily involve the fascial spaces in the palm and the tendon sheaths.

*Clinically* there is a painful swelling in the distal part of the palm around a callosity at the base of the adjacent fingers, and this spreads on to the dorsum of the hand. Tenderness is localised to the swollen area in the palm.

*Treatment* consists in a transverse incision over the swelling half-way between the skin of the web and the distal palmar crease. After the pus from the superficial pocket has been evacuated, careful search is made for the opening into the deeper pocket, which is adequately exposed and drained. The danger lies in the belief that the first gush of pus justifies no further exploration, and this results in the retention of pus in the deep pocket and its spread into more important structures in the hand.

**Fascial Space Abscesses.**—Infection reaches the middle palmar and the thenar spaces from many sources, and in difficult cases its origin may provide the clue upon which the final diagnosis is based. The routes of infection are tabulated below :—

Middle Palmar Space.	Thenar Space.
<ol style="list-style-type: none"> <li>1. Pulp infections of little, ring and middle fingers.</li> <li>2. Little-finger tendon sheath.</li> <li>3. Ring-finger tendon sheath.</li> <li>4. Middle-finger tendon sheath (usually).</li> <li>5. Lumbrical canals of little, ring and middle fingers.</li> <li>6. Ulnar bursa.</li> <li>7. Lymphatic spread.</li> <li>8. Direct puncture.</li> <li>9. Late spread from thenar space.</li> <li>10. Osteomyelitis of little, ring and middle metacarpal.</li> </ol>	<ol style="list-style-type: none"> <li>1. Pulp infections of index finger and thumb.</li> <li>2. Index-finger tendon sheath.</li> <li>3. Tendon sheath of thumb.</li> <li>4. Middle-finger tendon sheath (occasionally).</li> <li>5. Lumbrical canal of index finger.</li> <li>6. Radial bursa.</li> <li>7. Lymphatic spread.</li> <li>8. Direct puncture.</li> <li>9. Late spread from middle palmar space.</li> <li>10. Osteomyelitis of metacarpals of index and thumb.</li> </ol>

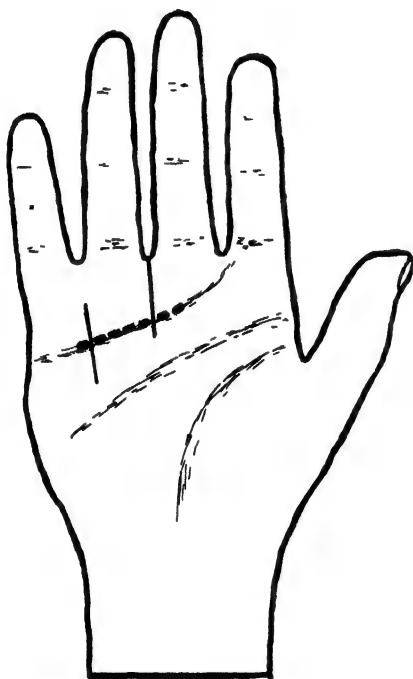


FIG. 113

Incisions for middle palmar space abscess. Two vertical ones as recommended by Kanavel; transverse in distal crease as advised by the author.

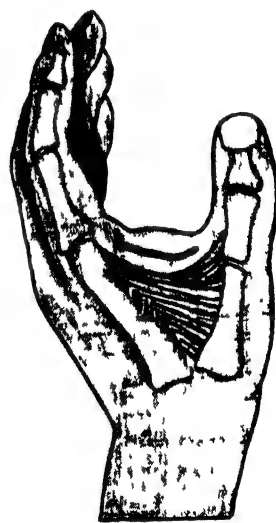


FIG. 114

Incision for thenar space. It should run parallel to and immediately below the lower margin of the first dorsal interosseous muscle.

*Symptoms and Signs.*—This table shows that space abscesses are secondary to infection elsewhere in the hand, and they appear as complications of the primary condition. It is probable that one or

more incisions have already been made and that the local and general conditions have steadily got worse. The diagnosis is not easy, as the whole hand is swollen, more particularly on the dorsum. The temperature ranges between  $100^{\circ}$  and  $104^{\circ}$  F., and the patient is exhausted by pain and lack of sleep. The cardinal signs are: (1) The type of swelling, and (2) the area of tenderness. In middle palmar space abscess the dense palmar fascia prevents any considerable swelling in the palm, but nevertheless it is sufficient to convert the normal concavity of the palm into a slight convexity, and further, the swelling is extremely tense. In thenar-space abscesses the thin fascia yields readily and the ballooning of the thenar eminence is characteristic. There is tenderness over the whole hand, but it is severe over the exact area of the surface marking of each space.

*Treatment of the Middle Palmar Space Abscess.*—An incision is made in the distal palmar crease and the flexor tendons of the ring finger identified. Behind these is introduced a blunt artery forceps which, on being opened, afford drainage to the pus. A wick of rubber tissue completes the operation (Fig. 113).

The *thenar space* is best approached from the dorsum. The incision is shown in Fig. 114. It follows the lower border of the first dorsal interosseus muscle. The lower margin of the adductor transversus pollicis is identified and a blunt-pointed pair of forceps is passed around this muscle. Drainage is established by a wick of rubber tissue.

### THE SPREAD OF INFECTION INTO THE FOREARM

The forearm becomes involved in lymphangitis and in those septic hands in which treatment has been inadequate or too long delayed.

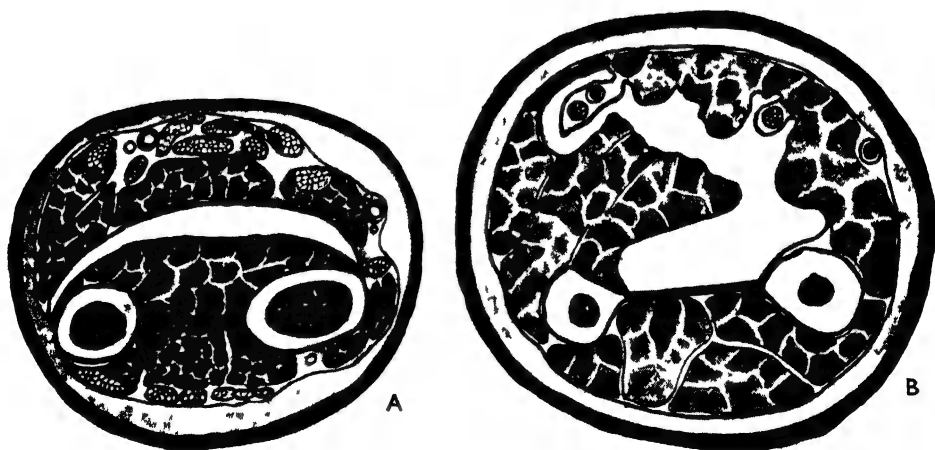


FIG. 115  
Cross-section of forearm showing forearm space.  
A, 3 in. above wrist. B, Mid-forearm.

Pus tracks from the hand, either by rupturing out of the upper limits of the ulnar and radial bursæ above the wrist or by following the upward prolongation of the middle palmar space. These routes lead to a similar space between the flexor profundus tendons and the



pronator quadratus (Fig. 115). The lower part of the forearm becomes swollen, tense and tender. Access to this space is obtained by an incision along the inner surface of the forearm opposite the anterior surface of the ulna. It starts 1 in. above the styloid process and extends for 3 in. upwards (Fig. 116). The attachment of the deep fascia to the

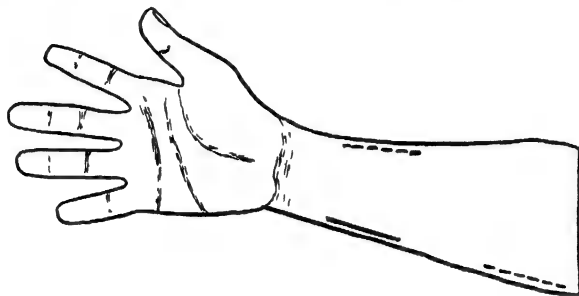


FIG. 116

Incisions for deep forearm space. Continuous line shows usual one, interrupted lines those for advanced cases.

bone is incised and a finger introduced between the tendons and the pronator quadratus. If necessary a similar incision on the radial aspect can be made to provide more free drainage.

### ACUTE LYMPHANGITIS

**Acute Lymphangitis** is almost invariably streptococcal in origin, the organisms being introduced by a trivial prick, usually in the distal segment of the finger. The condition is essentially an infection of the lymphatic vessels, and in the early stages no extra-lymphatic inflammation occurs and no pus is formed. The lymphatic plexus being infected, the process spreads rapidly through the normal lymphatic capillaries, enters the lymph vessels of the forearm and finally reaches the epitrochlear and axillary glands. Locally a diffuse red swelling results, and within a few hours "red lines" can be seen running up the forearm. These are the infected lymph vessels, to which the process is for a time confined. Later the infection spreads into and outside the lymphatic capillaries and the red lines become wider and finally fuse in a broad, red and swollen area. Soon the whole hand is swollen, the skin is red, tense and shiny and small vesicles appear, especially upon the dorsum. If the condition does not resolve, the brawny swelling spreads up the forearm and arm, the vesicles fuse to form large blisters, the colour of the skin changes to a dark blotchy purple and abscesses begin to develop locally in various parts of the hand, or the tendon sheaths become involved. The more virulent type of lymphangitis leads to a general septicæmia and death within a few hours. Kanavel classifies this condition as follows:—

1. Acute lymphangitis without local complications. The swelling is localised to the finger and hand, there are the red lines in the forearm and the temperature is about 102° to 103° F. The condition subsides as rapidly as it arose.

2. Acute lymphangitis with local complications. In this group a small abscess develops at the site of infection after the lymphangitis has subsided.
3. Acute lymphangitis with septicæmia and associated with severe secondary lesions, *e.g.*, space abscesses or tenosynovitis.
4. Acute fulminating lymphangitis with a virulent septicæmia ending fatally within forty-eight to seventy-two hours.

The clinical picture in the first few hours is of great importance because the original site of infection is frequently the end of the finger and the condition may be mistaken for a distal pulp abscess. The onset in both is similar in that a trivial prick is followed after a few hours by discomfort in the finger-end. In lymphangitis the swelling of the pulp is slight and generalised, and it spreads up the finger and on to the dorsum of the hand. This swelling in the distal compartment never becomes so tense as in a pulp abscess, and the tenderness is far less severe but is present over all the swollen area. Red lines will be seen in the forearm, and the high temperature and possibly rigors suggest the presence of a lesion more serious than a distal pulp abscess. The differential diagnosis between the two conditions is of paramount importance, for the pulp abscess demands early incision and drainage, whereas in acute lymphangitis an incision may convert a mild case into a fatal one. They are contrasted in tabular form below :

#### DIFFERENTIAL DIAGNOSIS

—	Distal Pulp Abscess.	Acute Lymphangitis in Finger-end.
Cause . . . .	Slight injury—prick, etc.	Slight injury—prick, etc.
Earliest sign . .	Tense discomfort with throbbing.	Discomfort ; no throbbing.
Swelling—		
Early . . . .	Tense—swelling moderate.	Swelling slight ; not tense.
Late . . . .	Marked ; red, tense, and shiny.	Moderate ; not tense or shiny.
Spread . . . .	Up finger and on to hand.	Up finger and on to hand unexpectedly marked.
Tenderness . . .	Over infected pulp only ; marked.	Over the whole swollen area ; moderate only.
Red lines . . . .	Absent.	Present.
Constitutional signs .	Mild.	Marked.

*Treatment* is local and general. The patient is kept in bed and a Bier's bandage is placed around the arm just below the axilla, and may be safely left for six hours and re-applied after one hour's interval. The upper extremity from the tips of the fingers to the upper part of the arm is swathed in a linseed poultice and splinted with large quantities of wool. This is renewed every three hours. It cannot be too strongly emphasised that an incision can do nothing but harm

by opening up unaffected lymphatics and allowing infected lymph to enter them, by which means the area of absorption is greatly increased. General treatment is directed towards elimination of toxin and the reinforcement of the patient's natural defences. Absolute rest and full nursing attention must be enforced and free diuresis and copious evacuations from the bowels obtained. The patient's strength is maintained by highly nutritious and easily assimilable food.

The cases which progress to severe constitutional involvement become very gravely ill, and some of these patients will die within forty-eight or seventy-two hours without responding to anything that is done for them. Others succeed eventually in overcoming the



FIG. 117

The author's special radiant-heat box for the treatment of infected fingers. (Weiss.)

septicæmia after a long illness, in which local complications in the hand and forearm arise and need surgical treatment.

Sulphapyridine has been proved to possess the power of destroying hæmolytic streptococci. This drug marks a very great advance in the treatment of severe streptococcal infections, of which acute lymphangitis is one of the most dangerous examples. A blood transfusion also is of the utmost value and a suitable donor should be kept on call. One of the gravest problems is the management of the restlessness and delirium which characterise septicæmia. Quiet and sleep must be obtained, but morphia is dangerous in these conditions, yet it may be the only drug to take effect. Finally a strict watch must be kept for metastatic collections of pus, particularly those in the pleural cavities.

### GENERAL TECHNIQUE IN TREATMENT

The incisions and details of treatment have been described in each section, but certain general considerations are of importance.

**A. Anæsthetic.**—Gas and oxygen or general anæsthesia is required ; but in mild localised lesions nitrous oxide alone may suffice. It is never justifiable to use a local ethyl chloride spray.

**B. A Tourniquet** should always be used, preferably the arm compressor of a sphygmomanometer.

**C. Incisions** are made to relieve tension and afford drainage. No incision must be made until the operator has decided exactly where the pus is situated. No incision should ever be made on the dorsum of the hand until every other possible site has been eliminated.

**D. Drainage Tubes** should be avoided and rubber tissue used.

**E. Fomentations** are rarely needed after seventy-two hours, when gauze soaked in paraffin and flavine (1 : 2000) is laid lightly in the wound.

**F. Baths of Iodine** (1 dr. of the tincture to 1 pt. of water) at 110° F. are useful in clearing up the infection. Baths of warm dry air are even better, and a special radiant-heat box for the hand will be found of great service (Fig. 117). During the first five days wet and dry baths may be used alternatively morning and evening, but after this dry heat alone will produce the best result. As soon as the wound has begun to heal infra-red treatment should replace the radiant heat.

**G. Active Movements** should be begun at once, and for this reason dressings should be reduced to a minimum and no tight bandaging allowed. Patients will find that these movements are more easy and less painful if performed while the hand is in the wet or dry bath. If such movements are constantly practised, no stiffness in the joints will remain.

R. M. HANDFIELD-JONES.

## CHAPTER XV

### THE SURGERY OF THE BLOOD VESSELS

**A***NATOMY*.—An artery has three coats. The internal coat or tunica intima consists of a single layer of endothelial cells supported by fine fibrous tissue and a layer of longitudinal elastic fibres. The middle coat or tunica media is a thick layer of unstriated muscle, arranged circularly and reinforced by elastic tissue and some longitudinal muscle fibres. The external coat or tunica externa is a mixture of fibrous and elastic tissue. The vessel walls have their own blood supply—the vasa vasorum—their own lymphatic drainage, and are richly supplied with sympathetic nerve fibres.

A capillary has no muscle or elastic fibres, and is composed of an endothelial lining inside a fine fibrous sheath.

A vein is thinner walled than an artery, having only two coats, the inner composed of endothelium standing on a firm fibro-elastic layer, and the outer consisting of fibrous tissue with a small amount of elastic fibre. Most veins have valves at intervals in their course to prevent back flow, but certain important ones are valveless, *e.g.*, the portal system, the superior and inferior cavæ, the common femoral, internal jugular, renal and spermatic veins. In the skull certain special venous channels lie within the reflections of attachment of the dura mater and are known as venous sinuses.

*Collateral Circulation*.—When a large vessel is injured or obstructed, the vitality of the part supplied by it depends upon the anastomosis between the branches arising above and below the obstruction. By active vasodilatation and by a rise in blood pressure the anastomotic channels are opened up and eventually enlarged by hypertrophy, thus allowing blood to be carried to the main vessel below the lesion, and the supply to the distal parts is restored. This process is known as the establishment of collateral circulation; several days are required for its completion, which may be delayed or prevented by disease of, or pressure on, the vessel walls. It is likely to be established more efficiently if the arterial obstruction has not been of sudden onset, and gangrene is less likely to occur under these conditions than in cases of sudden complete rupture or occlusion.

### INJURIES TO ARTERIES

**Contusions**.—An artery is bruised by a crushing force directly applied to it, but this contusion is unlikely to occur unless the vessel is running near a bone. A normal vessel suffers little owing to its natural elasticity and lateral mobility, and the bruise speedily heals. If, however, the artery is atheromatous or otherwise diseased, thrombosis usually occurs and dry gangrene may develop in the parts beyond, or the wall may be so weakened that it will yield slowly and give rise to a traumatic aneurysm.

**Rupture** without external wound may follow blows or crushes, or the vessel may be injured in fractures and dislocations by the displaced bone or during attempts to reduce long-standing dislocations, particularly of the shoulder. Such injuries do not occur unless the artery is diseased.

**PARTIAL RUPTURE** consists in a tear of the intima and media, the external coat remaining intact. When the whole circumference of the vessel is affected the elastic fibres of the intima cause it to curl up inside the lumen and thrombosis ensues, the vessel becoming occluded. If the tear is limited to one side only, a localised thrombosis forms and a weak spot may result, which leads to the formation of a traumatic aneurysm. In certain instances a dissecting aneurysm may follow this type of injury.

*Symptoms* consist in local tenderness and slight swelling, absence or weakening of pulsation of the artery distal to the injury, and coldness and loss of power in the limb. Gangrene is unlikely to follow, and in a few weeks the limb returns to normal, unless the vessels are seriously diseased.

**COMPLETE RUPTURE** affects all three coats and leads to an extensive extravasation of blood. This bleeding may be neither so free nor so immediate as might be expected, owing to the obstruction of the lumen of the artery by the coiling up of the intima and to a fall of blood pressure due to shock. The surrounding tissues are forced apart by the escaping blood, and a large cavity is formed, filled with clot and liquid blood. Fibrin forms at the periphery, and later, fibrosis leads to the formation of a definite capsule to the swelling. The size and shape of this swelling are decided by the anatomical arrangement of the parts concerned and by their resistance. The best examples of this type of injury are rupture of the popliteal artery due to fracture of the lower end of the femur, of the axillary artery in dislocations of the shoulder, and of the middle meningeal artery in cranial injuries.

*Symptoms* are general, local and distal. At the time of injury the patient may experience a feeling as if something had snapped in the limb.

(a) General symptoms are those of internal hæmorrhage with shock, due partly to the severity of the accompanying injuries and partly to the loss of blood.

(b) Locally a swelling appears, either immediately or after a few hours, and steadily increases in size. It is firm, tense and tender and exhibits an expansile pulsation, a thrill and a systolic bruit, all of which disappear later as the layer of fibrin slowly increases in depth. The skin is distended and may be very tightly stretched, and extensive bruising appears after a few days. The swelling cannot be reduced by pressure, and no such attempt should be made for fear of displacing the clot and precipitating a fresh hæmorrhage. The local condition is described as a "*Pulsating Hæmatoma*" (Fig. 118).

(c) The distal symptoms are produced by vascular disturbance and by pressure on surrounding structures. Vascular interference includes cutting off of the arterial supply and pressure of the extravasated blood on the veins. The limb becomes cold, cyanosed

and swollen, and no distal pulse can be felt. Pressure on the nerves produces pain, tingling and numbness, and later there is some loss of power and sensation, but these symptoms may be due to actual nerve injury at the time of accident.

*Complications.*—(1) Gangrene of the moist type is likely to occur, as the pressure of the clot on surrounding tissues makes the opening up of the collateral circulation a difficult matter. (2) Rupture or sloughing of the skin may follow if the distension is very great, and the patient will die of hæmorrhage unless help is at hand. (3) The injury may include rupture of the serous lining to a cavity such as the pleura or peritoneum, and the patient may bleed to death internally. (4) Suppuration of the hæmatoma will require urgent surgical treatment, lest the skin give way and a secondary hæmorrhage result. (5) A true traumatic aneurysm will be formed in some cases in which the vitality of the limb has been maintained. The cavity slowly shrinks by fibrosis of its fibrinous wall, and eventually endothelium grows in and lines the sac.

*Treatment* consists in operation as soon as possible. The main vessel is controlled by tourniquet, digital compression or Crile's clamp, and the clot is exposed and shelled out. The damaged artery is identified and the extent of the injury determined. If the tear does not

involve the whole circumference, if it is complete but clean cut and without loss of tissue, if the walls are healthy and if there is no possibility of sepsis, then an arterial suture may be attempted by expert hands. In all other cases ligature of each end of the torn vessel is the correct procedure; proximal ligature of the main trunk must never be practised as it predisposes to gangrene and secondary hæmorrhage. If gangrene or a severe secondary hæmorrhage occurs, amputation is urgently called for.

Injuries to arteries in the buttocks provide the exception to all these routine procedures. The bleeding cannot here be controlled by pressure, so that an incision must rapidly be made into the clotted area, two fingers thrust down into the depths of the wound and the actual bleeding points thus compressed. The clot is removed and the parts widely exposed. It may be impossible to ligate the proximal end as the artery may have retracted within the pelvis, and if packing fails to arrest the bleeding, the internal iliac artery must be tied through an anterior incision.

**Penetrating Wounds** produce conditions similar to those of subcutaneous rupture, except that the external wound is likely to permit unrestrained visible bleeding and to introduce sepsis. The



FIG. 118

Pulsating hæmatoma due to diffusion of a popliteal aneurysm. The whole region rose and fell with each pulse beat.

amount of hæmorrhage depends on the nature of the wound and the size of the vessel. If the skin lesion is small and valvular in type, little external bleeding will occur. Complete division of an artery allows retraction and curling up of the intima and the bleeding may cease spontaneously. Clean longitudinal cuts bleed but little, as the elastic and muscular fibres tend to close the gap, but incomplete wounds, especially if ragged and lacerated, cause profuse and prolonged bleeding because the opening is enlarged by the retraction of the coats. Clean punctures with a needle close immediately, and the modern small high-velocity conical bullet has caused through-and-through wounds with little bleeding. Partial injuries produce similar results to the non-penetrating type, traumatic aneurysms following either from fibrosis of the clot and endothelialisation of the cavity, or from the slow yielding of an intact intima through the divided middle and outer coats.

Injuries to arteries play an important part in the surgery of gunshot wounds, shell fragments causing septic and widely lacerated wounds, whereas bullet wounds may be mere aseptic punctures.

The local symptoms are those of the wound and of external bleeding. Distally, the appearance of gangrene depends on the particular artery injured, the extent of damage to surrounding structures (particularly the accompanying vein), the state of the arteries, the general condition of the patient and the degree of sepsis present. If gangrene is avoided, recovery is usually complete, although in a few instances a permanent ischæmia results with coldness, weakness and anæsthesia of the limb,



FIG. 119

Wounds in the front of the thigh from Bren gun bullets. An arterio-venous aneurysm resulted.

comparable to Volkmann's ischæmic paralysis of the forearm (Chap. L).

**Arteriovenous Wounds.**—The simultaneous wounding of an artery and vein lying in close contact results from penetrating wounds, in which the contiguous surfaces of the two vessels are punctured, without much disturbance of the surrounding structures, with the result that a communication is established between the two vessels. The majority follow war wounds, though some are met with in civilian practice, especially from wounds with sharp fragments of glass. The vessels most commonly affected are the popliteal artery and vein, femoral artery and vein (Fig. 119), internal carotid artery and internal jugular vein, whilst others provide occasional examples, *e.g.*, brachial artery and median basilic vein, internal carotid artery and cavernous sinus, facial artery and vein, the posterior tibial vessels and those in the orbit. The communication occurs at the time of injury and little extravasation takes place. No swelling appears and the vascular injury remains unsuspected until the establishment of an arteriovenous aneurysm produces its symptoms of vascular derangement in the parts concerned. These are discussed on p. 271.



## DISEASES OF ARTERIES

## ACUTE ARTERITIS

This is of three types : traumatic, infective and embolic.

**Traumatic Arteritis** is a process of plastic repair whereby a contusion or wound of an artery is healed.

**Infective Arteritis** results from involvement of the arterial wall in a septic process surrounding it, as in an abscess cavity, the floor or wall of a spreading ulcer (*e.g.*, peptic) or a tuberculous cavity in the lung. The use of an infected ligature in tying a vessel in continuity may also produce the condition. The coats become hyperæmic, oedematous and softened, leading in the smaller vessels to thrombus formation which seals the lumen, while in the larger arteries the wall is likely to stretch and then give way, a secondary hæmorrhage being the result.

**Embolic Arteritis** occurs from the lodgment of a septic embolus in a small artery. If the clot is virulently infected suppuration occurs and a pyæmic abscess is formed, but if the infection is mild, a softening of the arterial tunics leads to a gradual aneurysmal dilatation. This is the commonest cause of a spontaneous aneurysm in young people.

## CHRONIC ARTERITIS

**Atheroma** (localised or nodular arteriosclerosis) is seen in the arch of the aorta, in the thoracic and abdominal aorta, around the orifices of the aortic branches and in the larger arteries, particularly in those areas where these pass over bony prominences. Greyish white raised patches appear and slowly increase in size. They are due to a thickening of the intima from an inflammatory lymphocytic infiltration, accompanied by a deposit in the space between intima and media of necrotic cells undergoing fatty degeneration. Calcareous plaques are formed by calcification of the patches and atheromatous ulcers follow. The condition has little surgical interest except as a cause of aneurysm, thrombosis and embolism (Fig. 120).

**Diffuse Arteriosclerosis** occurs in the smaller arteries and in the main vessels to the limbs. It may be associated with atheroma of the aorta. The changes in the intima are of secondary importance, the middle and outer tunics being affected by extensive fibrosis which leads to loss of elasticity and narrowing of the lumen. The vessels are thickened, cord-like and tortuous. In certain instances the degenerated areas become calcified and the arteries so hard and rigid (the "pipestem" artery) that when visible they may be seen to move backwards and forwards with each pulsation. A primary calcareous degeneration, known as Mönckeberg's disease (Fig. 121), is a particular variety of arteriosclerosis which affects the media only, leading to extensive calcification. X-rays will demonstrate the extent and distribution of these changes.

The causation of these arterial degenerations is not clearly understood. In a mild form they are probably an expression of the reaction

to fair wear and tear, and are therefore to be expected in elderly people. Heredity undoubtedly plays a part in those cases which are met with in young subjects. Chronic alcoholism, gout, lead poisoning, chronic nephritis, physical and mental overwork, prolonged anxiety and certain toxic states are all said to be contributory factors. Syphilis may be the cause in some, but is certainly absent in many patients.

Arteriosclerosis concerns the surgeon because of its possible



FIG. 120  
Atheroma of the aorta.

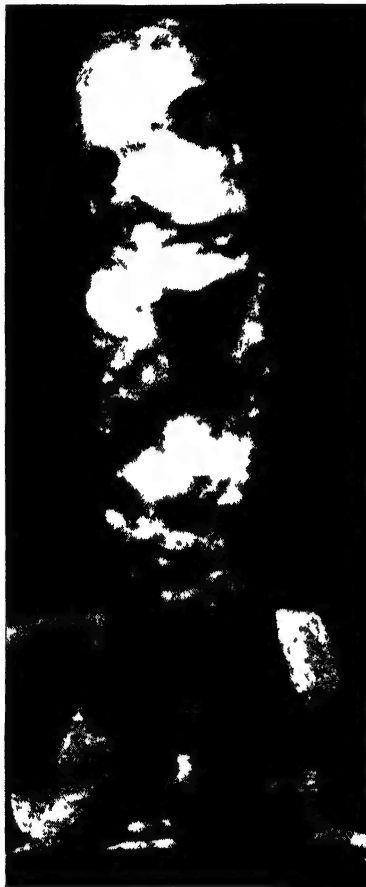


FIG. 121  
Monckeberg's disease.

association with gangrene and of the influence it may exert upon the suitability of patients to withstand operations. In the pre-gangrenous stage, there are coldness, numbness, tinglings and prickings in the extremities, while walking produces cramp-like pain in the calves, which passes off after a rest. This "intermittent claudication" may be the first complaint. Gangrene usually starts in the big toe and is of the "dry" and "senile" type. The treatment of arteriosclerosis comes within the province of the physician, but that of gangrene is essentially surgical and has been described in Chap. IX.

**Chronic Syphilitic Arteritis** takes the form of an endarteritis obliterans leading to complete occlusion of the lumen of small arteries

and arterioles by thickening of the intima and externa. It is the dominant change in many of the tertiary lesions, affecting especially the vessels of the brain and kidneys, and is the determining factor in the formation of a gumma.

**Chronic Tuberculous Arteritis** is a similar pathological condition and accounts for caseation in tuberculous foci.

### TOXIC ARTERITIS

This is exemplified by diabetes, which affects the anterior and posterior tibial arteries and may lead to a moist gangrene. Amyloid disease is a toxic degeneration of the middle tunic of the smaller arteries seen in cases of long-standing mixed infection, especially those which have sinuses discharging on the skin surface.

### THROMBO-ANGIITIS OBLITERANS

Buerger's Disease is an inflammatory or toxic degeneration which attacks both the arteries and veins of a limb, combined with intermittent spasmodic contraction of their walls. The vessels may ultimately become occluded by a clot, which in time may be canalised. It is most often seen in the Jewish race and is practically confined to men. Little is known of its etiology, although excessive smoking has been proved to be a factor. As a rule it affects one leg first then the other leg, and later also the arms (Fig. 122); it may last for years before the onset of gangrene with attacks of numbness and claudication. The treatment is most unsatisfactory as the disease is steadily progressive. One case of the author's illustrates the difficulties, the disease first appearing as an acute abdominal emergency from thrombosis of the ileocolic vessels, which necessitated an intestinal resection for gangrene of the ileocæcal region. Some years later, first one leg and then the other participated in the disease, an amputation being performed on one side. Fortunately, it is a comparatively rare condition. In the early stages, the object is to prevent the occurrence of gangrene.



FIG. 122

Gangrene of the hand, resulting from angiitis obliterans in a patient who had previously had both legs amputated for the same condition.

Sympathectomy (*i.e.*, either lumbar or cervical ganglionectomy) holds out some hope of arresting the progress of the disease.

### ANEURYSM

An aneurysm is a sac containing either fluid or clotted blood which communicates with the lumen of an artery. They are termed "true" aneurysms if the wall is composed of one or more of the arterial tunics, and "false" if they are formed by condensation and fibrosis of the surrounding structures. They are further differentiated into two groups: "internal" when in the thorax or abdomen, and "external" if in the neck or limbs. Three varieties of aneurysm are described, *viz.*, spontaneous (or pathological), traumatic and arteriovenous.

#### SPONTANEOUS OR PATHOLOGICAL ANEURYSM

The two chief causes are disease of the arterial wall and rise of blood pressure, these usually being present together. Syphilitic aortitis, atheroma and arteriosclerosis bring about that fibrosis and loss of elasticity which leads to yielding of the arterial wall, while other causes of localised weakness are pyæmic, bacterial or mycotic emboli. The rise of blood pressure which predisposes to aneurysmal dilatation is not the constant type seen in the elderly, but the intermittent variety which accompanies sudden bursts of heavy work or violent exertion, especially in those who usually lead sedentary lives. Men are more commonly affected than women in the ratio of 8 : 1, and the people of the northern and colder parts of the world provide the majority of the victims. Syphilis is unquestionably an important predisposing factor, and it is probable that in women all pathological aneurysms are syphilitic in origin. There are three types.

1. **A Fusiform Aneurysm** is a spindle-shaped dilatation of the whole circumference of the vessel wall. All three tunics are present in the walls of the sac, the inner and outer coats being thickened and hypertrophied. Atheromatous patches may be present on the intima. Spontaneous cure is unlikely to occur because the intact intima and the direct flow of blood militate against the formation of clot. They advance in size slowly, produce no urgent symptoms until eventually they reach a great size, and press upon all neighbouring structures. Fusiform aneurysms are confined to the aorta and great vessels, and being typically "internal" they are of more interest to the physician than the surgeon. Rupture is unlikely to occur (unless at any time one part of the wall yields to form a sacculated aneurysm).

2. **A Saccular Aneurysm** is formed by the yielding of a localised area of weakened arterial wall and does not therefore involve the whole circumference of the vessel. The sac communicates with the lumen of the artery by an opening of varying size in its lateral wall. It is commonly found in the limbs, as trauma has usually played some part in its formation. The intima and media stop short at the mouth of the sac, the wall of which is the external tunic reinforced by a compressed and fibrosed layer derived from the surrounding tissues.

The absence of the endothelial lining encourages the deposit of successive layers of blood clot, which vary in colour from greyish white at the periphery to reddish brown in the centre. This laminated clot not only strengthens the walls but may progress until the sac is completely filled with firm fibrin and a spontaneous cure is achieved. These aneurysms increase in size more rapidly, are more liable to end in rupture or diffusion and are more amenable to treatment than the fusiform.

3. **A Dissecting Aneurysm** is seen only in the aorta and its main branches. If an atheromatous patch ulcerates and a plaque is shed, blood may be forced between the arterial tunics. A cavity is thus formed bounded by the intima and part of the media, internally, and the rest of the media and the externa, externally. The blood strips

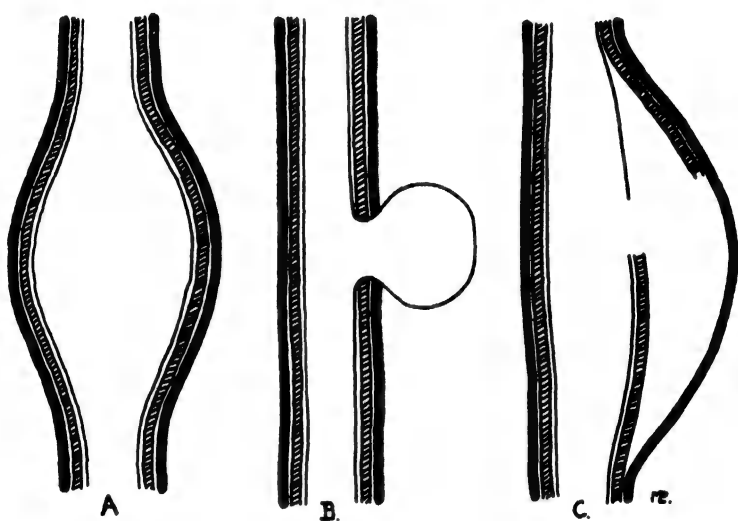


FIG. 123

Diagram showing A, a fusiform aneurysm ; B, a saccular aneurysm ;  
and C, a dissecting aneurysm.

The sac in B is formed, not by intima, but by the thinned external tunic.

up the wall of the artery for considerable distances and either re-enters the lumen through another patch of atheroma or bursts outside the vessel. The condition cannot be diagnosed with any certainty before death, and no treatment is of any avail (Fig. 123).

*Symptoms and Signs.*—The aneurysms which come within the ken of the surgeon are the external and the saccular. The following description therefore refers to these types. Their clinical features may be described as intrinsic and extrinsic.

**Intrinsic Signs.**—A smooth rounded tumour, which is tense and tender, is present in the line of an artery. It can be moved from side to side across the long axis of the limb, but not longitudinally. It presents an expansile pulsation with each beat of the heart, in such a way that the swelling does not merely move forward with each pulsation, but expands equally in every direction. When the main vessel is compressed on the proximal side, the swelling becomes smaller

and softer and the pulsation ceases. It can be still further diminished in size by local pressure over it. When the pressure is released the swelling rapidly (within two or three beats) resumes its original size, shape, consistence and pulsation. Distal compression of the artery causes the swelling to become more tense and the pulsation stronger. The examining fingers can usually detect a vibratory thrill with each pulsation, and the stethoscope reveals a systolic bruit which is conducted both proximally and distally in the line of the vessel. This murmur is usually harsh and loud, but may be soft and musical and is more pronounced in the fusiform than in the saccular aneurysm. All these physical signs will be proportionately diminished in relation to the amount of clot present in the sac. A "consolidated" aneurysm presents as a solid tumour attached to the artery, having only communicated pulsation.

**Extrinsic signs** are produced by pressure and by circulatory interference. They will be trivial at first but become progressively worse as the sac increases in size. The distal pulse is diminished and delayed the parts supplied are cold and numb, and if the swelling compresses the vessels of the collateral circulation, gangrene may occur. Pressure on veins gives rise to cyanosis and oedema of the limb; on nerves to pain, altered sensation and muscular weakness; on bones and joints to great pain from erosion, which may end in a spontaneous fracture. Soft tissues and cartilage are less extensively atrophied and eroded than bone owing to their greater resilience; this is well exemplified by aneurysm of the descending aorta which wears away the vertebral bodies more deeply than the intervertebral discs.

**Differential Diagnosis.**—Aneurysms have to be distinguished from other pulsating swellings. One of the things the student finds misleading is the "transmitted pulsation" of a normal artery to the examining hand by an intervening structure, *e.g.*, an abscess, a cyst, a solid tumour or even a normal organ. A similar difficulty arises when a normal artery is pushed forward by a pathological swelling behind it. The abdominal aorta in thin nervous women is frequently regarded by the inexperienced as an aneurysm, but in each instance the pulsation is not expansile and none of the classical signs of aneurysm are present. True expansile pulsation is seen in some bone sarcomata, nævi and goitres, but these swellings do not lie in the line of an artery, or diminish in size when the artery is compressed, and other methods of examination will eliminate an aneurysm. Finally, peripheral pain, such as sciatica, may be due to an aneurysm at a distance, and such pain must never be attributed to trivial causes, until the patient has been thoroughly examined for evidence of serious trouble.

**End Results and Complications.**—1. **A Saccular Aneurysm** may undergo spontaneous cure by the clotting of its contents and fibrosis of the sac, being then known as a "consolidated aneurysm." This happy result is accelerated by the blocking of the opening with a clot, or by the sac itself pressing upon the proximal artery and so reducing the flow of blood. It can never occur in the fusiform variety.

2. **Diffusion and Rupture.**—Diffusion means that the escaped blood has no access to the surface and so extravasates into the cellular

tissues (Fig. 124). Rupture implies that the blood flows externally or into a mucous tube or serous cavity. When an internal aneurysm gives way, blood enters the pericardium, pleura, mediastinum, peritoneum, œsophagus or trachea. The signs and symptoms are pain in the heart and collapse, death resulting usually within a few minutes or, at most, a few hours. If the trachea or œsophagus have been eroded, there will be profuse bleeding from the mouth.

Rupture may be slow or sudden. When a slow leakage occurs the swelling gradually increases in size and is no longer clearly defined; pulsation diminishes in force, the signs of circulatory disturbance in the distal part of the limb become more advanced, and gangrene may set in. Sudden rupture is accompanied by acute pain in the region of the aneurysm, and the whole area becomes greatly swollen and tense and gangrene rapidly supervenes.



FIG. 124

Dissection of popliteal aneurysm shown in Fig. 118. White guide is anterior to artery; immediately above is the aneurysmal which has leaked.

3. **Suppuration** may follow ligation of the artery, leakage, rupture of the aneurysm, or a septic embolus. The tumour shows all the signs of local inflammation, and if no treatment is given the skin breaks down and pus, blood clot and fresh arterial blood are ejected. The patient dies either immediately or after a short interval, unless treatment is speedily undertaken.

4. **The Heart** is dilated and hypertrophied in all cases, and a hæmic bruit—systolic in time—is audible at the apex.

5. **Gangrene** may be due to (a) pressure on the main venous trunks and is then moist in type, or on the arteries of the collateral circulation when it is of the dry variety; (b) plugging of the vessel below with detached pieces of fibrin; (c) diffusion or rupture.

6. Finally, death may occur from **cerebral embolism** or from pressure on vital structures in the neck and chest.

*Treatment.*—**General Treatment** is to be regarded as preliminary or accessory to operation, except when this is impracticable. It is directed towards reduction of the blood pressure and increase of the clotting power of the blood. Complete rest must be insisted upon and all sources of mental anxiety removed. The bowels should be freely



opened daily, and potassium iodide (gr. xv t.d.s.) and calcium lactate (gr. v t.d.s.) given by mouth. Diet should be light and fluid intake restricted to one pint a day.

**Operative Treatment.**—Many methods are now of historical interest only, and these will be mentioned briefly.

1. The obliterative endaneurysmorrhaphy of Matas is in theory the ideal procedure, and indeed may be the only method available when ligature has failed and excision is impossible. A tourniquet having been applied, the sac is exposed, opened along its free border, and the clot shelled out. The openings of *all* vessels entering or leaving the sac are firmly closed with silk sutures. Any redundant folds of the sac are removed, and the walls oversewn by successive layers of silk sutures, so that the sac is replaced by a solid fibrous pad which obliterates the lumen of the parent vessel. The procedure may be difficult, if not impossible, in some cases of lobulated and extensive sacs, or dangerous if it entails a delicate dissection which may injure the collateral circulation. Matas has suggested two modifications, which are of brilliant conception but of doubtful practical value, viz., the conservative and the reconstructive endaneurysmorrhaphy, in which an attempt is made to preserve or reform the lumen of the parent vessel.

2. Excision of the sac is an admirable method, except for one disadvantage, the danger of injuring the nerves, veins and collateral arteries which may lie in close contact with the sac. It is the operation of choice in all aneurysms of small vessels, *e.g.*, below the knee and the elbow.

3. Ligature of the artery may be either proximal or distal, and either close to the sac or at a distance. (a) Anel's proximal ligature is placed close to the sac, so that no branches intervene between it and the ligature. (b) Hunter's proximal ligature at a distance is so placed that one or more branches intervene and aims at a gradual reduction of the blood stream. (c) Brasdor's distal ligature is placed as close to the sac as possible. (d) Wardrop's distal ligatures are placed on the main branches of the affected vessel as in the case of an innominate aneurysm, when the common carotid and subclavian arteries are tied.

4. The introduction of foreign bodies into the sac cannot be regarded as a satisfactory procedure. Steel wire coils have been passed through a fine needle. D'Arcy Power and Colt employed an ingenious contrivance which could be collapsed into a small compass for introduction, and then expanded into a spherical cage *in situ*.

5. Electrolysis and acupuncture have found an occasional advocate for internal aneurysms, but are really only of historical interest.

6. Compression methods are obsolete except as a preliminary measure.

7. Amputation will be called for in all cases of gangrene, rupture, leakage and suppuration if other methods fail, of secondary hæmorrhage and of erosion of bones and joints rendering the limb useless.

It will be realised that Matas' obliterative operation is the method of choice, but it may be impracticable if the artery is the seat of



degenerative as well as aneurysmal changes. Excision is equally valuable if it can be done without injury to surrounding structures, and especially to the collateral circulation. The proximal ligature of Anel is satisfactory and will be practised as the routine procedure if radical methods appear too formidable. Distal ligature is used only when other methods are impossible, and is far from satisfactory.

A leaking aneurysm demands urgent attention, the artery being tied immediately above the sac, and if this fails to arrest the bleeding, excision or Matas' operation should be attempted. An infected aneurysm also calls for energetic measures similar to the above, but considerable anxiety remains till all danger of secondary hæmorrhage is past.

#### ARTERIOVENOUS ANEURYSMS

These have been defined above (p. 262). Two main types occur, the aneurysmal varix and the varicose aneurysm.

**The Aneurysmal Varix** is a direct communication between artery and vein without an intervening sac. The veins are incapable of withstanding the arterial pressure and become dilated, tortuous and varicose, both above and below the opening. The branches in the vicinity are so distended as to render the operative approach to the main vessels a formidable procedure. The plexus of dilated veins forms a pulsating swelling, exhibiting a thrill on palpation which is continuous, but accentuated with each heart beat. The stethoscope distinguishes a loud murmur which also varies with the heart beat, the noise being likened to the angry buzzing of bees in a paper bag. A systolic bruit is conducted distally in the artery and a diastolic bruit can be traced proximally in the vein for a considerable distance. A systolic bruit will be heard also at the cardiac apex and after some weeks the heart begins to dilate and hypertrophy. Although the process is slow, it is persistent and in time X-rays will show a greatly enlarged heart. The symptoms are dull aching pain in and swelling of the limb, which lead to a reduction in working capacity by about 35 per cent.

*Treatment.*—Although the local conditions can be controlled by elastic support in many patients, operation should be advised in every case in order to prevent dilatation of the heart. The vessels are exposed, the communication between them cut through, and the walls of each carefully sutured. If this is impracticable, the artery should be tied above and below the opening.

**The Varicose Aneurysm** differs from the varix by reason of the existence of a sac between the vessels, through which blood passes from the artery to the vein. The sac is of the false aneurysmal type, the walls being composed of fibrous tissue. The results and clinical features, except for the presence of the pulsating sac, are similar to those of the varix, but, whereas this latter may be present for years without serious inconvenience to the patient, the varicose aneurysm enlarges rapidly and ends in diffusion or rupture. Operation should be undertaken in every case, the ideal procedure being to expose and excise the sac, the vessels being separately closed by lateral suture.

This is rarely possible and the damaged area of both artery and vein should be resected. Fig. 125 shows diagrammatically the difference between the types.

Cirroid aneurysm and aneurysm-by-anastomosis are described in the section on Angiomata (p. 287).

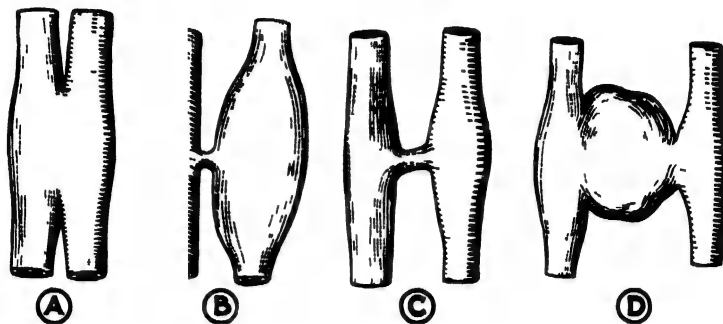


FIG 125

Varieties of arteriovenous fistulae

(A) Direct communication between artery and vein. (B) Aneurysmal varix. The vein is dilated evenly. (C) Arteriovenous fistula united by a small fibrous canal. (D) Varicose aneurysm.

### ANEURYSMS OF INDIVIDUAL ARTERIES

**Aneurysm of the Thoracic Aorta** is essentially a medical condition and comes within the scope of the surgeon only as a diagnostic problem. The signs and symptoms depend on the position of the sac and the structures involved by erosion or pressure. The clinical picture is admirably set forth in Conybeare's "Textbook of Medicine." The results attending such surgical operations as "wiring" and electrolysis are not sufficiently encouraging to warrant their performance.

**Aneurysm of the Innominate Artery** is rare, fusiform, and usually associated with a similar condition of the aortic arch. It presents as a pulsating swelling behind the right sternomastoid muscle and may push the right sternoclavicular joint forward. Symptoms are due to pressure, and are (1) local pain if the sternum or clavicle is being eroded; (2) referred pain in the distribution of the brachial plexus; (3) muscular weakness in the right arm; (4) swelling and œdema of the right arm and right side of the neck, which swelling may involve the left side if the sac is pressing on the left innominate vein; (5) dyspnoea; and (6) dysphagia. The signs will be (a) diminution in the right carotid and right radial pulses; (b) contraction or dilatation of the right pupil due, respectively, to paralysis or stimulation of the sympathetic; (c) sweating and flushing of the right side of the face; (d) local signs of the tumour—an area of dulness, pulsation, bruit, etc.; and (e) a characteristic shadow shown by radiography.

*Treatment* consists in partial distal ligation at a distance (Wardrop's operation). Total ligation is impracticable because of the danger of cerebral anæmia. The method of choice is the simultaneous ligation of the right common carotid and the third part of the right subclavian

artery. The danger of hemiplegia is a very definite one, but it has to be faced. If untreated, innominate aneurysms rupture either on the surface or into the mediastinum, trachea or oesophagus.

**Aneurysm of the Common Carotid Artery** is rare, but among women it is the commonest of all; even so it is more frequent in males than females, and on the right than on the left side. The dilatation is near either the bifurcation or, on the right side, its origin. The intrinsic signs and symptoms of all aneurysms are present and the pressure symptoms are pain, cough, dyspnoea, and those due to interference with the cerebral blood supply.

*Diagnosis* from other aneurysms at the root of the neck may be difficult, especially as dilatation of the aortic arch and of the innominate artery may be accompanied by dilatation of their branches. Careful analysis of the symptoms and signs and radiography should lead to an exact diagnosis. Pressure on the left recurrent laryngeal nerve is undoubted evidence of an aortic aneurysm; pressure on the right recurrent nerve points to an aneurysm of the right subclavian or of the innominate. Diminution of the radial pulse alone means a subclavian sac, of the superficial temporal artery alone a common carotid sac, of both temporal and radial on the right side an innominate aneurysm. "Tracheal Tug" is said to be found in aortic lesions only. None of these signs is infallible, however, as the sac of any one vessel may be large enough to press on others near at hand.

*Treatment* of those near the bifurcation is proximal ligature, and of those near the innominate artery, distal ligature (Brasdor's operation). If untreated, they will finally rupture on the surface, into the mediastinum or into the trachea or oesophagus.

**Aneurysms of the External Carotid** and of the extracranial part of the **Internal Carotid** arteries are rare, except as extensions from a dilatation of the common carotid artery near the bifurcation. The swelling is between the angle of the jaw and the thyroid cartilage and the hypoglossal nerve, pharynx and larynx may be pressed upon. The internal carotid aneurysm may project into the pharynx, where it is seen as a swelling beneath the mucous membrane, somewhat resembling a peritonsillar abscess. Failure to differentiate the two is likely to be followed by the most disastrous consequences.

*Treatment.*—The sac of the external carotid aneurysm should be dissected out and excised; if this is impossible an attempt should be made to apply a proximal ligature, failing which the common carotid will have to be tied. An internal carotid sac cannot be excised with safety, and the common and external carotid vessels must be tied. If the external is not tied, blood can flow down it into the sac, and so render the operation futile. If untreated, these sacs rupture on the surface or into the pharynx.

**Aneurysms of Intracranial Vessels** occur in the internal carotids, the basilar arteries and any of their main branches. They are either congenital or due to syphilitic arteritis or the lodgment of emboli, and the majority are symptomless until they rupture, the patient then dying suddenly of "apoplexy." Occasionally they cause pain, signs of pressure on localised areas of the brain, and general signs of increased

intracranial tension, *i.e.*, headache and vomiting. Some patients are conscious of a pulsation inside their skulls or of a whizzing bruit. Certain carotid aneurysms give the cavernous sinus syndrome due to pressure upon IV, V and VI cranial nerves. Diagnosis and accurate localisation is made by arteriography (p. 856). The internal carotid on the affected side should be ligatured, though the results are far from encouraging.

**Aneurysm of the Subclavian Artery.**—The first part is affected only on the right side, and then in conjunction with an innominate aneurysm. The third part is sometimes affected in men from carrying heavy weights on their shoulders, and is therefore more common on the right side. Women are rarely affected. The dilatation tends to spread to the second part and the sac is frequently loculated, secondary sacs passing among the many recesses formed by the bones and muscles in this crowded region. The first symptom in many patients is pain in the hand, whilst later, there will be muscular weakness and wasting, anæsthesia and œdema of the arm. The sac may compress the lung and pleura and cause hiccough from pressure on the phrenic nerve. A deep seated swelling will be found in the supraclavicular triangle.

*Treatment* is both difficult and unsatisfactory. Excision and Matas' obliterative operation would give good results, but for the difficulties due to the ramifications of the sac. If the first part is not affected, ligature here is the most promising of all methods, but if this is impossible, the innominate artery must be tied and, in addition, the common carotid in order to prevent back flow into the sac.

**Aneurysm of the Axillary Artery** is usually traumatic in origin—penetrating wounds, fractures and dislocations or their reduction being the predisposing causes. The condition is seen in men only, and the right side is usually affected. A very large sac forms rapidly and the clavicle and ribs may be eroded. Symptoms are pain, œdema and loss of power in the upper extremity. Rupture may occur on the surface or into the pleural cavity.

*Treatment* is excision of the sac, or, failing this, ligature of the third or second part of the subclavian as near to the sac as possible.

**Aneurysms of the Brachial, Radial, Ulnar** and other arteries in the arm are traumatic and should be excised.

**Aneurysm of the Abdominal Aorta** affects either that part from which the coeliac axis arises, or the lower part near the bifurcation. A swelling in the middle line is present, accompanied by pain from erosion of the vertebræ, œdema of the legs due to pressure on the inferior vena cava, and some gastro-intestinal disturbance. An X-ray film will readily distinguish the true aneurysm from those conditions which transmit the pulsation of a normal aorta. A few good results have been recorded of treatment by the introduction of coils of wire. Aneurysms are occasionally seen in the renal, splenic, hepatic and mesenteric arteries.

**Iliac or Inguinal Aneurysms** arise in the common and external iliac arteries or in the common femoral trunk. Owing to the density of the fascia of the thigh, they tend to spread upwards towards the

iliac fossa, enlarging to great size, eroding the ilium, and even reaching the loin. Eventually they burst either diffusely, on the surface or into the peritoneal cavity. They are recognised by the typical pulsating swelling, and pain and œdema in the leg. The iliac sacs may be sufficiently deep-seated at first to escape notice, and the pain down the front of the thigh may be diagnosed as rheumatism or neuritis.

*Treatment.*—Excision is ideal but rarely practicable. Proximal ligature as close to the sac as possible gives good results, the external iliac being approached extra-peritoneally, and the common iliac by the transperitoneal route.

**Aneurysms of the Gluteal and Sciatic Arteries** present pulsating swellings in the buttock and give rise to sciatica from pressure on the great sciatic nerve. Diagnosis is difficult because of the depth of the swelling, while a pulsating sarcoma of the bones of the pelvis may present a similar picture. Treatment consists in a transperitoneal ligature of the internal iliac artery, any attempt to secure the vessel from the buttock being certain to fail as the sac encroaches on the sacro-sciatic notch and may then enter the pelvis.

**Aneurysm of the Superficial Femoral Artery** is traumatic in origin and appears either at the apex of Scarpa's triangle or in Hunter's canal and causes little disturbance to the limb.

*Treatment* is excision.

**Aneurysm of the Popliteal Artery** (Figs. 126 and 127) is the commonest of all aneurysms in the limbs. It may follow injury, arterial degeneration or the lodgment of an embolus at the bifurcation. It is almost always seen in men and may be bilateral. The early symptoms are pain and stiffness in the knee; hence the diagnosis is likely to be chronic rheumatism or osteo-arthritis. The knee is held semi-flexed and a pulsating swelling occupies the popliteal space. If the sac extends forwards, the bones and joint capsule are eroded and severe pain and derangement of the joint results; if it spreads backwards, the leg is swollen, weak and painful, and gangrene is likely to supervene because both the veins and the vessels of the collateral circulation are compressed. There should be no difficulty in diagnosis if the leg is efficiently examined.

*Treatment.*—Excision should never be practised because of the danger of injury to nerves, veins and arterial collaterals. Matas' operation is excellent if it can be done, but in many cases the method of choice will be the classical Hunterian ligature in the canal.

Aneurysms below the knee are invariably traumatic, and are easily cured by excision.

#### PULSATING EXOPHTHALMOS

This condition is so frequently due to aneurysmal conditions that it is most conveniently described here. The causes are (1) a cavernous angioma behind the eye; (2) aneurysms of the ophthalmic or internal carotid artery (intracranial part); (3) a cirroid aneurysm; (4) cavernous sinus thrombosis; (5) an aneurysmal varix between the internal carotid artery and the cavernous sinus; and (6) a pulsating sarcoma of the orbit. It is frequently traumatic in origin

and follows a fractured base of skull which has damaged the region of the cavernous sinus. The eye is displaced forward, the conjunctival and retinal vessels are congested, and the whole globe appears cedematous. Corneal ulcers form, movements of the globe are restricted and vision is impaired. The patient complains of great pain and a feeling of tension in the orbit, and is conscious of a rushing sound. The stethoscope detects a continuous bruit, loud and musical,



FIG. 126

An irregular fusiform aneurysm of the popliteal artery.



FIG. 127

A sacular aneurysm of the popliteal artery completely thrombosed.

comparable to the "bee-in-the-paper-bag" murmur of arteriovenous aneurysm. Compression of the common carotid artery produces a marked mitigation of symptoms, and ligature of the internal carotid artery should be performed.

## THROMBOSIS AND EMBOLISM

### THROMBOSIS

**Thrombosis** is intravascular clotting of the blood, which may take place in the chambers of the heart, the arteries, capillaries and veins, particularly the last named. Predisposing causes are (a) damage to the endothelium by injury, inflammation or degeneration; (b) increased coagulability of the blood due to infection or toxæmia;

and (c) slowing or arrest of the blood stream. The clot may be of two kinds. A *White Thrombus* is the result of slow deposition of successive layers of fibrin and leucocytes, the best example being the whitish grey laminated clot in a saccular aneurysm. Rapid coagulation in a stagnant blood stream produces a *Red Thrombus*, in which all the constituents of the blood are included (Fig. 128). This is found in a thrombosed varicose vein (Fig. 129) and in a vessel after ligation. White clot is firmly adherent to the wall of the vessel, but red clot is quite loose.



FIG. 128

Red thrombus from the common iliac vein and its main branches, from a patient who died suddenly of a pulmonary embolus.



FIG. 129

Thrombosed varicose veins.

*Results of Thrombosis in a Vein.* **A. Local.**—(a) If the clot is sterile, it may gradually become organised into connective tissue and the vein converted into a fibrous cord. Secondly, the clot may be attached to one side of the vessel only, and gradual shrinkage towards its fixed point lead to re-establishment of the blood flow in the vein. Thirdly, organisation may stop short of fibrosis owing to canalisation of the clot by dilatation of the small vessels within it, so that in this way also the lumen is reconstructed. Lastly, a small clot fixed to one side of the vessel may become calcified to form the “phlebolith,” which is so commonly seen in X-ray films of the pelvis, where it may be mistaken for a ureteric calculus.

(b) If the clot is infected no organisation occurs, but softening and suppuration lead to abscess formation, at first in the vein and later outside it (see Phlebitis, p. 281).

**B. At a Distance.**—(a) The distal area drained by the thrombosed vein becomes congested and swollen, unless the affected vessel is small and the collateral circulation good. Blocking of the main vein of a limb leads to great swelling of the whole limb, which is white and pits deeply on pressure. The œdema persists for many weeks and may never clear up completely. Thrombosis of the inferior vena cava, if it does not prove fatal, results in an enormous dilatation of the collateral circulation, hugely dilated veins being seen beneath the skin running from the groin to the axilla. (b) On the proximal side, the thrombus may remain stationary, may gradually extend upwards by further deposition of clot, so reaching and involving larger trunks, or may shed pieces which are swept away in the blood stream as emboli.

*Arterial Thrombosis* is not common (Fig. 130). It is followed by an opening up of the collateral circulation, and gangrene is not likely to follow as the obstruction is of gradual onset.



FIG. 130

A septic thrombus in the aorta.

## EMBOLISM

**Embolism** is the name given to the condition in which a solid or semi-solid foreign substance is swept along in the blood stream, finally to become impacted in a vessel which is no longer large enough to let it pass on. As

the veins of the systemic circulation adilstey increase in size on their way to the heart, emboli obviously can never lodge in them. They are therefore found only in arteries and in the portal venous system. An embolus may consist of (1) a sterile clot derived from a thrombus; (2) an infected clot from a septic focus; (3) fibrinous vegetations from the cardiac valves or atheromatous plaques; (4) clusters of malignant cells in carcinoma or sarcoma; (5) globules of fat; (6) bubbles of air; and (7) parasites, as in hydatid or filarial diseases.

**Results of Embolism.**—As soon as the embolus comes to rest, fibrin is deposited on it and the obstruction of the vessel rapidly becomes complete. The results depend on the size and situation of the vessel blocked, the importance and delicacy of the tissues supplied,



the size of the embolus and whether it is sterile or infected. The effects are as follows :—

1. **TRANSIENT ANÆMIA.**—If the vessel is small, the capillary anastomosis free and the structures supplied unimportant, a transient anæmia occurs without any appreciable symptoms. If the tissues supplied are very delicate and unable to withstand temporary loss of nutrition, function may be lost even if the cells themselves do not die, *e.g.*, the blindness which follows embolism of the central artery of the retina.

2. **GANGRENE** of a limb or of part of the intestinal tract may follow blockage of the main vessel at a point where the collateral circulation is ineffective. Sudden pain is felt at the time of lodgment of the embolus and gangrene appears later.

3. **INFARCTION** occurs in certain organs, the arteries of which are end-arteries (*i.e.*, vessels whose capillaries have no anastomosis with those of adjacent vessels), *viz.*, the lungs, kidneys, spleen and brain. An infarcted area is usually wedge or cone shaped, having the occluded artery at its apex. In a “white infarct” the pattern and texture of the organ are no longer visible, and a white homogeneous area results. A “hæmorrhagic infarct” is purple in colour owing to the presence of extravasated blood. Small uninfected infarcts become organised and converted into puckered scars, except in the brain where softening occurs.

**Embolism in Certain Organs** produces symptoms of such grave importance that they are described separately.

1. **IN THE LUNGS.**—Pulmonary embolism is usually seen as a complication of operations in the abdomen, pelvis or lower extremities. The thrombus forms in the femoral and iliac veins as the result, probably, of low-grade sepsis and a slowing up of the blood stream due to enforced rest in bed. The embolus may be so large that one of the main branches of the pulmonary artery is blocked and death is instantaneous, or the detached fragments of clot may be so small that the effects are trivial; between these two extremes is a wide range of cases of varying severity. The very severe and the fatal cases occur one to three weeks after operation, usually from some movement in bed or on the first occasion that the patient gets out of bed. The symptoms are a sudden violent pain in the chest, a look in the face of anxiety amounting almost to terror, gasping struggling respirations and cyanosis. Death may occur so suddenly that a patient apparently well on the way to recovery falls back dead in the nurse's or doctor's arms, or survives for a few minutes or a few hours. The symptoms in the non-fatal cases vary greatly in degree. There is a sudden stab of pain in the chest, dyspnoea, general distress and anxiety, and after a few hours some blood-stained sputum is coughed up. The condition is then one of pleuropneumonia, and the severity of the subsequent illness will depend on the size of the affected area of lung.

**Treatment.**—Considerably more attention should be given to the prevention of this tragic complication. Patients are so often kept in bed unnecessarily long and are overwhelmed with instructions to lie

still and not move about in bed. Retention in bed should be restricted to the minimum and patients encouraged to move about in bed as soon as and as much as their local or general condition permits. All bands and all pressure on the region of the groins and Scarpa's triangle should be avoided and, whenever possible, splints should be so counterbalanced by pulleys and weights as to allow movements of the patient without interference with the immobilisation of a fracture. Lastly, massage of the lower limbs and the abdomen should be started as soon as possible after operation. Active treatment is symptomatic. Trendelenberg's operation of embolectomy, *i.e.*, removal of the clot from the pulmonary artery, cannot yet be considered to have emerged from the experimental stage. The use of heparin is described below.

2. IN THE BRAIN the middle cerebral artery is blocked and complete hemiplegia results but, although some permanent impairment of function persists, the degree of recovery is surprisingly good. An aneurysm may form later at the site of impaction of the embolus.

3. Embolism of the CENTRAL ARTERY OF THE RETINA produces sudden complete permanent blindness, in spite of the fact that the retinal cells appear to retain their vitality.

4. IN THE LIVER emboli are usually septic and come from some part of the area drained by the portal system. The condition of "Pylephlebitis" is described on p. 687.

5. Embolism of the MESENTERIC VESSELS leads to a form of acute intestinal obstruction (pp. 541 and 644).

6. IN THE LIMBS the embolus usually lodges at the bifurcation of the main vessels, *e.g.*, at the termination of the popliteal artery. The symptoms are sudden severe pain at the site of impaction, tenderness at this point, and coldness, numbness, loss of power and loss of distal pulse in the limb. If the patient is seen within twelve hours of the impaction of the embolus the artery should be exposed, opened longitudinally and the clot removed. The vessel wall is sutured with vaselinised silk with the utmost care. The results of this operation of peripheral embolectomy are distinctly encouraging. If this operation is not done gangrene will follow in almost every case, though recovery is not impossible.

7. In the spleen and kidney embolism causes pain, swelling and a slight rise of temperature. In the kidney there will be a slight transient hæmaturia.

## INJURIES AND DISEASES OF THE VEINS

### INJURIES OF VEINS

**Subcutaneous Rupture** of a vein is commonly seen in association with fractures, dislocations and severe contusions, while varicose veins may rupture subcutaneously from quite trivial blows. Although the vein walls do not curl up and retract in the same manner as the intima of an artery, the blood pressure is so much lower that the extravasated blood quickly compresses the vein and arrests the bleeding.

When a large vein is injured there will be some local swelling and œdema of the limb, but the swelling does not pulsate and the distal pulse is not weakened.

*Treatment* consists in firm bandaging and, in the case of a large vein, elevation of the limb for a week.

**Wounds of Veins** are very common as the result of penetrating injuries and of surgical operations. Small veins readily collapse and bleed little, but hæmorrhage may be profuse from the larger veins, especially if they are diseased or if they are injured where passing through a layer of fascia which prevents them from collapsing. Venous hæmorrhage is a continuous steady welling up of dark blood, which is easily controlled by light pressure. Wounds of the lateral wall of a vein should be closed by a lateral ligature, a vaselinised thread suture or a muscle graft. The ligature of the main vein of a limb may be unavoidable, but it should never be done unless absolutely necessary, as it will probably lead to severe œdema.

**Air Embolism.**—Injuries of large veins, particularly those near the thorax, may give rise to air embolism owing to air being sucked into the vein with each inspiration. If a sufficient amount of air reaches the heart, death may follow from cardiac failure. When the condition occurs during an operation, a hissing noise is heard and the patient becomes pale and collapsed, with a weak running pulse and widely dilated pupils. The heart action is irregular and auscultation reveals hissing and gurgling noises. If the patient survives, no ill-effects remain. This condition is very rare and can always be avoided by proper care in operating, large veins being identified and clamped before division. If a large vein is opened, the bleeding must be stopped by pressure and the wound filled with saline. Each end is then found and ligatured. If air embolism has occurred, the patient's head must be lowered, injections of camphor, ephedrin and digitalin given and, if the heart's action is failing, the peritoneum should be opened and cardiac massage performed; or the right ventricle may be aspirated.

#### DISEASES OF VEINS

**Phlebitis**, or inflammation of a vein, is invariably accompanied by thrombosis, and so many of the clinical signs and symptoms are referable to the latter that "thrombo-phlebitis" is a more exact term. Two forms are described.

**SIMPLE PHLEBITIS** is a localised inflammation of the vein wall, resulting from (1) injury; (2) a low-grade infection; (3) spread from an inflammatory focus outside the vein; or as (4) a toxic complication of typhoid fever, gout or rheumatism. A red thrombus is deposited on the inflamed walls, obliterates the lumen and spreads for a short distance up and down the vein, but it is unlikely to become infected, and the danger of small pieces of clot being detached and forming emboli is slight. It is commonly seen in varicose veins after slight trauma and in patients who have been seriously ill and confined to bed without being able to move about. It is therefore a complication of pregnancy and of operations on the abdomen and pelvis.

The changes in the vein consist in a thickening of its walls and a reddening of the endothelium, to which the thrombus becomes closely adherent. Organisation and possibly subsequent canalisation of the clot are the end results.

*Symptoms.*—Phlebitis of superficial veins is most common in the internal saphenous vein and its radicles, particularly if these are varicose. There is a sudden onset of pain in the vein, the temperature rises to 100° or 101° F. and the patient feels unwell. The affected length of vein becomes swollen and tender, and can be felt as a hard cord with localised knobs corresponding to the valves or varicose pouches. The overlying skin is dusky red, hot and oedematous. Owing to the abundance of collateral anastomoses there is no interference with the blood return from the distal area. These attacks are usually followed by organisation and fibrosis, a spontaneous cure of a varicose vein being thus achieved.

Phlebitis of deep veins usually affects the main trunks of the limbs—the iliac, femoral, popliteal and axillary. The onset is also sudden; there is the same localised pain in the affected vein and fever is present, but the local signs are slight, whereas the distal swelling is likely to be very marked. The signs of deep phlebitis are in fact chiefly those of the accompanying thrombosis. This condition is well exemplified by the “puerperal white leg” in which the lower extremity becomes swollen, white and oedematous (phlegmasia alba dolens). Deep phlebitis may end in fibrosis of the vein, but more often the thrombus becomes canalised and the circulation is restored.

*Treatment.*—The danger of embolism dictates a safety-first policy. The results of the injection treatment of varicose veins suggests that a *superficial* patch of thrombosis need not be taken so seriously as in the past. The limb should be kept at rest, painted with glycerin and belladonna and bandaged over a generous layer of cotton-wool. Ten ounces of 0.5 per cent. solution of sodium citrate in normal saline injected intravenously helps to relieve the pain and localise the thrombosis. As soon as the redness, heat and tenderness have subsided the area should be strapped with elastoplast and the patient allowed up. *Deep* phlebitis does need to be treated with great care. The patient must be kept in bed at least a month, and massage and infra-red therapy started at the end of the sixth week to reduce the swelling and restore the circulation. Many months may elapse before the limb regains its normal size and shape.

**Prevention of Thrombosis.**—HEPARIN is a natural anticoagulant found in many tissues especially the liver and lungs. Although it cannot remove clot already formed, it does prevent any addition to it. Its application to human surgery is still in an early stage. At the present time it is prepared as a solution for intravenous use and the exact quantity required has to be carefully checked in each patient, so that the clotting time is increased to about fifteen minutes. An average dose is about 750 units an hour in normal saline, and if this is continued for any length of time—as it must—the cost becomes a very serious disadvantage.

Some of the most promising fields for the use of heparin are :—

1. Conservative vascular surgery. Operations upon blood vessels, *e.g.*, embolectomy, repair of arteries and veins, repair of an arterio-venous aneurysm often fail because clot is deposited upon the intima traumatised by the operation.
2. War surgery. Under ideal conditions it may be possible to save limbs from gangrene by restoring continuity in main vessels to them.
3. Post-operative thrombosis. As we have seen on p. 279 almost any operation may be followed by intravascular clotting and it is hardly practicable to give heparin as a routine. But if immediately after the lodgment of a small embolus heparin is administered, it is possible that further emboli may be prevented.
4. Constant drip saline and blood infusions may be kept in action for many days without clot forming in the cannula.

INFECTIVE PHLEBITIS is the more serious but less common type. The preliminary changes are the same as in the simple variety, but the clot is infected and slowly breaks down to form dirty greyish pus and the thrombus spreads more extensively both up and down the vein. The inflammation affects the tissues round the vein and an infective periphlebitis follows. Finally, an abscess forms primarily inside the vein but later spreading outside it. Small pieces of disintegrating clot are likely to be detached and septic emboli thrown off into the blood stream.

*Symptoms.*—The picture of simple phlebitis rapidly changes with the advent of infection. The patient becomes gravely ill with rigors and a high temperature of 103° to 105° F., the local condition becomes more extensive and less circumscribed and diffuse suppuration occurs.

*Treatment.*—The pus must be evacuated by suitable incisions, and if there is an extensive area of periphlebitis this must be laid open and drained. If septic emboli are being set free, the vein should be ligated on the proximal side, *e.g.*, lateral sinus thrombosis (p. 392), but this can only be done if a simple venous trunk is affected. If several veins in a limb are affected, and if treatment does not put an end to the shower of emboli, amputation will have to be considered as a life-saving measure.

### VARICOSE VEINS

**A Varicose Vein** is one which has become permanently dilated, lengthened and tortuous. This condition may affect the whole length of a vein or only small isolated patches, and although theoretically any vein is liable to varicosity, yet in practice only the veins of the thigh and leg, the spermatic cord, the ano-rectal region and the lower end of the œsophagus are involved. Varicoceles are described on p. 807 and hæmorrhoids on p. 657. The œsophageal varices occur only in cirrhosis of the liver. The following description applies to varicose veins of the legs.

*Etiology.*—Varicose veins are rarely seen before the age of 15 years, but afterwards they are very common in both sexes and at all ages. Women are affected more than men because of the frequency of the condition in pregnancy. The exact causation is unknown, but there are many contributory factors.

1. Congenital defects, *e.g.*, weakness of the vein wall, absence or incompetence of valves, or over-development of the cribriform fascia in the region of the saphenous opening. The theory of congenital predisposition is upheld by the varix which appears in youth, affects many members of the same family and often the same part of the same vein in that family.

2. Prolonged standing. Waitresses are particularly liable to the condition.

3. Obstruction to the vein. Tight elastic bands, *e.g.*, garters in men and the lower end of knickers in women produce persistent distension of the veins; so also may the pregnant uterus and other abdominal or pelvic tumours.

4. Obstruction to the deep vein leads to a compensatory dilatation of the superficial veins, *e.g.*, in the white leg of the puerperium and in typhoid thrombosis.

5. Arteriovenous aneurysms cause varicosity of the veins.

6. Rupture of the valves may occur in athletes, and accounts for the frequency of varicose veins in an unexpected group of people.

7. The internal saphenous vein contains a long column of blood flowing against gravity. As soon as the valves cease to function properly, a vicious circle is formed and the varicosity rapidly increases in size and extent.

*Pathology.*—A varicose vein is thickened, tortuous and brittle. When cut across it remains cylindrical and does not collapse. The thickening is irregular and patchy, and weak spots are left through which thin-walled pouches project, forming little varices on the main varicose vein. One such secondary varix may occur at the upper end of the internal saphenous vein near the saphenous opening, and if it reaches an appreciable size it is called a "saphenous varix." Microscopically the vein has lost most of its muscle fibres, which are replaced by fibrous tissue.

*Symptoms* of uncomplicated varicose veins are dull aching pains in the leg below the knee with or without swelling at the ankles. The enlarged veins can be seen beneath the skin (Fig. 131), their extent, prominence and tortuosity varying widely in different patients. In



FIG. 131

One single large varicose vein below the knee. An enlarged pre-patellar bursa is also present.

long-standing cases there may be such thickening of the skin and subcutaneous tissues that the veins are not obvious, but can easily be felt as grooves or gutters. A venous thrill can always be felt in a saphenous varix and for some distance below it. Trendelenburg's test is of some importance. When the patient lies down and the limb is elevated the veins empty. The internal saphenous vein near the opening is then firmly compressed and the patient asked to stand up when blood will slowly fill the veins from below upwards. When the pressure is released, a sudden rush of blood from above to fill the veins below is clear evidence that the valves are faulty.

*Treatment.*—**A. Palliative Treatment** consists in the removal of any cause of obstruction, the application of a crêpe velpeau bandage, or of an accurately fitted elastic stocking. These stockings are expensive, they need to be renewed twice a year, and patients find them hot and irksome. No patient should be refused injection unless the general condition renders it inadvisable or there is ground for belief that it will not prove of much value.

**B. By Injection.**—The obliteration of the lumen of the vein by the injection of irritant drugs is now the established method of treatment. The only contraindications are the occlusion of the deep veins by previous thrombophlebitis, and such indifferent general health as to make injection inexpedient or unsafe. Many substances have been used, but the following are established as being the most efficacious : Sodium salicylate in 20, 30 and 40 per cent. solutions ; sodium morrhuate in 5 and 10 per cent. solutions ; quinine and urethane ; and lithium salicylate (30 per cent.). Sodium salicylate has the disadvantage of being painful, but all give excellent results. The patient lies on a couch (there being no need whatsoever for him or her to be standing up) and if the veins are NOT prominent a band is placed round the leg. A very fine hypodermic needle (No. 18 or 20) is inserted into the vein. The beginner will be well advised to withdraw a little blood into the syringe to satisfy himself that the needle is in the lumen of the vein. The band is removed, the vein emptied of blood and the injection made. The length of vein affected by each injection varies considerably, but a reaction of 2 to 8 in. may be expected. No estimate of the number of treatments required should be given until the result of the first has been seen. If any solution leaks into the subcutaneous tissues, pain is felt and sloughing may follow. After the injection a small pad of sterile gauze is firmly bandaged or strapped into position and worn for six hours. There is no need for patients to lie up, but on the contrary they should be encouraged to continue their daily occupation at once. The effects of the injection are as follows : within six hours the injected vein becomes painful and tender in a varying length, and on the following morning it is thickened and the overlying skin may be red and hot. The pain, redness, heat and tenderness wear off in a few days, but the thickening remains for six to eight weeks, during which time the thrombus is organising, until finally nothing remains but a fibrous cord. It is important that patients should be forewarned of this



sequence of events, lest they imagine the treatment has taken an unexpected course or has failed.

**C. Operative.**—If a saphenous varix is present, if the vein is greatly dilated in the thigh and if a thrill on coughing can be felt in the varices below the knee, a ligature should be placed on the saphenous vein close to its entrance into the femoral vein. This is a modification of the old Trendelenburg's operation. It should be combined with injection of the veins in the leg. Many procedures of removing lengths of vein through long incisions are now of historical interest only. Excision by multiple small or one long incision has been displaced by the injection method, which has everything in its favour, viz., time, economy, comfort and success.

**Complications.**—**A. Pigmentation of the skin** is seen in most cases of long standing. It is due to deposition of blood pigment.

**B. Varicose eczema** is preceded by pigmentation and is due to stasis of blood in the skin, combined with the chronic irritation of dirt or clothing. It may readily pass into—

**C. Varicose ulcer**, described on p. 166.

**D. Thrombophlebitis** is very common in varicose veins and may follow minor injury.

**E. Hæmorrhage** results from spontaneous rupture, injury or ulceration. Profuse bleeding occurs from *both* ends of the vein and shows no signs of ceasing as the thickened vein does not collapse. It may prove fatal if the patient is intoxicated and no help is at hand. It can be controlled by simple pressure over the bleeding points, and by elevation of the leg.

## TUMOURS OF THE BLOOD VESSELS

The tumours arising from the blood vessels are known as Hamangiomata and are of four varieties.

**Capillary Hæmangioma** (or Telangiectasis) is usually a congenital growth of tubular capillary vessels containing blood. They are of unequal size and have a very delicate endothelial lining supported by a fine fibrous stroma. They constitute the different types of "birth marks" or nævi, which are seen chiefly in the neck, face and hands. The "spider nævus" occurs on the face as a bright red central point of capillaries, from which spread fine red radiating lines. The "strawberry" and "port wine stain" nævi may reach large size and affect a considerable area of skin. The tumour is not raised above the surface, and is a deep purple or violet colour. They are sometimes associated with other tumours of the skin, such as the pigmented hairy mole and the cavernous angioma. They are also found in mucous membranes and in muscle.

**Treatment.**—Some nævi are readily destroyed by the application of CO<sub>2</sub> snow, the galvanic cautery, electrolysis or radium. The large ones are best left alone, as their destruction may lead to scarring, which is even more disfiguring than the original nævus.



**Cavernous Hæmangioma** occurs either as a congenital malformation in the subcutaneous tissue (Fig. 132) or as an acquired growth in an internal organ, *e.g.*, the liver. Its walls are thicker than those of the capillary nævus, large blood spaces are formed in it, and it is altogether a more fleshy tumour. In the subcutaneous tissues it forms a soft vascular swelling blue in colour (Fig. 133) which

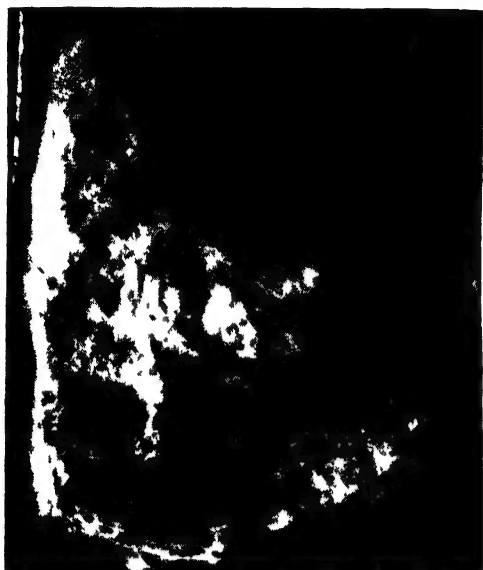


FIG. 132

Large hæmangioma from the subcutaneous tissues of the thigh.



FIG. 133

Hæmangioma on the back of a small child.

can be reduced in size by pressing the blood out of it, while it returns to its normal size when the compression is removed. Occasionally its surface becomes infected and it forms a rapidly growing fungating tumour (Fig. 134). In the liver it usually remains undiscovered except in the post-mortem room.

*Treatment* is excision, cauterisation or injection with sodium morrhuate.

**Plexiform Angioma** is one in which the main bulk of the tumour is composed of arteries. There are two varieties.

1. THE **TRISOID ANEURYSM** is most commonly encountered in the temporal region of the scalp. It may follow injury, develop from a pre-existing cavernous hæmangioma or arise spontaneously. Large tortuous pulsating arterial channels can be seen beneath the skin. It is soft, compressible and the component vessels are easily felt. There will be a thrill and a systolic bruit, while the patient complains of a loud continuous rushing or roaring sound.

*Treatment* is far from satisfactory. The ideal procedure would be total excision combined with ligature of all the vessels that feed



FIG. 134

An infected hæmangioma.

the tumour, but this is usually impracticable. Bilateral ligature of the external carotid arteries may have to be tried.

2. **THE ANEURYSM BY ANASTOMOSIS** consists of much smaller arteries together with veins and capillaries. It occurs in the scalp, neck and upper extremity, and one form is found in the interior of bones, in which it may closely mimic a pulsating sarcoma.

*Treatment* is by excision, electrolysis or radium.

**Glomangioma.**—This rare tumour arises from a normal glomus, which is a direct communication between arteriole and venule occurring in the subcutaneous tissues principally in the terminal segments of fingers and toes. Its function is in the nature of a “shunt” or short circuit and is concerned with regulation of temperature.

This tumour is composed of a plexiform mass of vessels surrounded by a well-defined capsule. They are lined by a single layer of endothelial cells and their walls consist of a thick coat of glomus cells and smooth muscle. Numerous nerve fibres, both myelinated and non-myelinated, can be seen.

Clinically they are quite small (from 3 mm. to 3 cm.), of a blue or purple colour and at first sight suggest a blood blister. Some of them can be emptied by pressure. Subungual tumours are likely to betray their presence by a faint tinge of cyanosis showing through the nail. Pain and exquisite tenderness are the outstanding symptoms.

Treatment consists in excision.

R. M. HANDFIELD-JONES.

## CHAPTER XVI

### THE DISEASES OF THE LYMPHATIC SYSTEM

#### THE LYMPHATIC VESSELS

##### INJURIES TO THE THORACIC DUCT

**T**HE thoracic duct opens into the junction of the left subclavian and internal jugular veins, or into either of them just distal to their union. The duct usually divides into several small channels before reaching its termination so that a wound close to the veins is more likely to involve one of the terminal branches than the main duct itself. Injuries to the duct in the neck occur during operations in the supraclavicular triangle, as a result of gunshot and stab wounds and rarely as a complication of a fracture of the clavicle. If the wound is recognised during the operation, a little spurt of chyle will be seen with each expiration, but usually it remains unsuspected until the subsequent dressings are found saturated with chyle, or until a fluctuant swelling appears beneath the healing skin incision. The divided duct should be ligated if possible, failing which the wound must be packed with gauze ; even if the main duct is ligated little anxiety need be felt, as the intrathoracic anastomoses with other lymph channels will ensure adequate drainage.

The thoracic duct may be injured in the thorax by a fracture of the spine or by crushing accidents, and the chyle may flow into the pleural cavity (chylo-thorax). In the abdomen, the receptaculum chyli may be injured by crushes or penetrating wounds, or may rupture spontaneously from over-distension if the duct is obstructed by the pressure of growths or masses of glands in the mediastinum. In such cases chyle collects in the peritoneal cavity (chylous ascites) and may appear in the urine (chyluria).

The right lymphatic duct may be injured in the right supraclavicular triangle, but little trouble results, and if a fistula does form it heals rapidly. Extensive removal of glands from any area may be followed by a flow of lymph, which delays the healing of the wound until in about four weeks' time new lymphatic vessels are formed.

#### LYMPHANGITIS

**Acute Lymphangitis** is most commonly seen as the result of infections of the fingers or the foot. It is fully described on p. 255.

**Chronic Lymphangitis** may follow the imperfect resolution of an acute attack, but is usually a specific condition, either tuberculous, syphilitic or, very rarely in human beings, due to glanders.

**TUBERCULOUS LYMPHANGITIS** occurs in the vessels of the mesentery, lung and upper extremity. In the arm the infection starts from a primary focus in the hand—often so small as to escape recognition—and spreads up the vessels as an ascending lymphangitis. The affected vessels become thickened, nodular swellings appear at the site of the valves and eventually the nearest group of glands is involved. The nodules are firm and solid at first but slowly soften and become adherent to the skin, which finally breaks down to form a tuberculous ulcer.

*Treatment* consists in excision of the primary focus, of every nodule and of the affected group of glands. The thickened lips in tuberculous children are due to a similar condition, the lymphatics being infected from labial cracks and fissures.

**SYPHILITIC LYMPHANGITIS** affects the vessels along the dorsum of the penis by extension from the primary sore. In well-marked cases an indurated cord can be traced from the sore to the enlarged glands in the groin.

### LYMPHANGIOMA

The processes underlying the formation of tumours of lymphatic origin are so obscure that it is impossible to differentiate with any certainty between the true lymphatic growths of new formation (lymphangiomata) and those swellings which are dilatations of normally existing channels (lymphangiectases). Three types of lymphangioma are described.

**Capillary Lymphangioma** or lymphatic nævus is congenital in origin and occurs in the tongue, lip and skin. In the tongue and lips it is red, in the skin yellowish brown, and it may be flat or papillary. If any treatment is needed it can be destroyed by diathermy.

**Cavernous Lymphangioma** is comparable to the cavernous hæmangioma and consists of thick-walled spaces lined with proliferating endothelial cells. It accounts for one type of macroglossia and macrocheilia which are congenital enlargements of the tongue and lips. It is also seen in the skin, and if any of the surface vesicles rupture lymphorrhœa will result.

*Treatment* is excision if possible. One very extensive type of cavernous lymphangioma is occasionally seen in the neck of infants, and may reach from the lower jaw to the clavicles. It is congenital in origin and consists of cysts of varying size embedded in fibrous tissue. It is rarely possible to remove, as it sends processes among the muscles and tissue planes of the neck, and a fatal issue is to be expected.

**Cystic Lymphangioma** occurs as a unilocular or multilocular cyst in the lower part of the neck and in the axilla. It is congenital in origin and is found in children under the age of ten years as a soft, flabby, lobulated swelling, subject to recurrent attacks of mild inflammation, which cause it to become more tense, somewhat painful and tender and may lead to spontaneous cure. In the quiescent period there are no symptoms.

*Treatment* consists in removal.

## LYMPHATIC OBSTRUCTION

Obstruction of lymph vessels will cause œdema of varying degree, but in many cases where this is extensive it may be difficult, if not impossible, to determine the relative proportion of lymphatic and venous obstruction. As a general rule the collateral anastomosis of the lymph vessels is so extensive that even the removal of large groups of glands or the obstruction of a wide field of lymph vessels may not be followed by lymphatic œdema. To produce this there must be blockage both of the collateral and main channels. Examples of lymphatic œdema are (1) the rare solid œdema of the legs of developmental origin, (2) that following acute lymphangitis of the hand and forearm, (3) a rare result of facial erysipelas and of multiple gummata of the leg. Blockage by carcinoma cells is best exemplified by "Brawny Arm," a late complication of carcinoma of the breast.

## ELEPHANTIASIS

Elephantiasis is a hypertrophic condition of the skin and subcutaneous tissues following prolonged lymphatic obstruction. The parts usually affected are the legs, thighs, scrotum, penis and vulva and occasionally the breast, face and arms. The skin and subcutaneous tissues become swollen from solid œdema, which does not pit on pressure. Later a diffuse hyperplasia occurs, the skin being greatly thickened and coarse. Warty outgrowths and lymph vesicles appear on the surface, and a profuse lymphorrhœa may result from rupture of the latter. The leathery skin is liable to fissuring, ulceration and recurrent attacks of lymphangitis. There are two types of elephantiasis.

**The Filarial Type.**—Elephantiasis Arabum is seen in Barbadoes, the West Indies, China, Japan, Malay States, India and South America, and is due to obstruction of the lymphatics of the inguinal region by the worm *Filaria Sanguinis Hominis*. The infection is transmitted by a mosquito (*Culex fatigans*), in which the intermediate stage of the life-history of the worm is passed. The ova reach the human stomach in drinking water, penetrate the gastric mucosa and enter the lymph vessels. The female worm settles in the inguinal lymphatics and gives rise to countless embryos, which either block the lymph vessels or enter the blood stream, in which they may be



FIG. 135

Elephantiasis involving the scrotum and lower extremities of a native of India.

seen under the microscope during the day (*F. diurna*) or the night (*F. nocturna*). A patient thus infected will pass on these embryos to the mosquito which bites him, and in this way the cycle is complete. The overgrowth of the skin may be enormous, the scrotum especially being so affected that it may rest on the ground when the man is seated on a chair (Fig. 135).

**The Non-filarial Type** includes all those cases of skin hypertrophy which follow any of the above-mentioned causes of lymphatic obstruction. There is also a type which is indistinguishable from the filarial except that the parasites are not present and no cause can be found. The author has had seven cases of this type, all in women, five of whom lived in seaport towns in the South of England and in whom the legs were affected to varying degrees (Fig. 136).



FIG. 136

Non-filarial elephantiasis.

*Treatment* is most unsatisfactory. If the lymph vessels containing the parent worms can be identified, they should be excised. If the disease is limited to the scrotum, this should be amputated. Sampson Handley's method of making strands of silk act as artificial lymphatics (lymphangioplasty) has had some success in cases of brawny arm, but none in the lower extremity.

Kondoleon's operation consists in the excision of elliptical portions of skin, subcutaneous tissue and deep fascia from the outer and inner aspects of the thigh and leg; it has had some successes and as many disappointments.

## THE LYMPHATIC GLANDS

### ACUTE LYMPHADENITIS

Acute inflammation of a lymphatic gland is due to its infection with pyogenic organisms, carried to it by the lymph vessels from a septic focus within its drainage zone. Staphylococci and streptococci are the usual invaders, and as they gain entrance from the outside the primary focus is in the skin, subcutaneous tissues or mucous membranes of the upper air passages and mouth. Such a focus can usually be identified, but at times this may not be possible, either because it has already subsided or because it is too small. The lesions which give rise to acute lymphadenitis are not very serious as a rule, for the grave deep-seated and spreading infections, such as gangrene and cellulitis, do not affect the glands. If the primary focus clears up rapidly and the patient's general resistance is good, resolution will occur without suppuration, but the tendency is for the glands to soften and break down. The inflammation

will then spread to the periglandular tissues and the glands become matted together, and in some unusually virulent infections periglandular suppuration may occur, leading to a diffuse spreading cellulitis. It has been noticed that lymph glands may swell up and suppurate after an injury which has not produced any abrasion or entry for sepsis to account for the adenitis; it is assumed that organisms may lie latent in the gland and be stimulated into activity by injury.

*Symptoms* vary with the severity of the infection. The primary focus may or may not be identifiable, the intervening lymph vessels may be the seat of a typical lymphangitis or the glandular swelling may be independent of either. The glands rapidly become enlarged, painful and tender, and if they are superficial the skin over them is red, hot and cedematous, and the surrounding tissues will be infiltrated. Many of these infections subside without suppuration, but in others the swelling becomes soft in the centre and an abscess forms. The local pain, tenderness and infiltration increase, and the patient becomes progressively ill with a raised temperature and severe malaise.

*Treatment.*—The primary focus must be sought for and suitably treated. Local treatment includes antiphlogistine poultices, radiant-heat baths and short-wave therapy, and when suppuration occurs an incision must be made and drainage obtained.

**Acute Cervical Adenitis** follows sepsis in the mouth, jaws, tonsils, pharynx and teeth, when the upper deep cervical group will be affected; it also results from sepsis of the scalp (*e.g.*, pediculosis), the face (*e.g.*, impetigo) and the lips, when the superficial cervical group is inflamed. Incisions should follow the skin creases and cut across the fibres of the platysma muscle.

**Acute Axillary Adenitis** may complicate infections of the fingers and hand, lymphangitis in the forearm, boils in the axilla and breast abscesses. The infection may be superficial but more frequently is deeply situated in the axilla, and if pus ruptures out of a gland it will spread widely beneath the pectoral fascia and may reach the clavicle. Incisions should be placed midway between the axillary folds and extend from above downwards, the abscess being opened and drained by Hilton's method.

**Acute Inguinal Adenitis** is less common than the foregoing. It results from infection in the lower extremity, penis, scrotum, vulva, perineum, buttocks, anal canal and the lower part of the abdominal wall. A vertical incision is not only the safer method, but also has the added advantage that when the patient is sitting up it will gape, better drainage being thereby assured.

#### CHRONIC LYMPHADENITIS

**Chronic Simple Lymphadenitis** is more common than is generally supposed, for many chronic enlargements of lymph glands are diagnosed clinically as tuberculous, while showing no evidence of tuberculosis histologically. This condition is commonly seen in the neck following chronic sepsis in the scalp of children, and infections of the tonsils, adenoids and teeth in people of all ages. The chronic

adenitis may be due to an incomplete resolution of an acute attack, but is often the initial lesion. The affected glands are moderately enlarged, rounded, firm but not hard, only slightly tender and painful, and not adherent to each other or to surrounding structures. Suppuration is unlikely to occur and when it does the infection is primarily tuberculous. Hard shotty glands in the groin are palpable in all men and many women, and are said to be due to slight injuries, strains and muscular action whereby lymph vessels are ruptured and small resultant hæmorrhages reach the glands.

*Treatment* entails the removal of the primary cause and attention to the general health. If enlargement persists for three months after the cause has been treated, the glands should be removed.

**Chronic Syphilitic Lymphadenitis** occurs in all stages of the disease.

1. The primary sore is accompanied by an enlargement of the glands draining the area, and the reaction is usually more severe in extragenital than in genital chancres. This is particularly true of chancres of the finger and lip in which large masses of fleshy glands may arise in the axilla or neck.

2. The secondary stage is marked by a transient slight enlargement of all the glands in the body, notably those in the posterior triangles of the neck and in front of the elbow.

3. Gummatous affections of the lymph glands are very rare.

**Chronic Tuberculous Lymphadenitis** occurs commonly in children and young adults, 80 per cent. of all cases being before the age of sixteen years. It is associated with poor hygienic conditions (lack of air and sunlight) and inadequate feeding. It is probable that the glands are prepared for the planting of the tubercle bacilli by having previously been the seat of a simple lymphadenitis. The bacilli gain entrance through the upper and lower air passages, the mouth and gastro-intestinal tract and very rarely through the skin; the glands affected therefore are the cervical, mediastinal and mesenteric. The portal of entry is often a chronic septic focus in the tonsils, adenoids or teeth, but the bacilli may penetrate normal mucous membranes. The inguinal and axillary glands are not commonly affected, though the latter may be involved in a spread of infection from the neck. The pathological changes in the glands differ in no way from those described as occurring generally in tuberculosis (Chap. IV).

The first stage of the disease consists in a soft fleshy enlargement of the gland, which may reach several times its usual size, but which is otherwise normal in appearance and consistence. It is freely movable, since the inflammation is as yet confined to the gland parenchyma and there is no periadenitis. This stage is characterised microscopically by a proliferation of the lymphoid corpuscles, the presence of typical tuberculous giant cell systems and an increase in the fibrous tissue of the gland and of its capsule. Early recognition and treatment will succeed in clearing up a number of cases in this stage, but the majority will proceed to caseation and periadenitis. Caseation starts as minute yellow points of necrosis in the giant cell systems, and these gradually enlarge and finally coalesce so that the gland is converted into a simple caseous mass inside a thickened



fibrous capsule, outside which periglandular inflammation spreads into surrounding tissues (Fig. 137). The glands become matted together and firmly adherent to other structures, *e.g.*, in the neck, the internal jugular vein and sternomastoid muscle. The ultimate fate of a caseated lymph gland may be :

1. To remain unaltered for many months ;
2. To undergo spontaneous cure by a slow process of fluid absorption, fibrosis and shrinkage until only a small hard nodule remains. Such glands frequently become calcified, especially in the mesentery ;
3. To form a cold abscess by liquefaction of the caseous contents ;
4. To suppurate owing to a secondary pyogenic infection.

*Suppuration* therefore may be due either to liquefaction or to pyogenic infection. In each case pus forms in several foci, the spread and coalescence of which lead to the formation of a single cavity. Peradenitis becomes more marked than before, and several adherent suppurating glands may break down to form a large multilocular cavity. If the glands are superficial, the skin will become adherent and involved in the inflammatory process. If adequate treatment is not instituted the skin breaks down and the caseous and purulent contents of the gland are discharged. The result is either a typical tuberculous ulcer or a sinus which will not heal readily, and even when healing does occur the scar will be puckered, keloidal and vascular.



FIG. 137

Caseating tuberculous glands.

*Treatment.*—In the early stages before caseation has occurred, conservative measures should be adopted. The first essential step is the removal of any chronic septic focus which may have been the portal of entry—the teeth, tonsils and adenoids being especially scrutinised. If possible, the patient should be sent to the seaside to live an open-air life and be given good food and vitamins B and D in the form of cold-liver oil, haliverol, bemax or ergosterol. In the absence of direct sunlight, ultra-violet ray treatment is beneficial. X-rays and radium have their advocates, and the former do have a good effect in some cases, but they render any subsequent operation more difficult owing to the fibrosis they cause. During conservative treatment, the progress of the glandular swelling must be carefully watched and the situation reviewed at the end of three months.

Operative treatment consists in complete removal if: (1) The routine medical treatment has failed to achieve any improvement; (2) the disease is spreading to other glands; and (3) caseation or suppuration is present. Extensive glandular involvement is to be recognised as an expression of generalised tuberculous infection and

treated by full sanatorium regime. Operation is often postponed too long and should always be done before the skin is involved. If this has occurred, complete removal may be impracticable and the operator will have to be content with incision and curettage of all tuberculous material.

**Tuberculous Cervical Glands.**—The glands chiefly affected are the upper deep cervical group, of which the first is usually the tonsillar gland behind the angle of the jaw. The disease is frequently bilateral, and the glands become adherent to the internal jugular vein and to the sternomastoid muscle. When suppuration occurs a “collar-stud” abscess may form, the inflammatory process eroding a narrow channel through the cervical fascia and pus collecting in the subcutaneous tissues. In operating upon these superficial abscesses the surgeon must carefully seek for the opening, enlarge it and remove the deep-seated gland.

In the neck great care must be directed to the incision in order to obtain the least visible scar. The cut along the anterior border of the sternomastoid muscle gives splendid access but a poor scar, and so is to be avoided. Curved incisions following the skin creases below the jaw and above the clavicle give the best results (Fig. 138). Peradenitis may make the operation difficult, and particular care must be taken to avoid the inframandibular branch of the facial nerve, the spinal accessory, vagus, hypoglossal and sympathetic nerves. The common facial and external jugular veins will usually need to be tied, and if there appears to be serious danger of tearing the internal jugular vein, it should

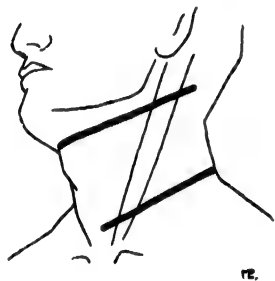


FIG. 138

Incisions for the removal of tuberculous glands in the neck.

be divided between ligatures both above and below the glands and the intervening portion removed *en bloc*. These operations should never be undertaken lightly, as the glandular involvement invariably proves more extensive than is apparent from the clinical examination; and further, the possibility of a “collar-stud” extension must never be forgotten in every superficial abscess.

Tuberculous axillary glands may be seen as the result of extension from the neck, the breast, a rib or the upper extremity. Complete extirpation of the affected glands is the most satisfactory treatment.

Tuberculous glands in the mesentery are described elsewhere (Chap. XXVI).

#### LYMPHADENOMA

**Hodgkin's Disease** is a relatively rare condition, which affects the lymph glands and the lymphoid tissue throughout the body. It occurs more frequently in men than women and usually first appears in the second or third decade of life, though no age is exempt. The cause is unknown, and considerable doubt exists as to its exact nature—either an obscure neoplasm of lymphoid tissue of the type of a malignant lymphoma or a chronic granulomatous inflammation, possibly due to

a virus infection. Its histological structure suggests the latter, while its clinical behaviour supports the former view. The disease begins in one group of glands, later spreads to the lymph glands all over the body and attacks the spleen (Fig. 139), liver, bone marrow and Peyer's patches in the intestine. The groups primarily affected are those in the neck (Fig. 140), groins, mesentery and mediastinum, and the disease may remain apparently localised for many months—or even years—before spreading widely, but in some patients the general involvement occurs rapidly and death may follow within eighteen months of the onset.

The glands themselves become enlarged and on cross-section are homogeneous, having lost all differentiation between cortex and medulla (Fig. 141). One type presents an increase of fibrous stroma, and in this the glands are hard and the disease is slow. Another type has little or no fibrosis, and the glands are soft and the progress rapid. The glands may show small grey or white spots, and the spleen often contains well-defined nodules of the same variety (hard-bake spleen). The histological picture is characterised by a relative decrease in the number of lymphocytes, an increase in endothelial cells, a well-marked eosinophilia and the presence of small, well-defined, multinucleated giant cells containing four to eight nuclei.

*Clinically* the group of enlarged glands is very characteristic. The swelling is large and soft, and even to the naked eye the individual glands appear separate. The glands vary in consistence, though they never become hard, and always remain discrete and freely movable on each other and on surrounding structures. The skin never becomes adherent and suppuration is unknown. Sooner or later the glands in the axillæ, groins, abdomen and chest are involved, and the spleen and liver become palpably enlarged. Mediastinal involvement may lead to venous engorgement and cyanosis in the neck, dyspnoea and stridor, while ascites may follow enlargement of the abdominal glands. In the later stages, especially of the more acute type, recurrent attacks of fever occur with painful swelling of the glands. Death follows from cachexia or from mediastinal pressure.

*Treatment* consists in the exhibition of arsenic and of intensive X-ray therapy. In the early stages while the enlargement is limited to one lymphatic field a radical removal of all glands in that area is considered justifiable. Such operative intervention is said to make X-ray treatment more efficacious and so improve the prognosis. As time goes on, treatment becomes less and less useful and fails to prevent a fatal issue; nevertheless, X-rays should be pushed to the ultimate limit of tolerance, as remarkable improvements do sometimes occur and many years of useful life may be preserved.



FIG. 139

Patient showing lymphadenoma of right cervical glands, right breast and spleen. Ascites is present.



FIG 140  
A large mass of lymphadenomatous  
glands.



FIG. 141  
Lymphadenomatous glands from  
the neck.



FIG. 142  
Mediastinal lymphosarcoma  
surrounding and compressing  
the heart.



FIG. 143  
Secondary carcinomatous glands in  
the neck with superadded pyogenic  
infection forming a large abscess.

## GROWTHS OF THE LYMPH GLANDS

**Lymphosarcoma** is a type of round-celled sarcoma arising in lymphoid tissue, having certain histological and clinical characters, which warrant its description as a separate entity (Fig. 142). It occurs in either sex and at any age, but especially in younger people, and is most frequently seen in the ileocaecal region (arising probably in a Peyer's Patch), the mediastinum (in the remains of the thymus), the tonsil and the cervical glands. It begins in one gland or area of lymphoid tissue, rapidly spreads by the lymph vessels to neighbouring lymph glands and, bursting through the gland capsule, infiltrates the surrounding tissues. The ileocaecal growth spreads in the submucous coat and forms a large mass with the adherent lymph glands. In the mediastinum lymphosarcoma grows rapidly to great size, invading the lungs and heart and spreading to the abdominal glands.

Lymphosarcoma forms a large, rounded, hard tumour with an irregular surface, and on cross-section presents a whitish homogeneous appearance speckled with areas of hæmorrhage and necrosis. Microscopically it bears a close resemblance to the small round-celled sarcoma, but possesses a much more abundant framework of delicate intercellular fibrils. Clinically the symptoms depend on the site of the growth, being produced by pressure on surrounding structures. Ulceration will occur if the growth is superficial (*e.g.*, in the tonsils and cervical glands), and a fungating mass will result. The progress of the disease is usually very rapid, and towards the end generalised blood stream metastases occur. Death results from toxæmia, cachexia or cardiac and respiratory failure. Treatment consists in complete removal in the early stages and intensive X-ray or radium therapy. The prognosis is quite hopeless in nearly every case.

**Carcinomatous involvement** of the lymphatic glands is common to every type of carcinoma in all parts of the body. The cells invading the lymph glands are similar in all respects to those of the primary growth, from which they have come. The gland tissue is replaced by tumour cells, which soon spread outside the gland capsule and invade glands in the same group and other neighbouring structures. Necrosis or superadded pyogenic infection will lead to the formation of an extensive abscess, the drainage of which results in a fungating and ulcerating growth on the surface. Fig. 143 shows such an abscess in the neck.

R. M. HANDFIELD-JONES.

## CHAPTER XVII

### THE FACE, LIPS AND JAWS

#### THE FACE

**D**EVELOPMENT.—Some simple explanation of facial development is necessary for a thorough understanding of the formation of hare-lip, cleft palate and other abnormalities. At about the fifth week of fetal life, the primitive cerebral vesicle extends forward to bend over the anterior end of the notochord. This medial prolongation is known as the frontonasal process. At the same time the stomodeum (primitive buccal cavity) is encroached on laterally by two pairs of processes, the maxillary above and the mandibular below, thus giving it the typical quinquiradiate appearance. The mandibular processes develop more rapidly and unite across the midline about the sixth week to form the lower jaw. The formation of the upper jaw is more complex and takes longer, not being completed until a month after this. The frontonasal process develops on each side of a median groove, an internal and external nasal process. [Between these two occurs a pouch which ultimately becomes the anterior nares, and between the external nasal and the maxillary process runs the naso-orbital fissure in which appears the primitive ocular vesicle. The upper lip is formed by the fused internal nasal (or globular) and maxillary process on either side together uniting across the midline. It will be noted therefore that the external nasal process does not take part in the formation of the free margin of the upper lip, but by its junction above this level with the united globular and maxillary processes leads to an infolding of the naso-orbital fissure, which remains in adult life as the nasal duct running from orbital to nasal cavities. The external nasal process itself forms the external part of the anterior nares. At the same time as these external changes are taking place both the internal nasal and the maxillary processes are sending in deeper subsidiary processes, the former resulting in the rudimentary premaxilla and the latter the hard palate behind and the incisor processes in front. The incisor processes meet in the midline to complete the upper jaw and bear the incisor teeth at the same time excluding the premaxilla from the alveolar margin, these small bones occupying a triangular area immediately posterior to the junction of incisor processes and anterior to the united palatal processes forming the hard palate. The limit of this triangular area is marked in the adult palate by the position of the anterior palatine canal. The reader is referred to Figs. 158 and 159 on p. 327.]

#### CONGENITAL DEFORMITIES

**Hare-lip** is a congenital cleft in the upper lip, due to failure of fusion between the internal nasal process and superficially the maxillary and deeply the external nasal processes. The term itself is a misnomer

as the hare's lip is a Y-shaped cleft, median at the lip margin and dividing above to reach each nostril. Hare-lip may be either *simple* or *complicated* (*alveolar*), either the superficial soft parts alone being involved or the alveolus also split, in which latter case the fissure extends back into a cleft palate. Hare-lip is further classified as *complete* or *incomplete*. In the former the cleft reaches into the corresponding nostril. Again it may be *unilateral* or *bilateral* or very rarely truly *median*. The unilateral and complete cases are much commoner than the bilateral and incomplete. The frequency of occurrence is approximately: unilateral complete, 50 per cent; unilateral incomplete, 35 per cent.; bilateral complete, 10 per cent.; bilateral incomplete, 5 per cent. In the unilateral cases the left side is involved twice as often as the right. The bilateral cases are usually alveolar in type, and the failure of development in the incisor outgrowths from the maxillary processes allows the premaxilla to present anteriorly. This protruding portion of bone and skin is often called the "os incisivum," and in most cases carries the central incisor deciduous teeth, the lateral incisors being on the medial limits of the imperfectly developed maxillary processes.

A flattened nose with splayed-out nostrils is typical of all cases of hare-lip, and the deformity is often associated with other evidence of maldevelopment, *e.g.*, spina bifida, talipes, syndactyly, etc. The minor degrees of hare-lip are important only in so far as they are disfiguring. The more severe forms, especially if combined with cleft palate, lead to difficulty in suckling and later in speech (Fig. 144).

*Treatment* is by operation, and this should be carried out as soon as the baby's general condition and state of nutrition permit. Weight is a valuable guide, but should not be the sole indication as to time for operation. The more the alveolar (and palatal) involvement associated with the hare-lip, the earlier should operation on the latter be undertaken, as the closure of the lip is of considerable value in effecting a partial natural narrowing of the bony defect behind. In general the operation for hare-lip should be carried out somewhere between the sixth and twelfth week of life.

Many methods are used, varying with the type and degree of deformity and the preference of the surgeon. The essential points in all may be briefly summarised. The split lip is dissected up from the maxilla on its under surface through an incision at the reflection of the mucous membrane from lip to gum. This is particularly necessary on the outer side of the cleft, and the more the flattening of the



FIG. 144

A baby with complete unilateral hare-lip and cleft palate.

corresponding nostril, the higher should this dissection be carried. The edges of the cleft are pared, and if necessary a small piece of skin removed from the floor of the flattened nostril. The two raw sides of the cleft are then united either directly (Rose's method) (Fig. 145) or by means of suitably fashioned flaps (Mirault's method) (Fig. 146). Two or three tension sutures from mucosa on one side through and back through mucosa on the other side are inserted. Buried sutures are best avoided. The red margins of the lip are then carefully approximated, and finally skin and mucous membrane sown up with interrupted sutures of very fine silkworm gut. A simple collodion or mastisol dressing or Whitehead's varnish may be used. Logan's tension bow is of great service in preventing any pull on the suture line. After-treatment should include application of padded splints to the baby's arms to prevent interference with the operation site,



FIG. 145

Diagram illustrating Rose's operation for unilateral hare-lip.



FIG. 146

Diagram illustrating Mirault's operation for unilateral hare lip.

and most careful feeding with a spoon or preferably a nasal tube. Sutures should be removed about the fourth day, but the tension bow retained for a week.

In bilateral cases the treatment of the soft parts is on similar lines, but the presence of the os incisivum requires further attention. The skin over this, pared down to a wedge shape, can usually be used in remodelling the lip. It is the treated cases of bilateral hare-lip which so often present that most disfiguring, tight, flattened upper lip with inverted red margin. For such cases Gillies' "cupid's bow" operation offers great improvement, but should not be attempted before the age of ten.

**Macrostoma and Microstoma.**—These terms are applied to the conditions resulting from either lack of normal or excessive fusion of the maxillary and mandibular processes in the formation of the cheek, resulting in either an abnormally wide or small oral aperture. Macrostoma is frequently associated with the presence of accessory auricles, microstoma with faulty development of the alveolar processes,



especially of the mandible. *Macrostoma* is treated by paring the outer portions of the cleft and suturing mucosa and skin; *microstoma* by slitting the small orifice laterally and uniting skin to mucosa.

**Facial Clefts, etc.**—(a) A FACIAL CLEFT is due to persistence of the naso-orbital fissure, and therefore replaces the nasal duct as a groove running from the outer side of the nostril to the inner canthus of the eye. It is a rare deformity and may be incomplete, involving skin only, or complete, when bone is also affected.

(b) A MANDIBULAR CLEFT is even more uncommon and is the result of non-fusion of the mandibular processes in the midline. In its simple form, with soft tissues only affected, it therefore produces a median lower hare-lip. In its complicated form there may be actual absence of bone and even an accompanying bifid tongue.

### INFECTIONS

Certain skin infections, *e.g.*, boils and carbuncles, erysipelas, lupus, impetigo, syphilis and acne, have special significance when occurring on the face. These subjects will be found fully discussed in Chaps. III, V, and XIII.

### GROWTHS AND CYSTS

The following growths may be found on the face: benign—papilloma, hæmangioma, lymphangioma and melanoma; malignant—melanoma, rodent ulcer and squamous-celled carcinoma. Of the cysts, sebaceous and dermoid are those of most frequent occurrence.

**Papilloma** may occur anywhere on the face and has the usual warty characteristics. Irritation from, for example, shaving may lead to a malignant metamorphosis, and if there is any chance of this happening excision should be advised.

**Hæmangioma.**—Both capillary and cavernous nævi are frequently found on the face and vary in size from a pin's-head to a complete involvement of one side. The smaller variety are frequently multiple. The lay names of "birth-mark" and "port-wine stain" are particularly applied to facial nævi. In the smaller types excision should be carried out where possible if increase in size is noted or disfigurement is considerable. Otherwise for the capillary variety carbon-dioxide snow application is usually satisfactory, whilst the cavernous type, which if anything favours the eyebrow and eyelid regions, may be treated with electrolysis, cauterisation or the injection of boiling water or some sclerosing solution.

Massive and extensive hæmangiomata can be adequately treated only by excision and carefully planned skin grafting.

**Lymphangiomata** are not very common, occur usually in an area just anterior to the external auditory meatus and are of the cavernous and cystic type. They are not large as a rule and should, if possible, be dissected out.

**Melanomata.**—The pigmented mole is of frequent occurrence on the face. The pigment, melanin, is found in the deeper layers of the

cutis vera and is partly intracellular and partly extracellular. It is typical of these growths that portions of them are completely un-

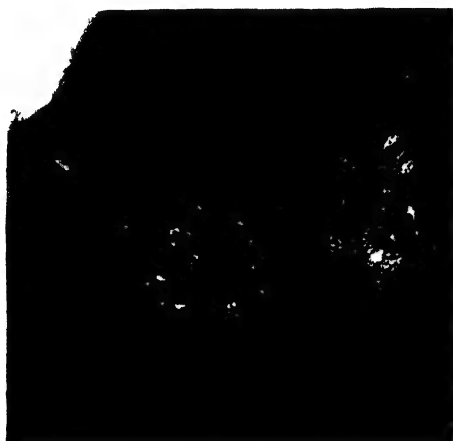


FIG. 147

A malignant melanoma of the cheek.

rapid and widespread, practically every organ in the body being affected. The secondary growths are frequently unpigmented.

Hence, although wholesale removal of all facial moles is not advocated, it cannot be overstressed that any changes occurring in a simple melanoma or any risk of constant irritation should demand immediate and radical excision.

**Rodent Ulcer.**—The face is the most common site of the rodent ulcer and the area it chiefly attacks is a triangular one, roughly bounded on each side by the outer end of the eyebrow and by the *alæ nasi*. Probably 90 per cent. of rodent ulcers occur within this area. Its features are described in detail elsewhere (p. 239). Where size and extent of deeper involvement make it possible, there is no doubt that excision is the best treatment. Failing this the use of radium (either by interstitial needles or superficial application of plaques) is preferable to deep X-ray therapy, and carbon-dioxide snow and caustics should not be used.

**Squamous-celled Carcinoma** is

of relatively frequent occurrence on the face. Starting either in a wart or less commonly in a persistent crack or fissured ulcer, it slowly

pigmented. Benign forms exist quite happily throughout life on the face and are, in fact, often cherished possessions ("beauty-spots"). But of their potential malignancy there is no doubt (Fig. 147), and very small irritative factors, often in fact unnoticed, are responsible for a change in their character. Once this has occurred they rank amongst the most malignant tumours yet known. There is usually but little increase in size of the primary growth, although areas of thickening or changes in degree of pigmentation may be noted. But lymphatic spread is exceedingly



FIG. 148

An unusually large slowly growing warty carcinoma of the face.

develops into the characteristic malignant ulcer. Rarely it forms a huge warty mass (Fig. 148). Glandular spread is late. Treatment should be by excision where possible, and failing this by radium and X-ray therapy.

**Sebaceous Cysts** are frequently found on the face. Their large central pore, fixity to skin, globular shape, painlessness and soft pultaceous feel make them easy to diagnose. Practically all the complications that may accompany sebaceous cysts (inflammation, suppuration, calcification, pedunculation, rupture and the formation of horns, adenomata and carcinomata) are also well exemplified in the face. The only satisfactory treatment is removal with subsequent careful suturing. A certain proportion can be successfully removed from the buccal aspect of the cheek, hence avoiding an external scar. Inflamed or suppurating cysts should be incised and curetted but not excised.

**Dermoid Cysts** are again of relatively frequent occurrence and can occur in any of the developmental "junction lines" of the face, *e.g.*, in the midline at the nasion or in the middle of the chin, at the outer canthus of the eye, in the line of the nasal duct from inner canthus to external nares or in the line from external auditory meatus to angle of mouth. Typically deep to the skin, fixed to deeper structures often to bone, relatively mobile, soft and painless, they are usually easily diagnosed.

If they occur in, or persist until, adolescence they should be carefully dissected out.



FIG. 149

Macrocheilia from a congenital hæmangioma.

A similar condition is seen in the tongue as a result of a congenital lymphangioma.

## THE LIPS

### CONGENITAL DEFECTS

**Macrocheilia**, or hypertrophy of the lip, is of two types, congenital and acquired. The congenital variety is a lymphangiectasis, and the typical swollen, firm, thick everted lip is further characterised by numerous small lymph vesicles. It is more commonly seen in the lower lip and is treated, if the degree of disfigurement merits it, by a wedge-shaped resection of a complete portion of the lip (Fig. 149).

The acquired variety is due to inflammatory causes and may be acute or chronic. The former occurs when acute septic conditions

of the skin of, or near, the lip are present, *e.g.*, boils, and subsides to normal once the causal lesion has been adequately treated. Not so the chronic variety, which is persistent and progressive, and may be due to the fibrosis of either tuberculous or syphilitic infections. The tuberculous type is usually seen in children or young adults, affects chiefly the upper lip and is due to a chronic lymphangitis. The lip is swollen with a firm brawny oedema, and often presents multiple fissures and cracks. It is to this type that the term "strumous lip" is applied. Syphilitic macrocheilia occurs in the tertiary stage of the disease and is typically confined to the lower lip. The hypertrophy is due to a diffuse fibrosis. Treatment of both these latter varieties is that of the causal disease.

### ULCERS

Ulcers of the lips may be simple, herpetic, syphilitic, tuberculous or malignant.

(a) **Simple Ulcers** ("cracked" or "chapped" lips) are usually due to exposure, particularly in those who are suffering from a "cold." Such patients frequently have other evidences of poor circulation, *e.g.*, chilblains. The ulcers, mainly on the lower lip, take the form of superficial and very painful fissures, which are kept open by the continual movements of the lips in talking and eating. They tend to heal spontaneously and are best treated by a simple emollient such as zinc ointment or lanoline. Stubborn and very painful fissures should be touched with the silver nitrate stick.

(b) **Herpetic Ulcers** occur on both lips but as a rule are unilateral. A series of submucous vesicles appear, usually in a patient with some catarrhal condition of the respiratory tract, and these are surrounded by small, painful, inflamed areas. The vesicles soon become purulent, burst, dry up and heal rapidly within a week or ten days from their onset. The surface application of absolute alcohol, surgical spirit, or some astringent lotion may relieve pain and hasten resolution.

(c) **Syphilitic Ulcers** are of four types—congenital (really tertiary in character), primary, secondary and tertiary. The congenital ulcers take the form of deep fissures and cracks, occurring particularly at the angles of the mouth. Secondary infection leads to further extension, and when healing ultimately takes place, radiating scars extending out into the cheek (*rhagades*) provide a very characteristic feature in the congenital syphilitic facies.

The primary ulcer (chancre) is acquired by contact infection with a secondary syphilitic or some object (*e.g.*, glasses, cups, pipe, etc.), used by him. It is rather more frequently seen on the upper lip, is a flat ulcer without the typical induration of the genital chancre, tends to involve neighbouring skin more than mucosa, and is accompanied by a more intense infiltration of surrounding tissues and a more marked glandular (submaxillary) enlargement than is seen elsewhere. (Fig. 15 on p. 65).

The secondary ulcers, on the other hand, affect mucous membrane almost entirely and are therefore more in evidence on the

inner aspects of the lips particularly near the angles of the mouth. The typical shallow elongated "snail-track" ulcers are seen with their surrounding "mucous tubercles." They are surprisingly painful.

The tertiary ulcer is essentially a broken-down gumma and has the characteristic serpiginous edge, punched-out deep crater and sloughing floor. Gland involvement is minimal.

The treatment of all types is that of the general disease.

(d) **Tuberculous Ulcers** are of two types—the fissures already described which, by their secondary infection, lead to the "strumous lip" of children, and the lupus ulcer, a progressively spreading and destructive superficial ulcer preceded by a crop of pale tubercles. General treatment is again indicated.

(e) **Malignant Ulcers** form one variety of squamous-celled carcinoma of the lip. The thickened, rolled, everted edge, indurated base and unhealthy ragged floor are characteristic. They are more commonly found in the lower lip.

Rodent ulcers may occur, chiefly on the upper lip.

### GROWTHS AND CYSTS

Benign growths of the lip include papilloma and hæmangioma; malignant growths, rodent ulcers and carcinoma. The mucous retention cyst is that most commonly found.

(a) **Papillomata** favour the lower lip, particularly the neighbourhood of the angle of the mouth. "Seeding" from one lip to another is frequently seen. They appear as the usual warty "cauliflower" excrescence, and the absence of basal induration, superficial ulceration and glandular involvement differentiates them from the papilliferous type of carcinoma. In view of the fact that they grow and multiply, and that ultimately they assume malignant characteristics, they should be excised locally.

(b) **Hæmangiomata** are seen in both their capillary and cavernous forms and show no particular preference for one lip. If small they should be excised completely; otherwise they are best treated by diathermy, electrolysis or the injection either of boiling water or some sclerosing fluid.

(c) **Rodent Ulcer** is not common on the lips and when found is usually on the upper. Starting as a hard nodule, it gradually breaks down to form the chronic non-healing ulcer, which resembles carcinoma except that it lacks the characteristic peripheral induration and accompanying glandular involvement. Such ulcers react well to radium applied either by interstitial needles or by a surface plaque, but if they are not too advanced excision is still the treatment of choice.

(d) **Squamous-celled Carcinoma** of the lip is of relatively common occurrence. It is practically limited to males, 95 per cent. of cases occurring in men. These patients are usually elderly, and it is said that nine out of ten of them have a syphilitic taint. The close relation of continual trauma—*e.g.*, the clay-pipe smoker—to the incidence of

this growth is well proved. It occurs more frequently on the lower lip and near the corner of the mouth (Fig. 150).

The growth is a typical squamous-celled carcinoma with well-marked "cell nests." It may be morphologically either papilliferous, ulcerative or infiltrative. The papilliferous type may start as an apparently simple wart or may assume malignant characteristics—induration, ulceration and secondary glandular involvement—from the first. This type soon fungates and so becomes secondarily infected. The ulcerative variety starts with induration in an already existing fissure. The infiltrative type shows no superficial ulceration, but a generalised thickening and hardening of a part of the substance of the lip. It is not common.



FIG. 150

A squamous-celled carcinoma of the lip at the angle of the mouth.

All malignant growths of the lip are characterised by their relatively slow rate of advancement. Glandular involvement is late, after about a year, and not marked until the primary growth is very advanced. The glands affected are the submaxillary and submental, with extension to the upper deep cervical in the later stages. Secondary infection leads to matting of the groups of glands and ultimately in the worst cases these break down, fungate on the surface and may lead to the patient's death from exhaustion due to pain and toxæmia secondary to widespread sepsis or hæmorrhage. Generalised metastases are exceptional.

*Treatment.*—Any indurated area in the lip, be it warty or ulcerated, should be regarded with suspicion, and a biopsy performed.

If operable, the primary growth should be treated by excision, a wide wedge of the lip being removed.

These growths react well to radium applied either by interstitial needles, surface plaques or teletherapy with a bomb, but when excision is possible it is preferable. To treat a carcinoma of the lip adequately with radium means, automatically, considerable danger to jaws and teeth. The average dose is in the neighbourhood of 2,000 mg.-hours.

Secondary glands should always be dealt with whether clinically affected or not. This is preferably done at the same time as the excision of the primary growth. Radium (preferably bomb therapy) can be used in the inoperable cases, although as a rule alleviation of pain and temporary regression are all that can be expected.

(e) **Mucous Cysts** of the lips occur on the inner aspect, more commonly of the lower lip. They are retention cysts of the small

mucous glands and are due to fibrosis of the opening of the duct, probably the result of trauma, *e.g.*, biting the lips. They present as small, tense, smooth swellings, globular in shape and typically painless. They contain a thick glairy mucoid fluid.

*Treatment* is excision.

**FACIAL NERVE** (Paralysis, Spasm or Tic, Neuralgia).—*Vide* Chap. XLIII.

## THE JAWS

### FRACTURES

**The Maxilla** is usually fractured by direct violence and this is often accompanied by damage to nasal, malar and lachrymal bones. The majority of these fractures are compound as, even in the absence of overlying skin injury, they open into various cavities of the mouth, nose and accessory air sinuses. Thus there is always considerable risk of sepsis with secondary bone necrosis. In the absence of this complication union is rapid and treatment is conservative and symptomatic. If depressions in the neighbourhood of the zygoma are likely to be disfiguring, they should be elevated by open operation.

**The Mandible** is again usually fractured by direct violence, but indirect violence, *i.e.*, a force, applied to one part or side of the lower jaw, producing a fracture in another part of the opposite side, accounts for a definite percentage. Pathological fractures due to underlying bone disease are relatively common.

The mandible can be fractured at the following sites :—

(a) **BODY**.—This is usually due to direct violence, and occurs through the weakest part of the bone at the level of the canine fossa, just anterior to the mental foramen. Unless bilateral, when the separate middle fragment is drawn markedly downwards and backwards, displacement is not usually very great. The outer fragment tends to be pulled upwards and slightly backwards. Owing to the firm attachment of the buccal mucous membrane to the alveolar periosteum, practically all these fractures are compound.

(b) **ANGLE**.—Another direct-violence fracture, the line of cleavage passes up obliquely from the junction of body and ascending ramus to the alveolar margin either in front of, or behind, the last molar tooth. If complete they are again usually compound. Displacement is minimal.

(c) **ASCENDING RAMUS**.—Usually the result of direct violence, not compound, and, owing to the mass of muscle on both internal and external aspects, shows practically no displacement.

(d) **NECK OF CONDYLE**.—More usually is due to indirect violence, not compound, although the temporo-mandibular joint may be involved, and shows a typical deformity. The small upper fragment is drawn forwards and slightly inwards by the action of the external pterygoid

muscle, the remainder of the jaw being displaced to the side of the injury.

(e) **CORONOID PROCESS.**—Fractured either by direct violence or muscular traction, is pulled upwards by the temporal muscle.

*Diagnosis* of mandibular fractures is usually simple. The history of injury with subsequent pain, swelling, discoloration and loss of function (opening and closing the mouth) make a characteristic picture. Crepitus is easily elicited in the common fracture of the body, and irregularity in the line of the teeth with displacement of the fragments of the jaw clinches the diagnosis. The pain at first is considerable and is probably due mainly to tension of the traumatic effusion beneath the closely attached alveolar mucosa rather than to involvement of the inferior dental nerve and its branches.

*Treatment* should be instituted as early as possible and should in no way deviate from that of fractures elsewhere, *i.e.*, it should consist in reduction, adequate immobilisation and maintenance of function of surrounding parts. But it cannot be too emphatically stressed that the advice and co-operation of a dental surgeon should be enlisted from the first in any except the simplest cases.

As a general rule loose teeth and those involved in the fracture line should be removed. Their presence is always an open invitation to sepsis. The one exception to this is in the case of the last molar, which may afford the only means of fixing the posterior fragment in fractures extending up to the alveolus from the angle.

Reduction, if necessary, should be carried out under full anaesthesia. Immobilisation may be obtained by the following methods :—

(a) **Bandages.**—The “four-tail” bandage, which slings up the point of the chin, the ends being tied, two over the vertex and two just above the external occipital protuberance, is still the commonest method, and all that is needed in simple fractures without much displacement. It has the great disadvantage of dragging the anterior fragment backwards, *i.e.*, increasing the deformity; the Barrel bandage, which goes under the chin, well back, and is attached above to a circular band round the forehead and occiput, is definitely preferable, the pull here being upwards and forwards.

(b) **Splints.**—Specially fitted splints of celluloid, leather, rubber or even light plaster of Paris can be moulded to the external aspect of the mandible and attached by cords, rubber bands or bandages to the vertex.

(c) **Dental Splints.**—These can be made either of dental wire, vulcanite or malleable metal. They are fitted over all the lower teeth, holding them firmly together, and may, if necessary, be fixed by bars between the lips to some external form of splintage. They are used in cases where displacement is not too great and where reduction can be easily maintained.



(d) **Interdental Splints**, especially in any bad fracture, are preferable to the above. They are made either from malleable metal or wire, the principle being to lock the fragments of the lower jaw using the available teeth of both as supports. In the methods where wire is employed the "eyelet" scheme, which is standard in America, is probably the best.

Immobilisation should be maintained for at least three weeks and longer in difficult cases. In uncomplicated fractures union is relatively firm in three weeks. During this period maintenance of function is achieved by breathing exercises and the sucking movements necessary in taking food, which must of necessity, when the jaws are fixed, be of a fluid nature. It is usually taken through a tube inserted either through a gap in the teeth or behind the last molar tooth. At the same time constant mouth-washes of some mild antiseptic will prevent the advent of serious sepsis.

Delayed union, non-union and mal-union are all relatively frequent in mandibular fractures. Cases of non-union should, after sepsis has been adequately dealt with, be treated by surgical measures, of which bone grafting, using either a rib or preferably the iliac crest, is better than any metallic fixation.

### INFECTIONS AND NECROSIS

Infection of the jaws and subsequent necrosis may result from any of the undermentioned causes :—

#### 1. Osteomyelitis :

(a) General blood-stream infection.

(b) Local infection . . . . . { i. Tooth infection.  
ii. Tooth extraction.  
iii. Mouth infection.

2. Compound fractures.

3. Post-exanthematous.

4. Mercury, arsenic, antimony or phosphorus poisoning.

5. Syphilis.

6. Tuberculosis.

7. Actinomycosis.

8. Radium "burns."

9. Neoplasms.

Certain of these require further consideration, but a few general facts have a bearing on all types, and the clinical picture of the group, as well as the principles of treatment, are common to all. Infection and necrosis are definitely more common in the lower jaw. Two possible causes are advanced to account for this fact. Firstly, its blood supply (mainly by the bone-enclosed inferior dental artery) is not so good as that of the upper jaw, and secondly, its position prevents it having the natural advantages of gravity drainage possessed by the maxilla. As a compensation for these drawbacks, however,

the lower jaw ultimately heals much better than the upper, with the formation of strong new bone. The upper jaw seldom regenerates beyond the stage of firm fibrous tissue.

The first signs of jaw infection are usually vague pain in the teeth and a disinclination to close the jaws tight. Pain soon becomes more intense and is accompanied by marked swelling of the jaws, face and neck. The temperature rises rapidly, often with rigors, and the pulse rate follows it. There is inability to open the mouth, either because of the degree of swelling or from reflex spasm. Rapidly abscesses form and discharge through multiple sinuses, either into the mouth or to the exterior. The breath is typically most offensive, the teeth become loose and excessive salivation is a constant source of worry to the unfortunate victim. The acute stage—if not sufficiently severe to kill the patient as it occasionally is—passes off, leaving the many discharging sinuses, and behind them sequestra are slowly formed. From these factories of sepsis, poison is also absorbed to the blood stream, and hence toxæmia, chronic dyspepsia, septic pneumonia and amyloid disease are all frequently seen.

*Treatment* in the early stages consists in local fomentations and mouth-washes. Later incision, sequestrectomy when indicated by X-rays, and irrigation of resultant cavities with mild antiseptics form the usual line of treatment, together with attempts to sustain and build up the patient's general health and resistance.

**Osteomyelitis.**—(a) The general blood-stream infection is usually due to spread from a staphylococcal focus elsewhere, is typically very acute and often involves completely one or both sides of the mandible. A most interesting form of the condition is seen in new-born infants. It has even been reported as occurring *in utero*.

(b) Local infection is most commonly derived from dental sepsis, resulting in the formation of an alveolar abscess (p. 322). Infection may also be introduced locally following dental extractions, either from the use of (bacteriologically) dirty instruments or from surrounding sepsis in mouth and gums. One of the worst offenders in this latter respect is the impacted or unerupted third molar (wisdom tooth), whose socket is often infected even previous to extraction. Such advanced septic conditions of the mouth as cancrum oris (*q.v.*, p. 176) can easily spread to the jaws and produce a necrosis of bone.

**Compound Fractures** always result in some degree of bone necrosis, in uncomplicated types only molecular, but in comminuted cases or in the presence of gross dental or buccal sepsis it is likely to be extensive.

**Post-exanthematous.**—Necrosis of the jaw, usually mandible, is one of the less common sequelæ of the specific fevers, but cases have been reported following measles, chicken-pox, smallpox, scarlet fever and typhoid. The condition is a secondary osteomyelitis predisposed to by the patient's lowered general resistance.

**Chemical Poisons.**—Mercury, arsenic and antimony are to-day seldom given in doses sufficiently large to produce a necrosis, although in the treatment of syphilis one still relatively frequently finds patients

who seem to have some particular idiosyncrasy to these drugs—especially arsenic—and as a result of their use develop a very acute stomatitis which, in the presence of dental sepsis, may lead to secondary infection of the jaw.

Phosphorus necrosis is not seen to-day, but pathological specimens resulting from fatal phosphorus poisoning are still seen in museums. This change has been brought about by the replacement of yellow phosphorus in the manufacture of matches by amorphous phosphorus. The fumes of the yellow phosphorus gained admission to the substance of the jaws (both were affected, the lower a little more frequently) through the roots of a carious tooth, thus setting up an acute inflammatory reaction. The sequestra were very characteristic, being grey in colour and porous in texture. Hence their description as "pumice-stone" sequestra. They were extremely slow in separating, and during the process reactionary periostitis was much in evidence.

**Syphilis.**—Tertiary syphilis, usually in the form of a diffuse gummatosis, is prone to attack the maxilla, particularly its palatal portion, and the consequent necrosis leads to one variety of perforated palate.

**Tuberculosis,** in the form of lupus, may spread either from skin or mucous membrane to the jaws, more particularly the upper. Again, the bony palate is the favourite site of attack.

**Actinomycosis.**—The streptothrix of actinomycosis (or "ray fungus") attacks the jaw region more frequently than any other part of the body. It gains entrance through either the root of a carious tooth or some abrasion of the alveolar mucous membrane, leading to a chronic osteomyelitis with necrosis. The mandible is much more frequently affected than the maxilla. Clinically, the widespread induration and superficial discoloration, the multiple sinuses and the typical "sulphur-granule," seropurulent discharge all go to form a characteristic picture. This and the more detailed morphology of the streptothrix are described more fully elsewhere (*vide* p. 48). In the jaw the differential diagnosis from sarcoma may sometimes be very difficult.

**Treatment.**—Cervico-facial actinomycosis responds favourably to X-ray therapy, which should achieve a complete cure.

**Radium.**—The use of radium in the treatment of cases of carcinoma of tongue, cheek and lip will, unless the neighbouring bone be carefully shielded by lead, give rise to a very chronic and intractable necrosis of the jaws. This is accompanied by constant and severe pain, and nothing can be done except await the formation of the inevitable sequestra. Unfortunately this process is often much delayed, and two years or more may elapse between the date of the application of radium and that of possible sequestrectomy.

**Growths.**—Malignant growths of the jaw, if left untreated, ultimately break down, probably due to the relatively poor blood supply and ulcerate to the surface, when secondary infection from mouth and teeth may lead to a very extensive necrosis of bone. This is especially true of the mandible.

### LEONTIASIS OSSEA

This disease is limited to the facial and cranial bones. Those chiefly affected are the maxillae, nasal bones and mandibles. The etiology is unknown. Attempts to establish a causal relationship to rickets, syphilis and tuberculosis have all failed. But the pathology of the condition — a “creeping periostitis” leading to approximately symmetrical hyperostosis — is very suggestive of a chronic low-grade infection as being the underlying cause. The fact that many of the cases appear to start in the neighbourhood of the ethmoid may point to the nasal cavity as the source of infection.



FIG. 151

Leontiasis ossea.

exostoses consist of soft spongy surgically in the milder cases, but this, unfortunately, only affords temporary relief. No cure is possible.

*Osteitis fibrosa* is not uncommon in the lower jaw, and in its clinical picture closely resembles an epithelial odontome (p. 323), but its X-ray appearances are characteristic (Fig. 152). Treatment is local removal in the early stages, but later complete excision of that part of the jaw will be necessary with subsequent replacement by bone grafting.

The disease occurs usually in young adults and is progressive. The massive outgrowths of bone on the face (Fig. 151) lead to a most hideous appearance, and ultimately produce pressure symptoms in various localities, as evidenced by nasal obstruction, lachrymation, exophthalmos, neuralgic pains and finally coma from cranial extension. The bone. These can be removed



FIG. 152

X-ray appearance of osteitis fibrosa of mandible.

### GROWTHS OF THE JAWS

Both benign and malignant neoplasms are of relatively frequent occurrence in the jaws. Amongst the former are osteomata, chondro-

mata, lipomata, odontomata, fibromata and osteoclastomata, whilst malignant growths include sarcomata, carcinomata and Ewing's tumour.

**Benign.**—**OSTEOMATA.**—Both the "ivory" and cancellous forms are found. Some pathologists include the hyperostosis of leontiasis ossea amongst the latter, although they occur in their more characteristic form, usually in the neighbourhood of the symphysis menti. The ivory type of osteoma either grows from the maxilla into the antrum of Highmore or from the neck or condyle of the mandible. Any of these growths, in particular the last group, may give pressure symptoms on major nerves or produce obvious deformity.

*Treatment* is surgical removal.

**CHONDROMATA** are rare, occurring chiefly in the mandible, either at the symphysis or the region of the condyle. If giving symptoms or if disfiguring they should be removed.

**LIPOMATA** are also uncommon. They occur subperiosteally in relation to the alveolar margin of the mandible, and are frequently not correctly diagnosed until after removal.

**ODONTOMATA** are tumours derived essentially either from the primary or secondary tooth germs. They are fully described on p. 323.

**FIBROMATA** form one type of "epulis," and are further discussed on p. 321.

**OSTEOCLASTOMATA.**—These tumours were formerly classed as "myelomata." Two types of osteoclastoma are found, almost exclusively in the mandible, one occurring in the gums, the other in jaw-bone proper. Further consideration of clinical signs, pathology and treatment will be found on p. 321.

**Malignant.**—**SARCOMATA** occur chiefly in the upper jaw and may originate in the maxillary antrum, the nasopharynx or the sphenomaxillary fossa. They are more commonly found in younger people and even in children. The pathological types vary—spindle-celled and round-celled predominating. A very undifferentiated growth corresponding to Ewing's tumour has been reported. The so-called "recurrent epulis" is usually a fibrosarcoma and favours the mandible. Secondary sarcomata are rare.

**CARCINOMATA** again more frequently attack the maxilla than the mandible. In the case of the latter, the growth is usually an extension from the tongue, floor of mouth, lips or gums. A certain proportion of maxillary carcinomata appear to start in the ethmoidal air cells, but the majority originate in or near the maxillary antrum. Various cellular types may be found, the commonest being cubical-celled, the growth being an adenocarcinoma. Occasionally these cells undergo metaplasia, with the result that a squamous-celled carcinoma is found. More rarely proliferative papilliferous types occur, and even primary basal-celled carcinomata ("rodent ulcers") have been described. Despite their marked local effect, these growths metastasise comparatively late in the disease and then usually to the submaxillary lymphatic group first.

Secondary carcinomatous deposits are infrequently found in the jaws, but primary growths of breast, prostate, kidney and thyroid sometimes metastasise to these bones—usually the mandible.

*The Clinical Picture and Treatment* of all types of malignant neoplasm of the jaws may be considered together—as exact diagnosis in primary growths is usually only possible pathologically. In the upper-jaw growths there is typically a preliminary clinical stage when the actual growth itself is not obvious, but produces facial pain either of a neuralgic type from involvement of branches of the Vth cranial nerve or boring in character and worse at night from internal expansion of bone. With this pain there may be a purulent discharge from the nose, occasional epistaxis or polypi presenting at the anterior nares. One of the first complaints may be loose teeth. Later an obvious bulging of the cheek can be observed, and as the growth spreads exophthalmos appears from pressure on the floor of the orbit, downward bulging of the hard palate may be visible and obstruction of the nasal duct leads to epiphora. The affected side of the face will be dull on oral transillumination, and an X-ray examination may show an opacity in the antrum or a characteristic shadow in the facial bones. All the signs and symptoms are more exaggerated when the growth originates in the maxillary antrum than when it spreads into the antrum from neighbouring regions. Ultimately fungation and ulceration occur, with the accompanying possibility of massive hæmorrhage, and the growth appears on the surface either through the skin of the cheek or the mucosa of mouth or nasopharynx. It is usually only in this final stage that glandular involvement becomes obvious.

In the lower jaw malignant growths are clinically of two types—*intra-osseous* (the sarcomata and secondary growths) and *superficial ulcerative* (the carcinomata spreading from surrounding soft parts). In the latter the characteristic malignant ulcer, with its rolled, everted, indurated edge and irregular, bleeding, unhealthy floor is easily recognised. Starting in the alveolar periosteum, it soon invades and destroys the underlying bone, leading to a foul, sanious, purulent discharge in which spicules of bone are frequently found. Until the later stages pain is not marked.

In contradistinction to this the *intra-osseous* type gives the boring pain of pathological bone expansion and, as in the upper jaw, looseness of the teeth may be complained of. Ultimately an obvious swelling occurs with breakdown of the growth either to the exterior or to the mouth.

*Treatment* of malignant growths of the jaw consists in excision when possible, followed by radium or X-ray therapy, and for inoperable cases in these latter methods alone, together with the administration of suitable drugs to alleviate pain.

Complete excision of the upper jaw is to-day very rarely deemed necessary. A growth which demands such radical treatment is essentially inoperable and should be dealt with by radiotherapy. In operable cases Mouton's method ("lateral rhinotomy") or some modification or extension of it is the one usually adopted. This involves an incision from the inner end of the eyebrow on the affected side, passing down alongside the nose, turning round the ala and going through the midline of the upper lip. This flap is turned back

and the soft tissues separated from the underlying bones, which are then removed to the extent required either to eradicate the growth or fully expose it in the antrum. It is obvious that for growths involving the floor of the antrum or palate this method is not applicable. It has the great advantage of leaving little residual deformity.

Complete excision of one-half of the mandible causes considerably more residual deformity, and whenever possible the ascending ramus is preserved. This allows subsequent bone grafting, which gives surprisingly good functional and cosmetic results (Fig. 153).

In both these operations hæmorrhage is unexpectedly slight. Suturing should be done by interrupted stitches, particular care being taken to obtain accurate approximation of the red margin of the lips. Drainage is always necessary as is pre-operative attention to mouth sepsis and the use of intratracheal anæsthetic, to allow the pharynx to be plugged during operation.

**Maxillary Antrum.**—Apart from the involvement of the maxillary sinus in growths of the upper jaw, as described above, a more detailed consideration of diseases affecting this cavity will be found in Chap. XXI.



FIG. 153

Result following excision of ramus of mandible with subsequent grafting.

## TEMPORO-MANDIBULAR JOINT

### DERANGEMENTS

**Dislocation.**—The temporo-mandibular joint is a diarthrosis having two complete synovial cavities separated by a fibrocartilaginous disc. Dislocation of this joint is not common, and when it occurs is practically always forward. The very rare backward and upward dislocations, the former accompanied by fracture of the tympanic plate and the latter by fracture of the middle cranial fossa, require no further description. In the typical forward dislocation the mandibular condyle rides forward over the eminentia articularis to lie in the zygomatic fossa and the intra-articular cartilage, owing to the attachment to it of the external pterygoid muscle, is also carried anteriorly. Dislocation may be unilateral or bilateral and is caused either by trauma (*e.g.*, blows on the chin with the mouth open, excessive pressure from dental gags, or during operations inside the mouth) or by the exaggeration of such actions as laughing, yawning or even biting.

The condition is easily recognised. The mouth cannot be shut—a permanent gap of about an inch remaining between upper and lower teeth. In unilateral dislocation the point of the jaw is pushed across to the unaffected side. There is an easily palpable abnormal depression in front of the ear. Jaw movements are very limited, speech and deglutition are interfered with and salivation is excessive.

Reduction is usually easily effected by pressing the lower molars



downwards and backwards with the well-guarded thumb inside the mouth. After reduction a four-tail bandage, such as is used for mandibular fractures, should be worn for a week. There is little tendency to recurrence.

**Subluxation.**—Subluxation of the joint—also called “clicking” or “locking” of the jaw—is due to a looseness of the intra-articular cartilage. This cartilage is dome-shaped, to increase the height of the condyle of the mandible, and to its anterior border is attached the tendon of the external pterygoid muscle. If the cartilage is at all loose, the pull of this muscle drags it forward so that it becomes pinched between the condyle and the eminentia articularis, when the mouth is opened. The cartilage itself is rarely torn, but its catching in this position may lead to “locking” of the joint or in more chronic cases to an audible and palpable clicking. Pain is in the early stages experienced locally and in the pinna, to which it is referred via the stretched auriculotemporal nerve. The joint can usually be easily freed by lateral movements of the jaw, but in the frequent recurrent cases and in those in which the clicking interferes with talking and eating, excision of the cartilage must be considered.

Very rarely subluxation may result from the locking of loose bodies in the joint—usually the accompaniment of osteo-arthritis.

**Trismus** is a general term descriptive of the condition of inability to open the mouth. This may be due to a variety of causes which can be briefly summarised as follows :—

1. **ANKYLOSIS OF THE TEMPORO-MANDIBULAR JOINT.**—This may be either fibrous or osseous and results from arthritis due to such organisms as staphylococci, streptococci, gonococci, pneumococci, typhoid and tubercle bacilli. In an established case, treatment consists in forming a false joint, either by excision of the head or of a portion of the neck, followed by the insertion of a muscle graft from the internal pterygoid or masseter. In such operations the danger of injury to the parotid gland, facial nerve and middle ear must be remembered.

2. **EXCESSIVE BONE FORMATION** in the neighbourhood of the joint. This may be due to the callus of fractures, articular proliferation following dislocation, osteophytes of osteo-arthritis or osseous neoplasms, particularly of the maxilla.

3. **MUSCULAR SPASM**, in such conditions as tetanus, hysteria or reflex irritation from acute arthritis of the temporo-mandibular joint, carious teeth and impacted wisdom teeth.

4. **EXTERNAL SCARRING** in the neighbourhood of the joint resulting from bad burns, lupus, operations, cancerum oris and the application of radium. Myositis ossificans of the masseter muscle may be included under this heading. Treatment in this type of case may rarely be possible by complete excision of the causative scar. More usually an arthroplasty is required, the accepted method being that of Esmarch, in which a wedge of the ascending ramus in the region of the angle is excised, the apex of the wedge pointing towards the alveolus and a muscle graft inserted into the gap. Occasionally the whole ascending ramus has been excised.

5. **NEIGHBOURING ACUTE INFLAMMATIONS** (e.g., parotitis, both



mumps and pyogenic, Ludwig's angina, lymphadenitis, alveolar abscess, tonsillitis and stomatitis). The chronic fibrosis of actinomycosis may also be a cause.

6. NEIGHBOURING NEOPLASMS—more particularly those of the parotid and maxilla.

### INFECTIONS

**Acute.**—1. **SYNOVITIS.**—This is typically an accompaniment of rheumatic fever, and its treatment as such is symptomatic. Other infective fevers, *e.g.*, scarlet fever, rarely give an acute synovitis of this joint, with a tense serous effusion and trismus.

2. **ARTHRITIS.**—This is found as a complication of gonorrhœa, a non-suppurative type which requires only local conservative treatment, and in pyæmia. In these latter cases the local signs of inflammation are well marked, and the general reaction is usually correspondingly severe. Pus in the joint demands incision and drainage. Very frequently this results in an ankylosis, and subsequent excision of the condyle may be necessary. A suppurative arthritis may also result from direct extension of pus from the parotid or more rarely in children from the middle ear.

**Chronic.**—1. **SYNOVITIS.**—This is most commonly the outcome of repeatedly recurring subluxation.

2. **ARTHRITIS.**—(a) Osteo-arthritis is relatively frequent in this joint and is usually symmetrical. The mandibular condyle becomes flattened, enlarged and eburnated, and both the articular and intra-articular cartilage tend to disappear. Pain, crepitus, loss of movement and deformity make up the clinical picture. In the typical bilateral cases the chin becomes pushed forward, in unilateral cases to the unaffected side. Loose bodies may be formed and lead to locking of the joint. Treatment by physiotherapeutic and medicinal methods may give temporary relief, but in severe cases excision of the condyle should be seriously considered.

(b) Tuberculous arthritis is quite well recognised in this joint, both synovia and bone being attacked. The clinical resemblance to osteo-arthritis makes differential diagnosis very difficult, unless other features of the case point to a specific etiology. As immobilisation of this joint is virtually impossible, earlier operation than is usual in other examples of joint tuberculosis should be undertaken to prevent sinus formation with its inevitable secondary infection.

### GUMS

#### HYPERTROPHY

This is a condition of fibrous overgrowth of the gums usually seen in young children. It is practically always associated with the eruption of carious teeth, and the patient is usually a weedy underdeveloped infant often mentally deficient. The gum hypertrophy is very irregular, the changes frequently being restricted to one side. Mastication is interfered with, salivation is excessive, an external swelling may be obvious and bleeding is common. The condition is very chronic, and

treatment may have to be repeated many times. This consists in paring off the excess gum, preferably by diathermy, and extracting obviously carious teeth. In resistant cases a small slice of underlying alveolar bone should also be removed.

### INFECTION OF THE GUMS

**Spongy Gums.**—This term, which is in itself descriptive of the œdematous, easily bleeding, possibly ulcerated state of the gums seen in this condition should be restricted to those cases, in which, despite the secondary infection which inevitably occurs around the roots of the teeth, the underlying etiology is either a fault in general nutrition (*e.g.*, scurvy and rickets) or a local chemical irritation (*e.g.*, overdosage of mercury or phosphorus poisoning). The treatment is essentially that of the underlying cause, combined with the local use of astringent and antiseptic mouth-washes.

**Gingivitis.**—Bacterial infection of the gums may be specific or non-specific. Of the former, mention may be made of tuberculous and syphilitic types, both of which arise by extension from the mucosa of mouth or tongue, and of Vincent's spirillum infection. The non-specific form is more commonly called *pyorrhœa* or Rigg's disease. All types of gingivitis are primarily due to lack of oral cleanliness. This in due course leads to a deposition of tartar on the gums around the crowns of the teeth, and in this tartar bacteria find a most suitable nidus for development. Another very common accompaniment is the habit of mouth-breathing which, by drying up the buccal mucosa, prevents the natural salivary currents from cleansing teeth and gums.

The commonest causal organism in *pyorrhœa* is the streptococcus, which gains entrance either via the coating of tartar between the gums and the crowns of the teeth, the infection slowly spreading through the periodontal membrane to the periosteum of the alveolus, or through a carious tooth via the pulp cavity to the root, leading to a periapical infection, a change which can be accurately recognised by X-ray examination. The condition affects adults and is very chronic, the resultant absorption of toxins being responsible for many generalised infective conditions of the body. The gums are discoloured, congested and bluish, they bleed easily, are œdematous and tender and ultimately ulcerate. This leads to the formation of mucosal pockets, in which suppuration takes place and from which pus can be squeezed. Later fibrosis occurs and the gums are retracted from the teeth, which become exposed and frequently fall out. The breath is typically offensive.

The importance of the prophylactic use of the tooth-brush cannot be overstressed. Once the condition is established, in the early stages scaling of the teeth and the use of suitable mouth-washes (*e.g.*, hydrogen peroxide) may effect a cure, but if advanced either dental extraction or the operation of gingivectomy offers the only hope of clearing up the condition. An autogenous vaccine may be used to advantage.

If the infection passes through a carious tooth to the apex of its fang a small localised abscess cavity is formed in the substance of the alveolus. Such an *alveolar abscess* by pressure on surrounding bone

leads to osseous absorption, and unless the pus has free outlet through the infected tooth socket, the abscess will slowly track through the jaw and present externally. This pointing usually occurs on the outer side of the jaw, the bone on this side of the alveolus being thinner. In this way a "gumboil" is formed. More rarely an abscess may break through the palate or into the maxillary antrum. The condition is accompanied by severe pain of a throbbing character, and the general reaction, with high temperature, is marked. The face in the neighbourhood of the abscess is swollen, the affected tooth or teeth are loose and tender, a certain degree of trismus is usual and the glands draining the area are enlarged and painful.

*Treatment* may be efficiently achieved in some cases simply by extracting the offending tooth, but more often incision of the gum and drainage of the cavity are required. Antiseptic mouth-washes should be used freely after either treatment. Even so, some residual infection is not uncommon, and a chronic bone cavity, discharging small sequestra via an external sinus, is formed. This requires more radical exposure and the establishment of efficient drainage.

### GROWTHS OF THE GUMS

Growths of the gums may be either innocent or malignant. The former are relatively common, the latter as primary growths rare.

**Innocent Growths.**—**PAPILLOMA.**—This is a small wart-like growth springing from the gums near the teeth. The molar region of the lower jaw is that most frequently affected.

*Treatment* consists in local excision.

**EPULIS.**—Two types of epulis are described, the fibrous and the myeloid. The former is by far the commoner.

*The Fibrous Epulis* is a fibroma springing in most cases from the periosteum of the alveolus. It is slow-growing and presents clinically as a smooth, often lobulated swelling on the outer aspects of the gums, usually of the lower jaw. Increase in size often takes place inwards between the teeth, which may become completely hidden by the growth. This hypertrophic form is sometimes called a "polypus" of the gum. The association with dental caries is very constant, but the growth itself is painless and the only symptoms are those due to its size, which may be sufficient to interfere with mastication. Treatment consists in local removal, together with the extraction of contiguous teeth if these are infected. Recurrence is not uncommon, in which case a small V-shaped piece of alveolus should be removed in conjunction with the growth.

*The Myeloid Epulis* is a giant-celled tumour of the alveolar margin involving the gums. It is similar pathologically to the endosteal osteoclastoma of the jaw (p. 315), but its characteristic position makes it a clinical entity. It grows rapidly and either expands the alveolar bone, giving the typical "egg-shell crackling" feel when the outer plate of bone is sufficiently thinned, or presents on the gum margin as a soft, dark-red swelling which very soon tends to ulcerate.

*Treatment* involves opening the growth cavity, scraping and painting the walls with carbolic or zinc chloride. As recurrence is

common, this treatment should be followed by suitable radiation therapy. Many surgeons prefer as a primary measure to excise a portion of the alveolus with the tumour.

**Malignant Growths.**—Squamous-celled carcinomata, round or spindle-celled sarcomata and melanomata have been reported as originating in the gums, but, except as extensions from neighbouring parts, malignant growths are rare. Excision where possible or, failing this, irradiation is the treatment of choice.

## TEETH

**Development.**—The teeth are ectodermal in origin, being derived from downgrowths of the buccal epithelium into the mesoderm of the alveoli. Man has two complete dentitions. The deciduous or milk teeth erupt at the following approximate dates: central incisors, 6 months; lateral incisors, 9 months; first molars, 12 to 15 months; canines, 18 months to 2 years; second molars, 2 years. This dentition is replaced, in girls earlier than in boys, by the permanent teeth which erupt as follows: first molars, 6 years; central incisors, 7 to 8 years; lateral incisors, 7 to 8 years; first premolars, 9 to 10 years; second premolars, 10 to 11 years; canines, 10 to 12 years; second molars, 12 years; and third molars, 18 to 22 years.

## INFECTIONS

**Dental Infection** has to some extent been discussed in the consideration of pyorrhœa (p. 320). The causal organism is usually the streptococcus, and this gains entry to the tooth via a carious surface on the crown. Infection spreads through the pulp cavity giving necrosis, this process being responsible for "toothache." Relief of pressure in the pulp cavity occurs via the apical foramen at the root, and the subsequent course of events depends upon the virulence of the attacking organisms. If this is marked, bone infection is rapid and an alveolar abscess is formed (p. 312). In milder cases a small periapical bone necrosis occurs, with a resultant chronic abscess cavity. These can be recognised radiologically, and their importance as a source of generalised chronic systemic toxæmia cannot be overstressed.

Clinically, following the initial acute toothache, the affected tooth remains tender on pressure, and this with or without accompanying pyorrhœa of the gums points to periapical infection, a diagnosis which can usually be confirmed by X-rays. Treatment consists in extraction of the affected teeth, but a warning should be given against doing this in a wholesale manner. So much toxin may be freed thereby as to render the patient acutely ill.

**Dental Cyst.**—Dental cysts are discussed under the heading of teeth infections because of their invariable association with dental caries, but it would seem that they are not, as was previously thought, simply a chronic abscess cavity, but rather a unilocular type of odontome, in which the irritation of chronic sepsis has initiated changes in the remains of the enamel organ. Their relation to a dead tooth or root is constant. They are usually quite small and often

unrecognised until their presence makes extraction difficult. They are more common in the upper jaw and, in general, dental cysts of the maxilla are larger than those of the mandible. The region of the upper molars and bicuspid is that most frequently affected. When recognisable clinically they produce a painless expansion of the jaw, equal in all directions, and ultimately, when the overlying bone becomes thin enough, giving a characteristic "egg-shell" crackling. Expansion may proceed even further than this, so that a smooth fluctuant swelling is found in the jaw around which a sharply defined bony edge is easily palpable. The cysts contain a brownish mucoid fluid, cholesterin crystals and epithelial debris. Very occasionally definite pus may be found. The X-ray appearance showing the well-marked outline of a cavity with osteoporosis of surrounding bone is usually characteristic.

*Treatment* consists in opening the cyst, scraping the walls of the cavity and pressing the thin bony covering together to reform as far as possible a smooth surface. If this surface can be covered by a flap of mucosa so much the better.

### ODONTOMES

Odontomes arise from some atypical development of a part of, or the whole, embryological tooth germ. Many varieties are described, but here only the more important and those of relatively common occurrence will be described.

**Epithelial Odontome** (also called *Fibrocystic Disease of the Jaw* or *Adamantinoma*).—The etiology of this condition is in many respects similar to that of the dental cyst, but a multilocular cyst results from degeneration of the proliferated remnants of the enamel organ of the primary tooth. Histologically, cavities are found in the midst of masses of columnar or cubical epithelium, with occasional nuclei of fibrous or osseous tissue scattered throughout.

The epithelial odontome is found almost exclusively in young adults, particularly women, and favours the lower jaw, especially the molar region, ten times more frequently than the upper. It is slow-growing, but progressive. The expansion is chiefly towards the outer aspect of the jaw and the tumour may reach a great size (Figs. 154 and 155). It has a marked tendency to recur after removal.



FIG. 154

An epithelial odontome.

**Follicular Odontome** (*Dentigerous Cyst*).—This is a unilocular cyst derived from atypical growth changes in the enamel organ of one of the permanent teeth. The tooth, most frequently a lower molar, is always maldeveloped and is retained within the cyst (Fig. 156),

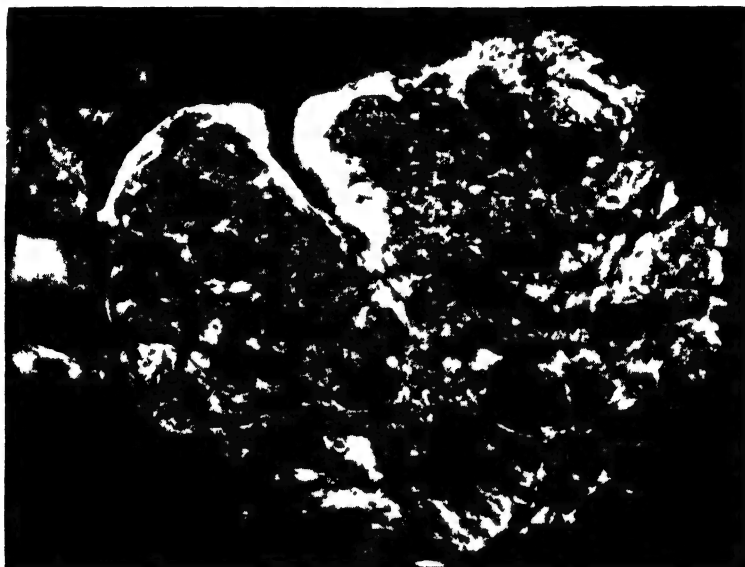


FIG. 155

A specimen illustrating the appearance in cross-section of an epithelial odontome.

either free in the cavity or attached to its wall. The cyst, which occurs usually in young people, is lined by a tough vascular membrane and contains a thick glairy fluid. The unerupted tooth, lying in a bony cavity, is easily seen by X-rays (Fig. 157), but it must be remembered that clinically there may be no absent tooth as would be expected, the milk tooth having remained in place.

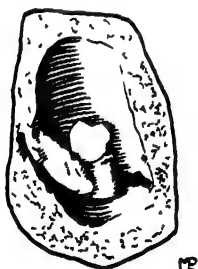


FIG. 156

A drawing of a dentigerous cyst from a specimen in the museum of the Royal College of Surgeons.

A sub-group of this type is the “**compound follicular odontome**,” in which several dental sacs of contiguous teeth are involved in the pathological change, but the multilocular cyst which results may contain many more teeth than would be accounted for by those which had failed to erupt. As many as forty of these “denticles” have been reported.

**Composite Odontome.**—In this type the whole tooth germ is disorganised, and masses of dentine, enamel and cement substance are found mixed indiscriminately, there being in contradistinction to the dentigerous cyst no tooth recognisable as such. The composite odontome is found in both jaws. slightly more frequently in the upper. It may grow to considerable size, but is always easy to remove, having very little apparent connection with surrounding bone.

**Radicular Odontome**, as its name implies, is formed in connection with one fang of a particular tooth. It occurs in old people, is usually quite small, and is characterised histologically by an absence of enamel.

**Fibrous Odontome**.—This is an uncommon type, secondary changes in the wall of the dental sac producing layers of thick fibrous tissue, in the midst of which in a relatively small cavity lies the unerupted tooth. It is frequently associated with rickets.

**Cementoma** is also rare, and is most simply described as a calcified fibrous odontome.



FIG. 157

An X-ray of the face showing a tooth contained in a dentigerous cyst of the upper jaw. It will be noticed that the crown is facing in the wrong direction.

The treatment of all these growths involves opening the pathological dental sac, scraping out the contents, breaking down all septa and clearing the walls. The resultant cavity is flattened as far as possible, carbolicised and, if feasible, covered with a mucosal flap. If recurrence takes place a local excision of the affected portion of the jaw is indicated.

#### “WISDOM TEETH.”

The importance of the third molar lies in the frequency with which it gives rise to trouble. Both the time and the method of eruption are most irregular, and very often either before or after eruption it becomes carious. If late in erupting it lies wedged behind the second molar and its extraction may be extremely difficult. It is frequently considered advisable to remove the second molar and thus allow the wisdom tooth room to erupt rather than attempt a difficult extraction of this latter

tooth itself. The question of dealing with wisdom teeth is one in which the advice and assistance of a skilled dental surgeon should always be sought.

### EXTRACTION

This subject again belongs essentially to the realm of the dental surgeon, but in view of the emergencies that arise in ordinary surgical practice, every student should familiarise himself with the different types of dental forceps, their use and that of the dental elevator. The latter, which is used more particularly for the lower molar teeth and for stumps, must be used with care, as in inexperienced hands it can do much damage not only to gums but to the jaw itself and even the maxillary antrum. In any extraction a firm grip of the affected tooth should be taken with the particular forceps by insinuating the points between the tooth and the gums; the tooth should then be loosened by gently rocking inwards and outwards and finally extracted towards the labial aspect of the jaw, the outer alveolar table being thinner and therefore less resistant.

Order and method are very important in multiple extractions. In general it can be stated that lower teeth should be extracted before upper, back teeth before front and stumps before whole teeth. A system such as this keeps the field free of troublesome hæmorrhage as long as possible. It is obviously necessary to remember the number of fangs possessed by each particular tooth in order that stumps may not be left behind.

Nasal gas is probably the best general anæsthetic to use for dental extractions. Chloroform should never be given with the patient in a sitting position. Local anæsthetics and blocking the inferior dental nerve are extensively used.

Hæmorrhage from tooth sockets is usually slight, and is arrested naturally in a few moments. In cases where bleeding is prolonged, *e.g.*, in hæmophilia, scurvy, purpura, etc., or after particularly difficult extractions where considerable laceration of the gums has occurred, a warm astringent mouth-wash should first be tried. If this fails, recourse must be had to plugging the socket with adrenalin gauze and giving hæmoplastin. In really severe cases blood transfusion may be necessary.

A. E. PORRITT.

R. M. HANDFIELD-JONES.



## CHAPTER XVIII

### THE MOUTH, PALATE, TONGUE AND SALIVARY GLANDS

**D**EVELOPMENT.—The mouth is developed from the primitive stomodeum, a depression of the ventral epiblast, and is therefore lined by squamous epithelium. The tongue makes its appearance as a small median elevation (the tuberculum impar) in the floor of the mouth, which is soon joined at each side by lateral growths from the mandibular arches. This develops into the buccal part of the tongue, consisting of its anterior two-thirds. The posterior or pharyngeal portion arises from the third branchial arches.

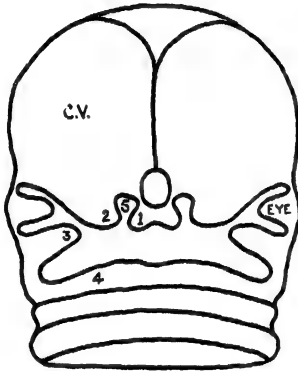


FIG. 158

Diagram showing the embryological structures taking part in the development of the palate.

1, The globular process; 2, The lateral nasal process; 3, The maxillary process; 4, The mandibular bar; 5, The nasal pit; C.V. is the cerebral vesicle.

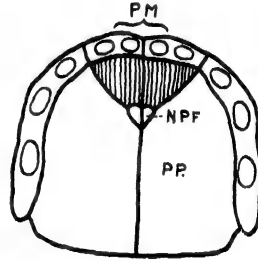


FIG. 159

Drawing showing the component parts of the hard palate.

P.M. is the premaxilla, and the shaded area represents that part of the palate derived from it; P.P. is the palatine plate; N.P.F. is the nasopalatine foramen.

The development of the palate is a complex procedure. The fronto-nasal process appears as a median bud in the roof of the stomodeum. This bud is further differentiated into a median nasal and two lateral nasal processes. The latter have no further significance here, but from the median nasal process are developed the nasal septum, the premaxilla and the philtrum or middle third of the upper lip. The premaxilla is derived from the anterior end of the median nasal process from which two protuberances jut forwards and downwards. These two globular processes later fuse together and form the anterior part of the hard palate and the central part of the alveolar margin of the upper jaw bearing all four incisor teeth. While these changes have been taking place, the maxillary process is budding inwards and forwards from the upper border of the mandibular bar on each side. From the buccal aspect of each maxillary process, the palatine plates grow in towards the middle line, fusing first with the premaxillary section

of the palate and then joining with each other and with the lower or free edge of the nasal septum. This fusion takes place from before backwards and for this reason it is easy to explain why a partial cleft palate is more common than a complete lack of fusion (Figs. 158 and 159).

*Surgical Anatomy.*—The buccal mucous membrane lines the inner aspect of the cheeks and lips, the alveolar margins of both jaws and the palate. It is continuous with the skin at the red margins of the lip and with the mucous membrane of the pharynx posteriorly. It covers the floor of the mouth, from which it passes to the under surface of the tongue and thence to the dorsum.

The tongue is composed of an oral and a pharyngeal portion, separated from each other by an inverted V-shaped groove, which is marked on the dorsal surface by the circumvallate papillæ. Its anterior two-thirds is intrabuccal and its dorsal surface is covered by a rough, thickened mucous membrane which is studded with both filiform and fungiform papillæ,

while its under surface has a thin, smooth and glistening lining, through which can be seen the ranine veins. The front half of the buccal portion is free and has considerable mobility. The posterior part is attached by muscles and reflections of the mucous membrane to the lower jaw and by a median longitudinal fold, the frenum, to the floor of the mouth.

The substance of the tongue consists of interlacing muscles, which are either intrinsic or extrinsic. The latter provide attachment to the hyoid bone, mandible and styloid process. The tongue is divided into equal halves by a median fibrous septum, which prevents any exchange of vascular or lymphatic circulation except at the tip. The presence of this septum is indicated on the dorsal surface by a longitudinal groove which ends in the apex of the inverted V, a point which is believed to represent the foramen cæcum, *i.e.*, the site of origin of the thyroid anlage. The muscles are supplied by the hypoglossal nerve. Sensation is recorded by the glossopharyngeal nerve in the posterior third and the lingual in the anterior two-thirds, while taste is appreciated through the medium of

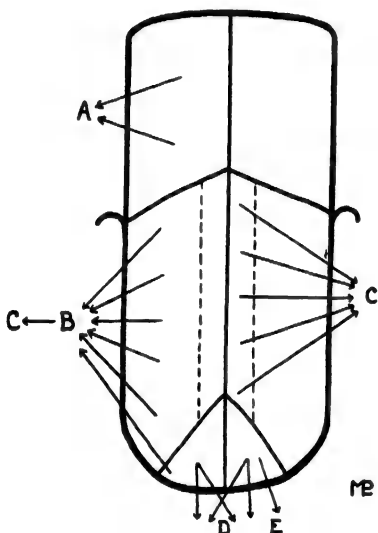


FIG. 160

Diagram representing the lymphatic drainage of the tongue.

A is the posterior third; B represents the submaxillary lymphatic group; C is the deep cervical group; D represents the submental group; and E an occasional lymph vessel draining to the lower deep cervical group.

the chorda tympani and the glossopharyngeal nerves. The arterial blood is carried to the tongue chiefly by the lingual arteries.

The *lymphatic drainage* of the tongue has immense surgical importance, and is arranged as follows: (1) the apical vessels drain into the submental glands of *both* sides, the efferents of which pass to the deep cervical group, and an occasional vessel may go direct to one of the lower glands in the deep group; (2) the lateral set drain the side of the tongue into the submaxillary lymph glands and thence to the deep cervical glands; (3) the central set drain direct to the lower deep group; (4) the posterior set pass through the superior constrictor muscle to the upper deep cervical glands. There is a certain amount of overlapping, as Fig. 160 shows.

The floor of the mouth is covered with thin smooth mucous membrane

and is thrown into folds, namely, the frenum and the submaxillary ridges, the latter formed by Wharton's ducts, the openings of which are situated on prominent little papillæ. Many mucous glands are situated in this area, in addition to the sublingual salivary gland. One group in the frenum was described by Blandin and Nuhn and another by Bochdalek.

*Examination of the Buccal Cavity* should always be done by artificial light with a spatula. A most useful combination of the two is available, a smooth wooden spatula sliding into a holder on which is mounted a small lamp, activated by an ever-ready battery in the handle. It is often an advantage to place a piece of gauze or lint over the tip of the tongue, so that it can be manipulated without discomfort. All ulcers and tumours must be carefully palpated with the ungloved finger.

## THE MOUTH

### STOMATITIS

Stomatitis is an inflammation of the mucous membrane of the mouth. It can be secondary to many infective processes in structures in close proximity to the mouth, but in this section stomatitis of primary origin only will be described.

**Catarrhal Stomatitis** is associated with the infectious fevers, gastrointestinal disturbances, abdominal operations and local irritants, such as carious teeth, ill-fitting dentures and excessive smoking. The mucous membrane becomes red and swollen.

*Treatment* seeks to eliminate any evident cause and local measures include hot mouth-washes and painting with glycerin and borax. A very pleasing mouth-wash is made up of

Glycerini Ac. Carbolic	.	.	.	℥xx
Tinct. Lavandulæ Co	.	.	.	℥x
Potassii Chloratis	.	.	.	gr. xv
Aquam	.	.	.	ad. ʒi

**Aphthous Stomatitis** occurs in children in whom small vesicles appear in the mucous membrane. These coalesce and break down to form small grey ulcers. They heal spontaneously if the general health is good.

**Thrush** is due to a parasitic fungus, *Oidium Albicans*, and attacks undernourished children. The lesions resemble the aphthous ulcers, but the child is more seriously ill and diarrhoea is probably present.

*Treatment* is directed towards improvement in the general health and local mouth-washes and paints will assist in keeping the mouth clean.

**Vincent's Angina** may give rise to widespread ulcerative stomatitis (see Chap. XXII, p. 427).

**Gangrenous Stomatitis.**—*Cancrum Oris* is associated with the infectious fevers, especially measles. It is described in Chap. IX, p. 176.

**Ludwig's Angina** may start as an acute infective stomatitis in the floor of the mouth. It spreads rapidly to the submental region, where

the gravity of the condition overshadows the buccal symptoms. It is therefore described in Chap. XIX, p. 358.

**Mercurial and Syphilitic Stomatitis** have or should have ceased to occur owing to early diagnosis and improved methods of treatment.

**THE FLOOR OF THE MOUTH.**—A sublingual abscess is the infrequent result of infection in the sublingual salivary gland or of minor injuries to the mucous membrane. It forms a painful, tender and fluctuant swelling in the floor of the mouth and the œdema may spread via the frenum into the under surface of the tongue. It must be incised and drained.

A squamous-celled carcinoma in this region is a well-defined lesion. It is so closely associated with carcinoma of the tongue that its description will be postponed till later (see p. 346).

## THE PALATE

### CLEFT PALATE

The development of the palate has been described above (p. 327). In order to achieve a perfect palate, the premaxilla must unite with the alveolar margins of the maxilla, the palatal plates must meet and join in the middle line, except in front where they unite on each side with the palatal portion of the premaxilla, and finally the nasal septum must join the upper surface of the palate. This process of fusion begins in front and extends backwards so that the two halves of the uvula are the last to join together.

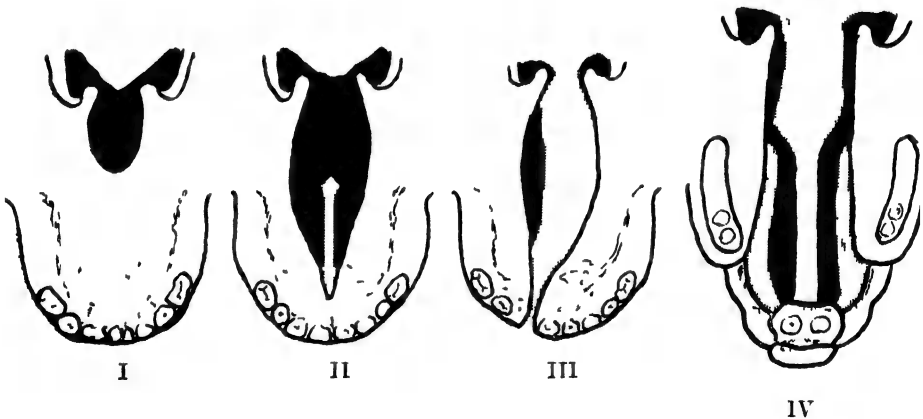


FIG. 161

Veau's four types of cleft palate.

### VARIETIES OF CLEFT

Veau has described four types. **Type I** affects the uvula and soft palate; in **Type II** the cleft spreads into the hard palate but does not extend further forward than the nasopalatine foramen; **Type III** is a complete unilateral cleft of palate and alveolar margin and is

associated with a single hare-lip; **Type IV** shows a complete bilateral cleft with a double hare-lip (Fig. 161).

In a unilateral complete cleft the division is in the middle line behind, but in front swings to one side between the premaxilla and palatal plates. In bilateral cases the cleft is Y-shaped with the result that the premaxilla is detached from the alveolar margin and is suspended from the nasal septum above, being rotated forwards and upwards in such a way as to make it appear to be attached to the nose.

The relation between a cleft palate and the nasal septum varies. In Veau's Type II the latter may be attached to either side or be free; in Type III it is usually fixed to that side of the palate opposite to the alveolar cleft (Fig. 161). The width of the gap is greater in Types I and II, in which there is an appreciable deficiency in the lateral elements of the palate. In all cases there is a marked increase in height of the palatal arch.

#### EFFECTS UPON FUNCTION

These are severe and comprise difficulties with (1) nutrition, (2) speech and (3) dentition, while the double cleft presents a hideous deformity. **Nutrition** is affected because the cleft not only makes sucking weak or impossible but also allows regurgitation through the nose. This difficulty can usually be partly overcome by the use of special teats or by spoon-feeding. **Speech** is seriously interfered with, since the explosive consonants B, D, G, T and P cannot be produced properly and a characteristic "cleft palate voice" results. **Dentition** is disturbed in complete clefts and badly planned operations can damage the dental area of the upper jaw. Finally, the free communication between nose and mouth renders these children liable to minor inflammatory lesions in either cavity, and the growth of the face is retarded.

#### ROLE OF THE PALATE IN SPEECH AND SWALLOWING

These two functions demand a roof to the mouth and a freely moving diaphragm or sphincter to shut off, when necessary, the nasopharynx from the mouth, nose and lower pharynx. An artificial roof to the mouth can be supplied by a dental surgeon, but nothing can replace an inefficient palatal musculature.

The Soft Palate is comprised of the following muscles :—

1. Levator Palati, which acting as a single unit with its fellow of the opposite side through an intermediate tendon lifts the palate upwards and backwards;
2. Tensor Palati of each side gives rise to a tendon which, passing round the hamular process of the internal pterygoid plate, is inserted into the palatal aponeurosis. Acting together they tense the palate and oppose the levator.
3. Glossopalatine, which forms an incomplete sphincter and draws the palate away from the postero-superior wall of the pharynx. It therefore opposes the nasopharyngeal sphincter.
4. Azygos Uvulæ is attached to the uvula.

Acting in conjunction with these muscles is the superior constrictor, the upper fibres of which form a nasopharyngeal sphincter, which is reinforced laterally by the pharyngo-palatine and salpingo-pharyngeus muscles. The uppermost fibres of the superior constrictor muscle during contraction raise a ridge of mucous membrane on the posterior pharyngeal wall, named "Passavant's cushion." Wardill and Whillis describe a similar cushion upon the upper surface of the palate at the point of insertion of levator palati.

It will now be realised that the nose is completely shut off from the mouth and pharynx during speech and swallowing by a complicated but beautifully co-ordinated muscular mechanism. Passavant's and the palatal cushions are apposed closely and the nasopharyngeal sphincter completes the closure, which in normal people is both air and water tight.

### TREATMENT

The surgery of cleft palate has undergone radical changes in the past twenty-two years and we owe much to Veau, Axhausen, Gillies, Kilner, Wardill, Denis Browne and Oldfield. To the last named I am indebted for his help and permission to use several of his drawings.

Operation must be performed before the child has commenced to speak and fortunately this is usually delayed in cleft palate children. The ideal age is between the eighteenth and twenty-fourth months. It need be denied to few, if any, patients; it should be delayed in the presence of (1) carious teeth, (2) grossly infected tonsils, (3) hæmolytic streptococci or diphtheria bacilli on culture from a swab, (4) active otitis media, (5) active respiratory tract infection and (6) hæmoglobin below 70 per cent. (Oldfield).

**Modified Axhausen Operation.**—1. **PARING THE LEFT EDGES.**—The edge of the soft palate is transfixed by a small scalpel and excised in its whole length. Over the hard palate the knife cuts along the junction of nasal and buccal mucous membrane and goes down to the bone.

2. **ELEVATION OF NASAL MUCOSAL FLAPS.**—(A) From the hard palate. The mucosa is first separated from the inner border of the palate with a blunt dental elevator and this requires great care. When this step is completed the flap is raised from the floor and side of the nasal cavity with a larger elevator. This freeing is carried from the anterior extremity of the cleft back to the junction of hard and soft palate. A small fibrous band on each side is attached to the posterior nasal spine and blends with the palatal aponeurosis. It must be divided to complete the mobilisation of the nasal flaps.

(B) In the soft palate. The mucosa is carefully separated from the underlying layer of muscles. In unilateral clefts the inner flap is taken from the nasal septum, while in bilateral cases an incision must be made in the free margin of the septum and the mucosa on each side turned up (Fig. 162).

3. **MOBILISATION OF PALATAL FLAPS.**—A lateral incision is made near the inner border of the teeth running forwards almost to the anterior limit of the cleft and extending backwards to a point  $\frac{1}{2}$  in.

behind the last molar tooth (Fig. 163, A). Through this incision the mucosa is lifted from the bone in its outer two-thirds, the remainder being elevated through the inner edge previously made. The soft palate is now mobilised by exposing the origin of the internal pterygoid



FIG. 162

A shows the paring of the edges. B demonstrates the elevation of the nasal mucosa (green), exposure of palatal muscles (red) and the fibrous band at the anterior end of the latter.

muscle through the posterior part of the lateral cut and by using it as a guide to the hamular process, the base of which is either fractured or cut through with scissors.

4. LATERAL PHARYNGEAL MOBILISATION.—On the inner surface of the internal pterygoid is an areolar space surrounding the pharynx.

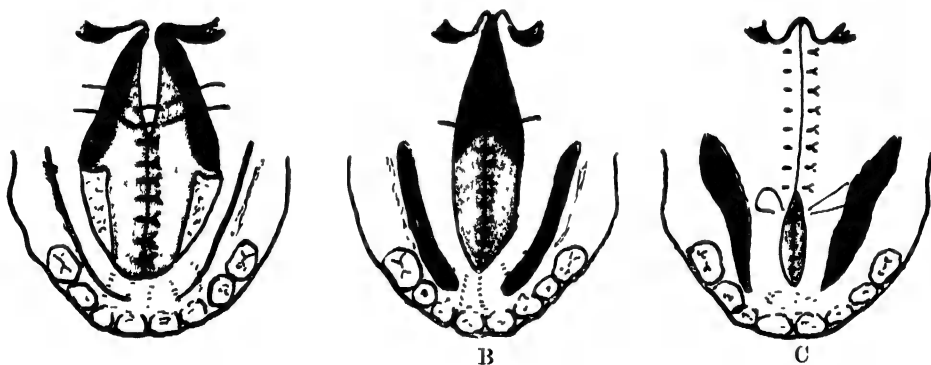


FIG. 163

Shows A, the lateral palatal incision and suture of nasal mucosa ; B, suture of palatal muscles ; and C, suture of buccal mucosa.

Blunt dissection in this space from either side further frees the soft palate. Finally the lateral half of the palatal aponeurosis is divided from its attachment to the posterior border of the hard palate.

5. SUTURING OF FLAPS.—(A) In the nasal mucosa fine catgut stitches are inserted in such a way that the edges are inverted upwards into the nasal cavity (Figs. 163, A and 164). This suturing is carried along the whole length of the cleft from front to back. (B) The muscles of the soft palate are approximated by three stitches which must not

include either nasal or buccal mucosa (Fig. 163, B). (C) Over the soft palate the buccal mucosa is brought together by a series of fine silkworm gut sutures introduced on an atraumatic needle. That over the hard palate is stronger and will tolerate somewhat stouter gut (Fig. 163, c).

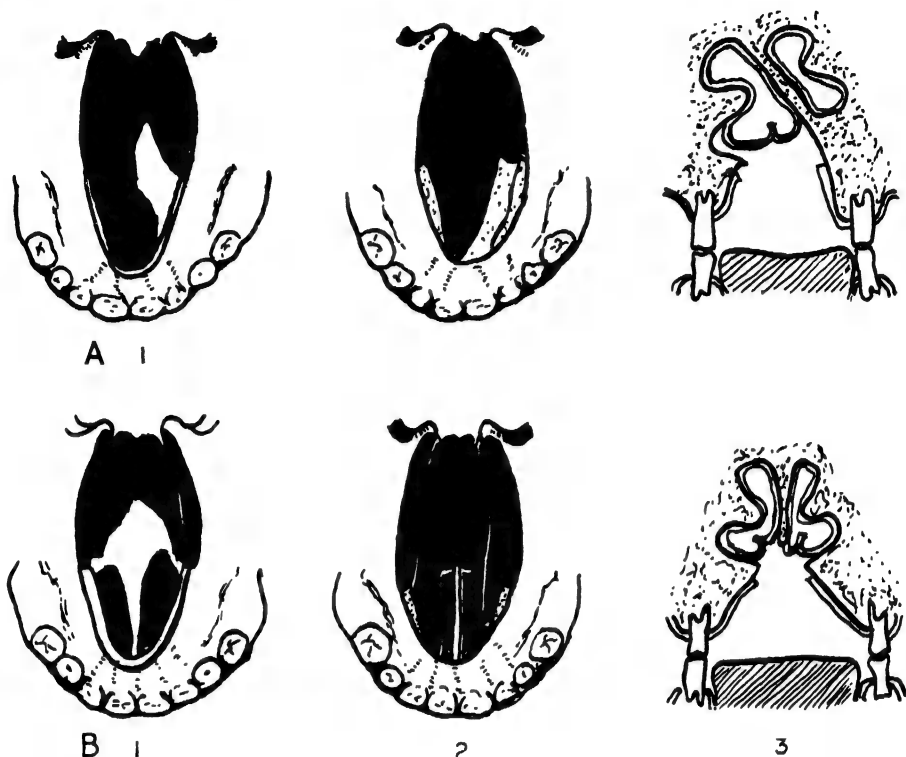


FIG. 164

Illustrating suture of nasal mucosa in A a unilateral and B a bilateral cleft.

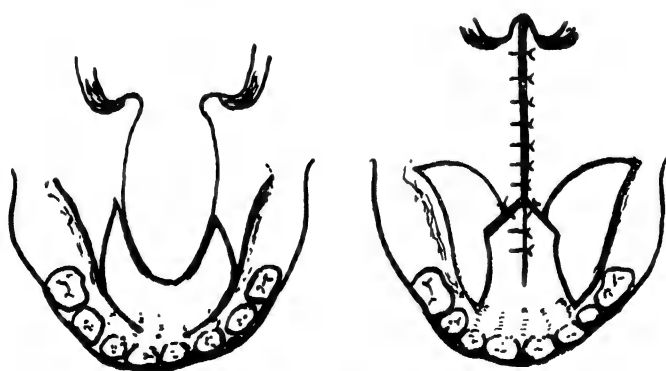


FIG. 165

Wardill's V-Y operation.

**Wardill's V-Y Operation.**—When the cleft is wide there is sometimes a shortening of the palate, so that after repair the uvula and soft palate do not reach far enough back. In such cases Wardill's operation is performed, the essential principles of which are seen in Fig. 165. It will be accompanied by Wardill's pharyngoplasty.



**Methods of Narrowing the Nasopharyngeal Isthmus.**—1. **WARDILL'S PHARYNGOPLASTY.**—A transverse incision is made at the level of the transverse arch of the atlas into the posterior pharyngeal wall passing through the mucosa and superficial fibres of the superior constrictor. This muscle is separated from the underlying buccopharyngeal fascia and the wound then sewn up vertically. The result is to narrow the nasopharyngeal isthmus and raise an exaggerated Passavant's cushion.

2. **DENIS BROWNE'S PHARYNGOPLICATION.**—A purse string is passed from one lateral relaxation incision behind the pharyngeal wall at the level of Passavant's cushion. It emerges at the opposite palatal incision and is then passed through the soft palate and tied firmly. In this way a ring suture encircles the nasopharyngeal isthmus and narrows it. This stitch undoubtedly relieves tension upon the repair sutures in the palate, but it is unlikely that it persists for a sufficient length of time to fulfil its primary purpose.

These procedures are not essential in every case but are especially useful in short palates and in all patients in whom incompetence of the nasopharyngeal sphincter is to be expected.

**Special Technique for Residual or Recurrent Clefts.**—These are based upon the amount of tissue available and the degree of scarring. They entail methods of lengthening the palate and mobilising flaps. Amongst them may be mentioned the Gillies-Fry operation, Moorehead's M and Dorrance's U incisions together with certain procedures in which flaps are swung from the cheek to cover a palatal defect.

**After-treatment.**—An intravenous drip of glucose-saline or blood is given during the first twelve hours. Soft foods are allowed by mouth after forty-eight hours. However, the most important part of after-care is speech training and that should be in the hands of an expert.

**ULCERATION OF THE PALATE.**—(1) Simple ulcers occur in association with stomatitis. (2) Secondary syphilis is responsible for snail-track ulcers, while in tertiary syphilis, a gumma in the midline is not uncommon, though much more rare to-day than formerly. It is prone to spread to the underlying bone, which becomes necrotic, and perforation of the palate follows. (3) Lupus spreads into the mouth from the face or nose, but a tuberculous cold abscess is rare in this situation. (4) Malignant ulcers are squamous-celled carcinomata.

**ABSCESS OF THE PALATE** is either traumatic in origin or due to spread from the alveolar margin (gumboil). A painful, tender and fluctuant swelling appears and may require a small incision.

**PERFORATION OF THE PALATE** is almost confined to tertiary syphilis, though rarely it may be due to trauma or lupus.

**TUMOURS OF THE PALATE.**—A simple papilloma is sometimes seen. An adenoma occurs as a small, smooth or lobulated swelling arising in the mucous glands of the mucous membrane and being similar in many respects to the salivary gland tumour. It is removed quite simply.

Malignant growths are primary or secondary, the latter spreading to the palate from the tongue, tonsils, alveolar margins or antrum of Highmore. The primary is a squamous-celled carcinoma which displays the characteristic appearance of malignant ulcers. It should be removed as early as possible by diathermy.

**ELONGATION OF THE UVULA** is usually seen in chronic pharyngitis, a hypertrophy of the mucous membrane taking place. In the early stages

gargles and paints should effect a cure, but later there is a troublesome cough. If very long and definitely causing symptoms, the uvula should be cocaineised and snipped off with scissors.

## THE TONGUE

### CONGENITAL DEFECTS

**Absence of the tongue** has been reported but is very rare, as is also bifid tongue. Hemiatrophy is usually the result of paralysis of the hypoglossal nerve and not a defect in development.

**Partial Ankyloglossia**, or Tongue-Tie, is a condition in which the frenum is short and the movements of the tongue are restricted. This is not a true pathological entity, but rather an "old wives' tale" that it can interfere with speech. The sole justification for "cutting the tongue-tie" is so marked a fixation that sucking is impossible. In such cases the tongue is raised between the fingers, and the frenum is snipped with blunt scissors near its attachment to the floor of the mouth.

**Complete Ankyloglossia** is almost identical with absence of the tongue except in the acquired varieties in which it is due to extensive infiltration by inflammatory or neoplastic processes.

**Tongue Swallowing** is the opposite of tongue-tie. Not only is the frenum unduly lax, but the tongue itself is relatively long. A few fatal results from asphyxia have been recorded.

**Macroglossia**.—Enlargement of the tongue may be classified as follows :—

- |                      |                          |
|----------------------|--------------------------|
| 1. Lymphangiomatous. | 4. Syphilitic.           |
| 2. Muscular.         | 5. In endocrine disease. |
| 3. Inflammatory.     | 6. In mental disease.    |

**CONGENITAL MACROGLOSSIA** is due to a condition of cavernous lymphangioma throughout the tongue, but is not necessarily present at birth. The process starts in one part of the tongue, but spreads until the whole organ is affected. The tongue is symmetrically enlarged, the papillæ are hypertrophied and clear vesicles appear on the surface. If untreated, it will become too large for the mouth and protrude through the lips, being grooved and ulcerated by the pressure of the teeth.

*Treatment* consists in a V-shaped resection with suture.

**MUSCULAR MACROGLOSSIA** may occur very rarely in normal people, but is usually seen in congenital idiots, cretins, or associated with various forms of gigantism. It is present in acquired hypothyroidism (myxœdema) and in some forms of mental disease.

*Treatment* is directed towards the cause, but a wedge resection may be necessary to overcome protrusion and ulceration.

Inflammatory causes are those leading to recurrent attacks of acute glossitis. Syphilitic macroglossia is a very rare manifestation of the disease to-day.

## INJURIES

The tongue may be severely bitten as the result of a fall or a blow on the chin or during the convulsions of epilepsy, tetanus or strychnine poisoning. A foreign body such as a pipe stem or fish bone may penetrate its substance. The dangers are immediate hæmorrhage, which can be very severe, and a remote acute glossitis with or without abscess formation. The hæmorrhage can be temporarily controlled by hooking the tongue forward with the index finger. Small wounds with moderate bleeding are cleansed and sutured, but a really severe hæmorrhage may demand ligature of the lingual or external carotid artery.

The tongue may also be injured by the stings of insects (*e.g.*, a wasp), or by being burned or scalded. This may occur in small children who put their mouths to the spout of a boiling kettle. These injuries lead to acute glossitis.

## THE INFLAMMATORY DISEASES OF THE TONGUE

**Acute Superficial Glossitis** is an inflammation of the mucous membrane, and is merely part of an acute stomatitis (p. 329).

**Acute Parenchymatous Glossitis** affects men more than women and follows penetrating wounds, stings of insects, infectious fevers or a severe stomatitis. The infecting organisms are either staphylococci or streptococci, and the condition, especially in the latter type of infection, is always grave. The swelling, which may be either unilateral or bilateral, comes on rapidly and may progress to such an extent that the tongue is indented and ulcerated by the teeth. It is very painful, tender and indurated, the breath is foul-smelling, the submaxillary lymph glands are enlarged, and there is a general febrile reaction.

*Treatment.*—Any obvious cause will receive attention and a brisk aperient be given. Large hot dressings are applied to the neck and face; frequent hot mouth-washes should be used, and in the intervals ice may be sucked. Sulphapyridine is given by mouth in  $7\frac{1}{2}$  gr. doses three times a day. If the swelling continues to increase in spite of treatment, an incision is made in the dorsum on each side of the middle line. Relief is instantaneous and hæmorrhage is slight. Pain in the tongue or in the nerve distribution of the auriculo-temporal may be so severe that morphia will be indicated.

*Complications.*—A localised abscess may form in the depth of the muscles, usually after the more severe inflammation has begun to subside. A tense and tender but rarely fluctuant swelling is present, and will call for incision and drainage. In the fulminating streptococcal cases, gangrene of part or all of the tongue may follow.

**Chronic Superficial Glossitis** comprises a number of conditions, the most important of which are grouped under the heading of **Leukoplakia**. This is not confined to the tongue but affects any part of the buccal mucous membrane, in which it may be seen while the tongue remains free. It is rarely met with in women, but affects men after the age of 45 years, in the great majority of whom (over 90 per

cent.) there is a history of syphilis. There are, however, other predisposing factors, among which are chronic sepsis in the mouth, dental caries, excessive smoking, the drinking of raw spirits, the eating of highly-spiced foods, chronic dyspepsia and possibly gout.

*Naked-eye Appearance.*—Stage 1. Red hyperæmic patches appear as a result of swelling of the papillæ. They are flat, smooth and very slightly raised, and need careful inspection in a good light before they can be critically defined.

Stage 2. Hypertrophy and Keratinisation of the patches, some of which have coalesced to form round, oval or polygonal plaques, now follow. The plaques are white, raised and firm though not indurated.

The term *ichthyosis* has been applied to this appearance (Fig. 166).



FIG. 166

Leukoplakia.

Stage 3. Interference with the blood supply beneath the white plaques by syphilitic endarteritis leads to shedding of the hypertrophied papillæ. A smooth, flat, red and glazed patch results. *Psoriasis linguae* and the red glazed tongue are terms applied to this condition.

Although the white plaque usually dominates the picture, all three stages may be seen in different parts of the same tongue.

*Microscopic Detail.*—The underlying process is a chronic inflammatory reaction in the deeper layers of the mucous membrane and submucous tissues, exhibiting a small round-celled infiltration and endarteritis in the small arterioles.

*Complications.*—**Fissures** are almost an essential feature in all long-standing cases of leukoplakia. The elevation of the keratinised plaques leads to the formation of clefts between them, and small particles of food and clumps of bacteria are apt to be retained. The subsequent irritation and infection cause linear ulceration or **cracks**, which do not yield easily to treatment. This condition may justly be termed "the threshold of lingual cancer," for chronic ulceration and continued irritation coexist with an unstable state of the epithelium. It is claimed that one in every four sufferers from leukoplakia develops carcinoma of the tongue.

*Symptoms* are dryness of the mouth, constant discomfort, pain in taking irritant foods or fluids and impairment of taste. The pain is increased when the tongue is fissured and cracked.

*Treatment.*—Leukoplakia is a most intractable disease and often progresses in spite of treatment.

1. **Local.**—Every source of infection and irritation must be removed. The patient is referred to his dentist, who must remain unsatisfied until every trace of dental sepsis is eradicated. Smoking must be forbidden absolutely and alcohol is likewise prohibited. The diet is carefully restricted to avoid any possible source of irritation and,

wherever possible, the teeth should be brushed after every meal. Local applications are both useless and dangerous, and nothing stronger than a chromic acid paint (4 gr. to the ounce) should be used.

An unremitting watch must be kept on every patient so that the least change suggestive of carcinoma can be quickly recognised.

2. **Antisymphilitic.**—In the presence of a positive Wassermann test or in a patient who has a history of syphilis, specific treatment in the form of potassium iodide and intramuscular bismuth should be given. Little improvement can be expected, but further spread may be arrested.

3. **Operative.**—Cracks and fissures are so pregnant with danger that they should be removed by the diathermy loop. Extensive leukoplakia justifies Butlin's operation of removing the mucous membrane from the dorsal surface of the anterior two-thirds of the tongue. Radium and new technique of local X-rays are giving encouraging results, but seem liable to be followed by recurrences.

THE SMOKER'S PATCH is a small red denuded area near the front of the tongue, similar to leukoplakia, but showing little tendency to spread.

GEOGRAPHICAL TONGUE is a rare condition seen in ill-nourished children. It is characterised by irregular patches, mapping out the tongue.

GLOSSODYNIA EXFOLIATIVA is another rare affection in which severe lingual neuralgia is associated with few local signs save a thinning of the mucous membrane.

BLACK TONGUE (HAIRY TONGUE, NIGRITIES).—An overgrowth of the filiform papillæ on the dorsum in front of the circumvallate ridge gives rise to this strange appearance, as if the tongue were covered with wet black hair. There are no symptoms, and mechanical scraping is all that is needed.

LINGUAL SYPHILIS.—Lesions occur in all stages. A primary chancre is seen near the tip and is accompanied by an extensive involvement of the lymphatic glands. In the secondary stage, mucous patches and snail-track ulcers coexist with similar patches on the fauces and palate. Chronic superficial glossitis and a localised gumma characterise the late tertiary period. A gumma forms a swelling of the dorsum, which later becomes soft and fluctuant. Finally the typical ulcer appears (see pp. 70 and 340).

LINGUAL TUBERCULOSIS manifests itself in several forms, all of which are rare, and none exist except as a complication of pulmonary or laryngeal infection. A solid tuberculoma occasionally forms beneath the mucous membrane, papillomata and fissures are described, but the only comparatively common lesion is the ulcer, described on the following page.

## ULCERS OF THE TONGUE

Ulcers of the tongue are of common occurrence and provide frequent difficulties in diagnosis. They may be classified as: (1) Dyspeptic, (2) Traumatic or Dental, (3) Tuberculous, (4) Syphilitic, and (5) Neoplastic.

**Dyspeptic Ulcers** arise on the dorsum of the tongue as well as in the reflections of the mucous membrane between the lips and the alveolar margins of both jaws. They are associated with attacks of

dyspepsia, and probably slight trauma also plays a part in their origin. They appear as very small, circular, acute ulcers which are bright red, slightly oedematous and exquisitely tender. They last for thirty-six to forty-eight hours and their disappearance can be hastened if they are touched with a crystal of alum.

**Traumatic or Dental Ulcers.**—Jagged edges of carious teeth are not the only source, if the most frequent one, of traumatic ulcers. They are more extensive than the dyspeptic type, but their edges are so oedematous that the actual breach of surface appears to be very small until they are separated. There is a zone of inflammatory redness and induration around the ulcer crater, and they are very painful and tender. Treatment consists in removal of the cause and attention to the general cleanliness of the mouth.

**Tuberculous Ulcers** are rare and invariably associated with pulmonary and laryngeal tuberculosis. They usually take the form of a fissured ulcer and their extent can be appreciated only by separating their edges. They are lined with pale grey tuberculous granulation tissue, and are very painful and tender. If carefully viewed through a magnifying glass, each ulcer will be seen to be surrounded by a ring of minute "sentinel" tubercles. Local treatment has little effect, and general institutional treatment should be insisted upon without delay. It is true of a certain number of patients that the lingual ulcer has called attention to a hitherto unsuspected pulmonary infection.

**Syphilitic Ulcers** result from the breaking down of gummata and the subsequent separation of the "wash leather" slough. They appear on the dorsum as deep punched-out ulcers with little pain or tenderness, but leukoplakia will probably be present in other parts of the tongue.

*Treatment* is that appropriate to tertiary syphilis.

**Neoplastic Ulcers** show all the typical characteristics of the ulcerating squamous-celled carcinoma. The differential diagnosis is assisted by careful reference to the table on p. 341.

## CYSTS IN THE TONGUE

**Dermoid Cysts** occur in the midline of the tongue either deep in its substance or in the floor of the mouth. They may arise in connection with the thyroglossal duct or as sequestration dermoids due to the inclusion of cells of the epiblast during fusion of the skin in the middle line. They should be dissected out from beneath the skin of the submental region.

**The Lingual Thyroid Tumour** is a rare phenomenon, which is to be seen in the middle line of the tongue on the dorsum near the foramen cæcum. A firm, soft, red and rounded tumour is covered by mucous membrane, which rarely becomes sufficiently large to embarrass speech, swallowing or respiration. It should never be removed unless causing symptoms, and then only if there is no doubt that normal thyroid tissue is present in its proper place.

DIFFERENTIAL DIAGNOSIS OF LINGUAL ULCERS

	DYSPEPTIC.	TRAUMATIC DENTAL.	TUBERCULOUS.	TERTIARY SYPHILITIC.	NEOPLASTIC.
Situation . . .	Any part of tongue and buccal mucous membrane.	Edge, usually towards the back.	Anywhere. Not confined to tip.	Dorsum.	Edge.
Colour . . .	Bright red.	Red.	Pale grey.	Grey.	Dirty grey.
Depth and size . .	Shallow and small.	Moderate depth and moderate size.	Shallow. Moderate extent.	Deep. Large.	Early—small. Late—deep and large.
Edges . . .	Slightly oedematous.	Very oedematous.	Thin, pale and sinuous.	Clean cut.	Raised and everted.
Sides . . .	Cupped.	Sloping.	Steeply sloping to meet in an angle.	Punched out. Right-angled.	Sloping.
Floor . . .	Acute pyogenic granulation tissue.	Sloughing. Acute granulation tissue.	No floor. Walls covered with pale granulation tissue.	Pale pink indolent granulation tissue.	Necrotic debris.
Discharge . . .	Thick mucopus.	Thick mucopus.	Thin watery mucopus.	Very little serous.	Products of necrosis and mucopus.
Induration . . .	Nil.	Present but slight.	Nil.	Present but slight.	Very marked.
Pain and tenderness	Exquisite.	Marked.	Exquisite.	Nil.	Early—slight. Late—very severe.
Local cause . . .	Nil.	Evidence of trauma. Jagged tooth.	Nil.	Nil.	? sign of irritation or injury.
Other signs . . .	Dyspepsia.	Nil.	Pulmonary and/or laryngeal tuberculosis.	Leukoplakia. Positive history.	Possible leukoplakia. Marked limitation of movement.
Lymph glands . .	Nil.	Nil., unless remaining untreated.	Enlarged usually, contrary to usual teaching.	Nil.	Early—none. Late—moderate. Later still—very extensive.



## NEW GROWTHS OF THE TONGUE

**Benign Growths.**—Papilloma (Fig. 167) is by no means uncommon, shows no predilection for either sex or any age, and may occur in any part of the tongue. It appears as a warty growth, which may be surmounted by a thickened white epithelium—the leukoplakic papilloma. It is not to be confused with the pseudopapilloma due to hypertrophy in response to chronic inflammation. The growth should be removed by diathermy.

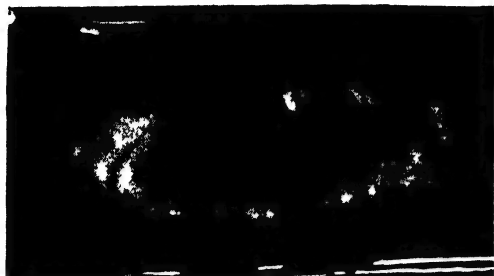


FIG. 167

A papilloma of the tongue.

Several other rare and unimportant innocent tumours are seen: (1) hæmangioma, which occurs in children as a very vascular red area, some-

times of considerable extent; (2) submucous lipoma and fibroma are seen beneath the smooth mucous membrane on the under surface of the tongue; (3) the very rare rhabdomyoma; and (4) the lymphangioma already referred to (p. 336).

**Malignant Tumours** are carcinoma and sarcoma.

## LINGUAL CARCINOMA

**Etiology.**—Cancer of the tongue is at the same time one of the most frequent and most fatal forms of malignant disease in the male. It is possible that, with the greater participation of women in smoking and cocktail drinking, the incidence in their sex will increase, but at present it is very rare. Chronic superficial glossitis is an acknowledged pre-cancerous condition, and other etiological factors include causes of irritation and sepsis in the mouth, and persistent trauma such as is supplied by the sharp jagged edge of a carious tooth. The buccal part of the tongue is more frequently affected than the pharyngeal area, and usually the growth starts on the edge and overlaps on to the dorsum.

**Naked Eye Appearance** is that of a squamous-celled carcinomatous ulcer (see diagnostic table above). Rarely it affects other guises, such as a flat warty growth, a malignant papilloma, or an indurated area around a fissure between two leukoplakic plaques; but however it first appears, the typical surface ulceration will soon become evident (Fig. 168).

**Microscopic Detail** is likewise characteristic, the epithelial down-growth, the formation of cell nests and the surrounding round-celled infiltration being present (Fig. 37, p. 107).

**Method of Spread.**—At first the growth will be confined to the intrinsic muscles, and during this period the movements of the tongue will remain unimpaired, but after a time the extrinsic muscles are invaded and movement on the affected side will be limited. The growth



can spread in many directions, forwards to the floor of the mouth, backwards to the fauces and pharynx, laterally to the gums and alveolar margin and downwards into the neck.

Reference to Fig. 160 will show routes of lymphatic spread. In an inverted V-shaped area at the tip, carcinoma will invade the submental glands of both sides. Growths on the lateral area of the anterior two-thirds spread to the submaxillary glands of the same side, while the less frequent cancers near the midline drain direct into the upper and middle deep cervical glands of the same side, and to a lesser extent, to the opposite side also. Eventually, all growths involve the deep cervical glands, whether the malignant cells have passed through intermediate glands on their way or not. The glands become enlarged,

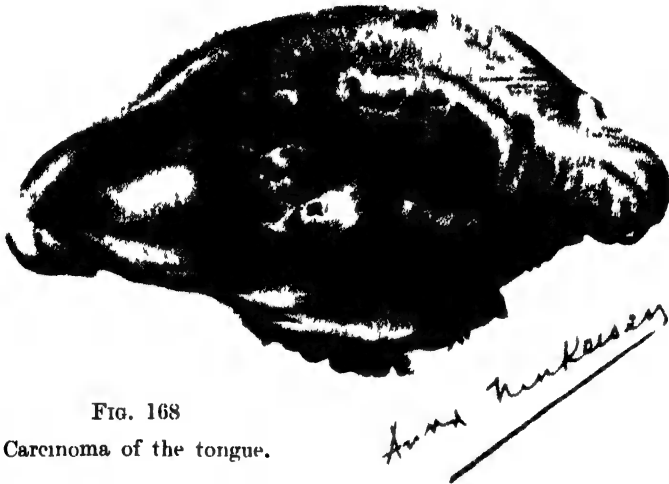


FIG. 168  
Carcinoma of the tongue.

very hard and adherent to each other and to the internal jugular vein. As the primary ulcer is invariably infected with secondary pyogenic cocci, the lymph glands tend later to become infected and break down, and, if not treated, pus will erupt through the skin, leaving necrotic ulcers in the neck.

Distant metastases are rare, but the local conditions are so terrible that death comes as a merciful release. This is unlikely to be delayed beyond eighteen months after the disease is first recognised, and is due to either septic bronchopneumonia, hæmorrhage, cachexia as a result of starvation, or exhaustion from pain and toxæmia.

*Clinical Picture.*—In the **early stages**, a small area of induration with commencing ulceration will be seen. There is little if any pain, no limitation of movement of the tongue, no enlargement of glands and no impairment of the general health. The patient complains of a curious feeling of stiffness in the affected area, and of a little smarting pain on taking irritating food and when smoking.

In the **intermediate stages**, the ulcer is large and necrotic (Fig. 169), the breath fœtid, and there is profuse salivation. Movement is restricted in the affected side, so that when the tongue is protruded it swings towards that side; because of this limitation of movement, speech is

blurred and difficult to understand. Pain is becoming a prominent feature, both in the tongue itself and in the referred area of the Vth cranial nerve, especially in the distribution of the auriculo-temporal branch, that is, to the ear, the temporomandibular joint and the temple. Enlarged lymphatic glands will be present in the primary drainage area.

In the **later stages**, the tongue has become fixed to the floor of the mouth and a large septic ulcerating growth is present. Eating and swallowing are so painful that the patient hardly dare take any food.



FIG. 169

Carcinoma of the tongue.

Saliva trickles steadily from the mouth and small hæmorrhages come from the growth at the slightest touch. The lymph glands are greatly enlarged and may be breaking down. Pain is severe and the whole state constitutes one of the saddest pictures in malignant disease.

*Differential Diagnosis.*—The diagnostic table (p. 341), serves to give the differential diagnosis in the case of the malignant ulcer. The sub-mucous nodule before ulceration should give rise to no difficulty because of its induration. Doubt should *never* be allowed to enter into this question. If there is the *slightest* uncertainty in the practitioner's mind, a piece of tissue must be removed for microscopy.

*Treatment.*—At the present time it is not easy to give an authoritative statement on the treatment of lingual carcinoma. Radium has not yet gained universal acceptance among surgeons as the method of choice, and many still advocate radical removal of the tongue, relegating radium to a subsidiary position for inoperable growths. We, however, are convinced that radium has rather more to offer than surgery in the treatment of the primary lesion. Radium therapy should have a negligible operative mortality, it is not a mutilating procedure, and the function afterwards is good. The radium technique will be briefly outlined as well as some of the accepted operative methods.

**Preliminary Treatment (all methods).**—Intensive treatment must be directed to the eradication of every cause of buccal sepsis. The teeth must pass a dental surgeon's examination or be removed. Constantly repeated antiseptic mouth-washes are used during the day, and it is wise to insist upon the patient being in bed in a nursing home for at least four days before operation.

**Radium Therapy to the Tumour.**—For growths in the anterior two-thirds of the tongue, radium needles (0·5, 1 or 2 mg.) are inserted into its substance beneath the tumour, as shown in Fig. 170. They are stitched in position and the silk sutures are brought out of the

mouth and fixed to the face by strapping. The dose must vary with the size of the growth, but an average exposure will be about 1750 mg. hours, and owing to the small number of needles that can be usefully employed, they will have to be left in position for from seven to ten days. Patients suffer considerable discomfort, but severe pain should be absent. Diet will have to be limited to fluids for the first four days, and soft semi-solids afterwards. A careful watch is kept each day to estimate the reaction. It is wise to have a general anæsthetic for the insertion of the needles, but they can be removed under evipan.

Growths in the posterior or pharyngeal part of the tongue are preferably treated by the bomb (Cade), because it is exceedingly difficult to gain an adequate enough view or access to be certain that the needles have been inserted in such a way that they are irradiating the whole growth.

**Treatment of the Glands** is an absolutely essential part of the technique. It should be postponed for fourteen days after the insertion of the needles, and must *never* be carried out *before* the irradiation of the primary tumour.

A. Glands not palpable. External irradiation is obtained either by a radium collar or deep X-ray therapy.

B. Glands palpable but operable. A complete block dissection of one or possibly both sides of the neck, followed by prophylactic X-ray exposures, constitutes the best treatment available.

C. Glands palpable and inoperable. Cade advises the combination of interstitial needling and external irradiation.

**Operative Treatment.**—1. Growths near the tip. The anterior half of the tongue is removed by dividing it with the diathermy needle; and either at the same time or preferably after ten days, the sub-maxillary and submental glands of both sides of the neck are completely dissected away. Prophylactic X-ray therapy to the deep cervical glands follows.

2. Growths of the anterior two-thirds on or near to the edge. The tongue is split down the middle line and the anterior two-thirds of the affected side removed, diathermy again being used. Ten days later, a block dissection of the cervical glands of the same side follows. This amounts to a modification of the original Whitehead's operation, and is always to be preferred to Kocher's extrabuccal removal.

3. Growths near the middle line. The whole of the anterior two-thirds of the tongue must be removed and the glands on both sides of the neck are dealt with ten days later.

4. For posterior growths. Syme's operation entails the splitting of the lower jaw through the symphysis and gives access to the root of the tongue and the attachments of its muscles to the hyoid bone. The whole tongue is removed.

**Inoperable Growths** should invariably be treated with radium.

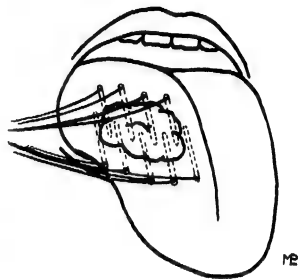


FIG. 170

Diagram illustrating the method of implanting radium needles in the tongue.

The prognosis is inevitably hopeless, but an effort must be made to clear up the growth in the mouth with the object of saving the patient from some of the worst of his pain and sepsis.

SARCOMA is a rare disease of the tongue, occurring either in children or adults. It may be a round-celled fibrosarcoma, but rare cases of rhabdomyosarcoma have been recorded. It may form a large swelling protruding from the surface (Fig. 171) or give rise to a generalised enlargement of the tongue.

*Treatment* is by radium therapy.

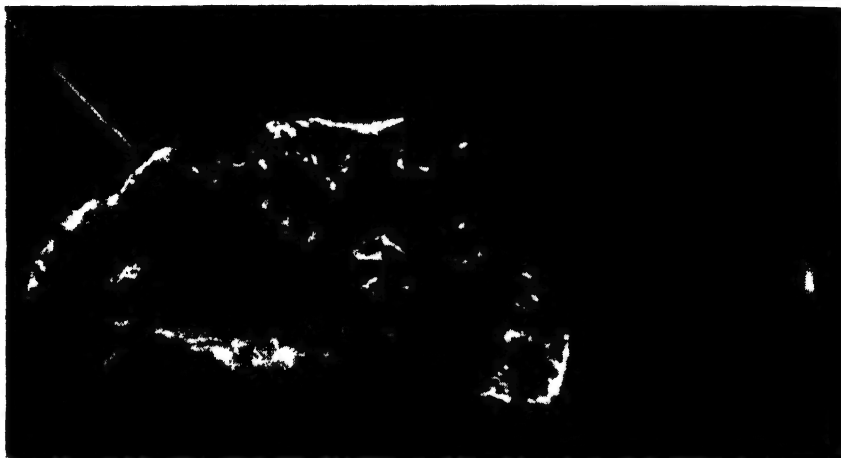


FIG. 171

A sarcoma of the tongue.

LINGUAL CANCEROPHOBIA.—So many patients come for advice, who present a perfectly normal tongue for examination, that a few words of advice may prove helpful. The majority are women, who are haunted by the fear of cancer and, having discovered their circumvallate papillæ for the first time, imagine these to be a tumour. Others have found cracks and fissures so infinitesimal that the professional eye cannot detect them, whilst others complain of neuralgic pain. If the underlying phobia is recognised, the patient can usually be readily convinced of her safety when the comparative immunity of her sex to cancer of the tongue is put to her; but a great deal of harm can be done if the phobia passes unsuspected and palliative treatment ordered, for the patient is then more than ever convinced that she has cancer and that the doctor is afraid to tell her or that he has failed to recognise its presence.

CARCINOMA OF THE FLOOR OF THE MOUTH is so closely allied to lingual carcinoma that it is best described here. It takes the typical form of the squamous-celled ulcer, which spreads into the frenum and so reaches the tongue. It does not appear to have so high a mortality rate as cancer of the tongue, but it is important to recognise it before it has invaded that organ or infiltrated the periosteum of the lower jaw.

*Treatment* consists in removal of the anterior third of the tongue,

the floor of the mouth and the alveolar margin of the mandible corresponding to the eight central teeth. The glands of the submaxillary and submental regions are removed at the same operation. The use of radium in these cases is contraindicated as it is difficult to protect the lower jaw from necrosis. The operation is followed by X-ray therapy to the deep cervical glands on both sides of the neck.

## THE SALIVARY GLANDS

*Surgical Anatomy.*—**The Parotid Gland** fills in all the spare space between the mastoid process and the ramus of the lower jaw. It rests on the styloid process and posterior belly of the digastric muscle, is limited above by the zygoma and the external auditory meatus, and in front it spreads out over the masseter muscle. It is enclosed in a capsule derived from the deep cervical fascia, and so firm is the anterior layer of this capsule that swelling of the gland is rendered difficult.

The parotid gland is intimately related to the facial nerve, which runs through its substance, in which it divides into its many branches. The deeper part is in contact with the IXth, Xth, XIth and XIIth cranial nerves and the termination of the external carotid artery.

Stenson's duct leaves the anterior margin of the gland, runs over the masseter to gain its anterior edge, round which it dips to pierce the buccinator muscle and so open on the mucous membrane of the mouth opposite the second molar tooth of the upper jaw. It lies one finger's breadth below the zygoma and can be palpated when the masseter is made tense.

**The Submaxillary Gland** lies under cover of the body of the lower jaw in its posterior half. It has two parts, a large superficial portion lying beneath the deep fascia and a small deep lobe under the mylohyoid muscle. Wharton's duct passes forwards from this deep portion to enter the floor of the mouth and opens on the summit of a papilla on one side of the frenum of the tongue. The submaxillary gland is intimately associated with lymphatic glands which lie in the hollow between its two parts.

**The Sublingual Gland** lies beneath the mucous membrane of the floor of the mouth on either side of the frenum. Several ducts of Rivini open in the mucous membrane of the floor of the mouth.

*Methods of Examination.*—In addition to surface palpation, bimanual examination with one finger in the mouth is of great help, especially for the submaxillary gland. In cases of doubt a "sialogram" taken after injection with uroselectan gives valuable information (Fig. 172).

**Injuries of the Salivary Glands** are not uncommon, but as they usually heal without complications, they provide little surgical interest, except in the case of the parotid gland, which cannot be removed



FIG. 172

Right-sided parotid "sialogram."

owing to the number and the importance of the structures which traverse it.

**FISTULA OF THE PAROTID GLAND** is usually due not to a wound but to suppuration in the gland around a calculus or in association with an ascending parotitis. An incision for drainage having been made, a sinus persists after the wound has otherwise healed and clear saliva is discharged, particularly at meals. This type of fistula usually heals spontaneously, though very slowly. It is wise to accelerate healing by cauterisation with a diathermy needle, and if this should fail, irradiation with radium will quickly bring about the desired result.

**FISTULA OF STENSON'S DUCT** is a far more difficult problem, but is fortunately a rare occurrence. It is almost always traumatic in origin, the duct being cleanly severed, and after a time its buccal end becomes shrunk and atrophic. The best treatment is immediate end-to-end suture over a few strands of silkworm gut, which protrude into the mouth. But this is not often possible because the salivary injury is not recognised till later. When the fistula lies in front of the masseter, the duct should be freely opened into the mouth by incising the buccal mucous membrane widely. The saliva flows without hindrance into the mouth and the cutaneous opening should heal.

If the fistula is on the surface of the masseter near the gland, the problem is one of great difficulty. Plastic operations are to be attempted, gradual reconstruction of the distal part of the duct with a small rubber tube being later followed by closure of the fistula. If all methods fail, the discharge can be finally cured by avulsion of the auriculo-temporal nerve.

#### **Inflammation of the Salivary Glands—THE PAROTID GLAND.—**

A. *Epidemic Parotitis*, or Mumps, is an acute infectious fever, and its description will be found in textbooks of medicine.

B. *Simple Parotitis* is a subacute catarrhal inflammation of one or both glands as a result of cold, injury, or the presence of a calculus. The gland swells up, is painful and tender. There is a mild pyrexia and slight constitutional disturbance, and within a few days the condition has subsided.

C. *Acute Suppurative Parotitis* is more serious. The organisms reach the gland either by ascending Stenson's duct from the mouth, by direct spread from the jaw or other neighbouring structure or by the blood stream in typhoid, scarlet fever and pyæmic diseases. It is still occasionally seen (but far less so than in the past) as a complication of abdominal operations as a result of extreme dryness of the mouth, lack of oral feeding and inattention to buccal hygiene.

The gland enlarges rapidly and becomes very painful and tender. The skin is red and oedematous. Owing to the density of the capsule, fluctuation as a clinical sign is very late in appearance. All movements of the jaw are painful, constitutional disturbances are marked and the patient is seriously ill. If outlet is not given to the pus, it will track in several directions, all of them dangerous; for example, toward the external auditory meatus, the pharynx, the base of the skull, the deep muscles of the neck and even possibly towards the mediastinum.

*Treatment.*—It is obvious, therefore, that it is most unwise to wait

for fluctuation before making an incision. The pus must be given thorough drainage, and all loculi must be broken down by digital manipulation.

THE SUBMAXILLARY AND SUBLINGUAL GLANDS may likewise be the seat of acute inflammation, but less frequently than the parotid. The infection may be secondary to oral sepsis or to the presence of salivary calculi. Early incision is advisable lest œdema of the glottis or Ludwig's angina appear as dangerous complications.

**Von Mickulicz' Disease** is a rare condition in which all the salivary and the lachrymal glands take part in a slow, painless and symmetrical enlargement. The etiology is unknown, except that in a very few patients there has been a definite association with lymphatic leukæmia.

Except in these cases treatment is by X-ray therapy.

**Salivary Calculi** form most commonly in the submaxillary gland or its duct, and somewhat rarely in the duct of the parotid gland. They are composed of calcium phosphate and carbonate deposited on a nucleus of mucus and epithelial debris. They show some tendency to occur in members of the same family.

STONES IN THE DUCTS do not cause complete obstruction as a general rule, so that saliva can leak past them except at times of great activity, namely, at meals. The symptoms are attacks of pain along the duct, especially during the taking of food, and a rapid swelling of the gland as soon as the meal is commenced. This swelling slowly subsides in the interval between meals, and some patients learn to hasten this subsidence by pressure upon the gland. On examination, the mouth of the duct will be seen to be red and œdematous, the calculus can be felt between the fingers or by a probe passed up the duct, and it is rarely necessary to have an X-ray photograph.

*Treatment* consists in removal of the stone by making a small incision over it in the duct through the mucous membrane of the mouth.

STONES IN THE GLAND SUBSTANCE.—Stones form in the submaxillary gland more frequently than is usually thought, and their clinical picture does not include the somewhat dramatic swelling at each meal time. They give rise to a dull aching pain at first, and then later recurrent attacks of subacute or chronic sialoadenitis occur. Slowly the gland becomes enlarged, thickened and painful, and is so fibrous that it may be impossible to say confidently that the stone can be felt, an X-ray photograph being needed to confirm its presence (Fig. 173).

*Treatment* consists in removal of the submaxillary gland.

If stones are allowed to remain either in the ducts or in the glands for any length of time, there is always the danger of an acute ascending infection from the mouth. This will cause an acute sialoadenitis with or without pus formation, and for this reason all salivary calculi should be removed as soon as possible.

**A Ranula** is a cystic swelling in the floor of the mouth to one side of the frenum. Its exact origin is undecided, but it has no connection with the salivary glands, since its contents are devoid of salivary ferments. It arises probably as a retention cyst in the glands of



Blandin and Nuhn or in that of Bochdalek, or it may possibly be an error in development of the mucous membrane of the floor of the mouth. It forms a soft, fluctuating, smooth, round swelling which may be so large as to displace the tongue upwards and to one side. Its characteristic blue colour serves to distinguish it from a dermoid cyst.

*Treatment.*—The wall is so thin that it is not possible to excise the cyst intact. The mucous membrane over it is incised and the projecting walls of the cyst are removed with scissors flush with the



FIG. 173

A, a patient with chronic sialadenitis of the left submaxillary gland due to a calculus shown in an X-ray, B.

level of the floor of the mouth. The remainder of the cyst wall is destroyed by diathermy, and the cavity allowed to granulate from the bottom.

**Growths of the Salivary Glands.**—Benign growths are limited to the so-called “Mixed Parotid” tumour, unfortunately named in that it is neither pathologically mixed nor anatomically confined to the parotid gland.

**THE SALIVARY GLAND ADENOMA** is quite common in both sexes. It does not usually appear before the age of 25 years and is of exceedingly slow growth. Although most frequently seen in the parotid, it may arise in the other salivary glands and in the lachrymal glands, whilst ectopic tumours may occur beneath any part of the buccal mucous membrane where mucous glands exist.

The tumour has a smooth lobulated surface, is white or gray in colour, firm and elastic in consistence, and on cross-section bears a resemblance to the fibro-adenoma of the breast (Fig. 174). Mucoid degeneration may cause many small cystic spaces or the whole swelling may be converted into one large cyst. This latter type is usually met with in that part of the parotid gland lying deeply behind the ramus of the mandible.

*Microscopically*, the tumour has the character of a true adenoma



of mucin-secreting epithelium. The areas of mucoid degeneration present certain staining reactions which bear a superficial resemblance to cartilage, and this accounted for the theory of their "mixed" origin.

*Clinically.*—After many years of very slow growth they may suddenly take on rapid growth, suggestive of a malignant change. They present no symptoms, and cause no disability save a somewhat unsightly swelling.

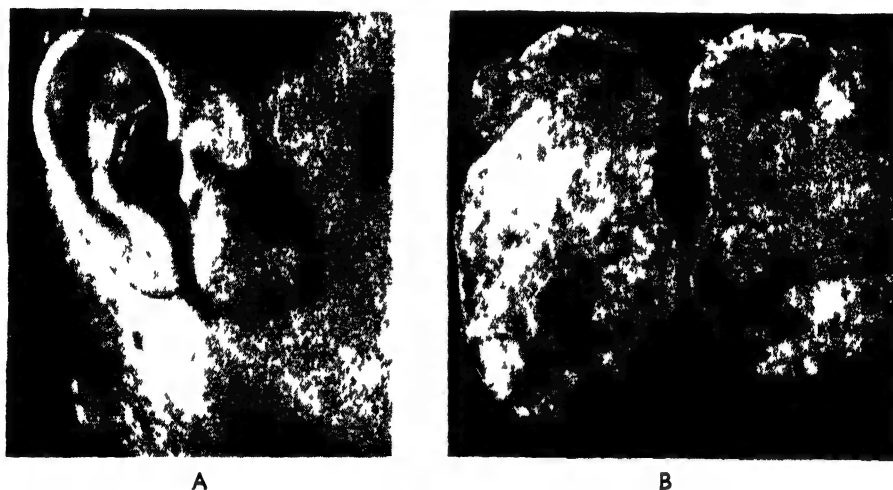


FIG. 174

Salivary gland tumour.

A, before removal; B, after removal, showing cut surface.

*Treatment.*—The adenoma should always be removed in spite of its slow growth and apparent benignity. Although the tumour has a definite capsule, it is fairly firmly adherent to the gland tissue, and careful dissection is needed. It is essential that the whole adenoma be removed and no part of the capsule left behind, but it is equally important that injury to the facial nerve be avoided. If any doubt exists concerning the completeness of the removal it is wise to leave one needle of radium in the cavity for five days; in this way a recurrence can be definitely prevented.

MALIGNANT GROWTHS are exceedingly rare. The majority arise as a malignant change in a previously existing adenoma, and the others as spontaneous growths. The signs of malignancy in the parotid are the hardness of the swelling, pain and the involvement of the facial nerve. It is most unlikely that any treatment will be possible except X-ray or radium therapy.

The salivary glands, especially the submaxillary, are liable to invasion by other malignant processes, such as carcinoma of the tongue and the floor of the mouth.

R. M. HANDFIELD-JONES.

## CHAPTER XIX

### THE SURGERY OF THE NECK

**A**NATOMY.—The reader is referred to textbooks for the detailed anatomy of the neck, and only certain relationships of surgical significance are described here. The Cervical Fascia envelops the neck and, sending two fibrous sheets across it, forms three compartments. The enveloping or superficial layer is attached behind to the ligamentum nuchæ, splits to enclose the trapezius, joins again to roof in the posterior triangle, and splits to enclose the sternomastoid muscle. At the anterior margin of this muscle the fascia spreads across the middle line to meet the other sternomastoid and so forms the fascial space of Burns. The prevertebral layer passes in front of the spinal column and its muscles, and the pretracheal layer extends across the neck in front of the larynx, trachea and thyroid gland, giving off subsidiary lamellæ to form the carotid sheaths. The anterior compartment is therefore purely muscular, leads to the bones of the shoulder girdle and has no connection with the chest. The middle compartment contains the pharynx, larynx, trachea, thyroid gland and carotid sheaths, and communicates with the superior mediastinum. The posterior compartment contains the spine, vertebral muscles and nerves ; it forms the posterior wall of the superior and posterior mediastina. The significance of these fascial paths of connection with the chest lies in the possibility of the spread of infection from the neck.

The development of the neck presents certain important features. In the third week of intra-uterine life pairs of branchial arches are formed in the post-oral area with clefts between them. They consist of mesoblast and are covered with epithelium on either side. Fusion with each other and across the midline with those of the opposite side occurs quickly. If this fusion is imperfectly completed various anomalies are seen in the neck. The component parts of the head and neck which arise in the various arches and clefts are :—

1. From the 1st arch, the mandible, the processus gracilis of the malleus, the mandibular division of the Vth nerve and the muscles of mastication.
2. From the 1st cleft, the Eustachian tube, tympanic cavity, external auditory meatus and the Glasserian fissure.
3. From the 2nd arch, the styloid process, stylohyoid ligament, lesser cornu of the hyoid bone, VIIth nerve and the muscles it supplies.
4. From the 3rd arch, the body and great cornu of the hyoid bone, IXth nerve and its muscles.
5. From the 4th arch, the remaining structures of the neck.
6. The 2nd, 3rd and 4th clefts are obliterated, but the 2nd is represented by the fossa of Rosenmuller and the 3rd by the pyriform fossa of the larynx.

**ANOMALIES OF DEVELOPMENT****BRANCHIAL FISTULA**

These fistulæ or sinuses, also known as Lateral Fistula of the Neck, or Persistent Cervical Sinus, are due to imperfect closure of the branchial clefts. Only rarely are they true fistulæ opening into the pyriform fossa or nasopharynx, being usually sinuses opening on the skin along the anterior border of the sternomastoid muscles near the clavicle, and passing upwards, inwards and backwards for a limited distance. They are lined with columnar epithelium, and secrete a scanty, glairy, mucoid fluid. They are usually present at birth, and are sometimes associated with other anomalies, such as accessory auricles and facial clefts. The majority will require no treatment, but if the discharge is a source of worry they should be dissected out. If the fistula is complete, the pharyngeal end must be invaginated.

**BRANCHIAL CYSTS**

During the process of closure of the clefts, small islands of cleft membrane (either 2nd or 3rd cleft) may be cut off and left in the developing mesoblast. These cell inclusions may be derived from the external cleft membrane (the future skin) or the internal membrane (the future lining of the pharynx and larynx). If later they should grow they form branchial cysts, which have thin walls and are lined either by squamous or columnar epithelium. The contents of the former are thick semi-solid sebaceous matter rich in fat and cholesterin crystals, and of the latter a glairy mucoid fluid. They may retain an attachment to the wall of the pharynx by a thin fibrous cord which passes between the internal and external carotid arteries.

*Clinically* they become obvious between the tenth and twenty-first year and are often preceded by an injury, which presumably galvanises the cell inclusion into active growth. They are commoner in males and on the left side of the neck. Those derived from the 3rd cleft lie between the anterior border of the sternomastoid muscle and the lateral ala of the thyroid cartilage and reach the greater cornu of the hyoid bone. The rare 2nd cleft cyst is higher up in relation to the mastoid process and the jaw. These cysts are never really tense and fluctuation is readily detected, but one of their chief characteristics is that they vary in size from time to time. They have no fixed attachment in the neck, and lose their marked mobility only when they become infected. The correct treatment is removal, as they may become the seat of a branchial carcinoma.

**THYROGLOSSAL CYSTS**

The thyroglossal duct (p. 363) is a solid column of cells stretching from the foramen cæcum of the tongue, through the geniohyoglossi muscles to reach the hyoid bone, anterior to which it passes in front of the larynx and thyroid isthmus. This tract should disappear

completely but may persist in two situations, viz., the tongue and the neck.

In the tongue a nodule of thyroid tissue may be present near the foramen cæcum, forming a dark red mass beneath the mucous membrane. It is discovered usually during routine examination of the mouth and throat for some buccal or pharyngeal disease. This nodule may be the only thyroid tissue present, and must not be removed until the presence of a normally placed gland is confirmed.

In the neck, thyroglossal cysts (Fig. 175) are seen in the midline, either just above or, more commonly, below the hyoid bone. The cyst is lined with embryonic thyroid tissue and contains a colloid substance. It appears in children of both sexes about the age of five, and presents a fluctuant, rounded, smooth swelling over which the skin moves freely. It may be firmly fixed to the hyoid bone, and if so, will move on swallowing. Those above the bone in the muscles of the tongue will move more obviously upwards and backwards when the tongue is protruded. If opened or allowed to burst, a median cervical sinus is formed.



FIG. 175

A thyroglossal cyst.

*Treatment* consists in complete removal, and the operation is apt to be tedious and difficult, the percentage of recurrences being high. If the cyst is attached to the hyoid bone, then no effort should be made to dissect it free, but the body of the bone excised with the cyst.

### CERVICAL RIB

A supernumerary rib arises usually from the 7th cervical vertebra and very rarely from the 6th. This cervical rib may be little more than an exaggeration of the costal element of a transverse process; it may be a short fine rib projecting hardly beyond the scalene muscles; it may pass downwards and forwards, being connected with the first rib by either a short fibrous cord or true bony fusion; and lastly it may have a true costal cartilage uniting it with the sternum. The latter two varieties emerge between the scalenus anticus and medius muscles, the former of which usually gains an attachment to the abnormal rib. The brachial plexus and the subclavian vessels pass over it (Fig. 176).

Cervical ribs are frequently bilateral; when unilateral they are more common on the left side, but symptoms are more often present on the right. Women are more frequently affected than men. Symptoms are absent in many patients, the rib being discovered in a routine examination, but in any event they do not appear until the age of 18 years. This is explained by the gradual descent of the shoulder girdle upon the thoracic cage which occurs during adolescence. Sargent found that symptoms were usually more severe upon the side of the smaller rib owing to the more intense compression by the narrow taut fibrous cord.

*Symptoms* are grouped as sensory, motor and vasomotor. They are aggravated by carrying heavy weights, certain types of work, wearing heavy clothes and certain games, *e.g.*, golf, and are relieved by rest and elevation of the arm.

*A. Sensory.* Patients frequently complain of tingling in the hands and fingers, particularly in the tips of the latter. These symptoms may be unilateral, a point of considerable importance in diagnosis. They are referred to either the ulnar or radial side rather than to the whole hand. Pain is felt in the forearm, hand or fingers radiating in a downward direction. It is sharp and lancinating and may be brought on by a sudden rotation of the head or a forceful downward pull of the shoulder. On the other hand it may be dull, aching or burning in character occurring late in the day when the patient is tired.

General sensibility is impaired and there may be actual anæsthesia. This does not always coincide with radial or ulnar distribution or indeed with a typical root supply.

*B. Motor.* There is an increasing loss of power in the hand with inability to perform fine movements. The muscles affected may be either

those supplied by the medial or ulnar nerve. They show wasting to a variable extent—sometimes to a marked degree.

*C. Vasomotor.* These are due to pressure on the sympathetic fibres in the lower roots of the plexus rather than to direct compression of the nerves themselves. The affected forearm and hand are cold and assume a dusky hue, and there are mild trophic changes in the finger-tips. A diminution in the volume of the radial pulse is sometimes noted and this may be accentuated by inspiratory movements of the chest and relieved by raising the arm above the head. Gangrene of one or more fingers has been described. Occasionally the subclavian artery may be seen pulsating above the clavicle.

*Diagnosis.*—Similar symptoms are often seen in patients with syringomyelia and progressive muscular atrophy. A careful clinical investigation should arrive at a correct diagnosis which is confirmed by X-rays. Most abnormal ribs can be demonstrated in the films.

*Treatment.*—When a cervical rib is causing no symptoms, treatment is not indicated. Young people in whom there are slight symptoms



FIG. 176

A well-marked right cervical rib, short rudimentary one on the left side.

benefit greatly from exercises designed to increase the tone and power of trapezius and levator scapulæ muscles. The resulting bracing-up of the shoulder girdle relieves the strain upon the nerve as it crosses over the abnormal rib.

In cases with severe symptoms the rib must be removed or the fibrous band divided.

### THE SCALENE SYNDROME

A clinical picture suggestive of cervical rib is not infrequently seen in patients who present a normal X-ray appearance and in whom an abnormal band is not found. Symptoms are rarely so severe as with a true cervical rib and they appear usually at a later age, often about 40 years. Tingling pain down the arm is sometimes marked especially after active exercise, and such routine daily occupations as needle-work, knitting, car driving, etc., may be curtailed. This syndrome is due to one of two conditions. The majority of these patients have a short contracted scalenus anticus muscle which elevates the first rib sufficiently to cause mild pressure upon the nerve; the remainder suffer from an abnormality of the brachial plexus and not of the ribs. In a "post-fixed" plexus a normal first rib bears a comparable relation to the lower cord as does a normal cord to a cervical rib, consequently pressure is exerted by the rib upon the nerve.

*Treatment.*—Many of these patients are relieved if the insertion of the scalene muscle is completely erased from the first rib, which is thus allowed to fall to a lower level. Others will require removal of a major part of the first rib to relieve pressure upon the nerve.

### TORTICOLLIS

Wry neck is characterised by lateral inclination of the head towards one shoulder accompanied by torsion of the neck and deviation of the face towards the opposite side. It may be either congenital, acquired, spasmodic or hysterical.

**Congenital Torticollis.**—It is generally held that during a difficult labour temporary acute obstruction of the veins in the sternomastoid muscle of one side occurs and this may be rendered permanent by intravascular clotting. This latter and the resultant effusion is apparent in infancy as the so-called "sternomastoid tumour." The swelling eventually disappears, its place being taken by fibrous tissue which later contracts. The mechanism, therefore, is comparable to that which produces ischæmic contracture of the forearm. During difficult labour, especially a breech presentation, one sternomastoid muscle may be torn and a hæmatoma forms with similar sequelæ to those described above. It can hardly be said, therefore, to be truly congenital and it is doubtful if any case can be traced to a developmental defect.

*Clinical Features.*—In the first few weeks of life an elongated swelling is seen in the lower half of the affected sternomastoid muscle. This is tender and the child cries when the muscle is stretched or palpated. Both swelling and tenderness disappear slowly, but towards the end of the first year the muscle is seen to be unduly tense owing

to the fibrous tissue in its substance. As this contracts still further the head is drawn down so that the ear on the affected side is pulled towards the sternoclavicular joint, while the face is rotated to the opposite side. If treatment is not instituted early, a gradual atrophy of the face on the affected side develops. All soft parts on this side shorten, while the bones of the cervical and upper thoracic spine acquire a fixed scoliotic deformity.

The condition is easily diagnosed. In many cases there is a history of difficult birth, followed by a tender swelling in the neck. Later the typical deformity of the sternomastoid is obvious.

*Treatment.*—In mild cases the deformity may be prevented if treatment is given at an early stage. It is not wise to manipulate and stretch the tender muscle, but as soon as the child is strong enough the tumour should be excised. In very slight cases manipulation and exercises are sufficient.

If the child is not seen until after the age of 2 years an open operation is necessary. The contracted part of the muscle is divided  $\frac{1}{2}$  in. above the clavicle and the cervical fascia may need similar treatment. The head is placed in an over-corrected position and retained there by bandage or plaster (Fig. 177).

During the six months following operation, active and passive exercises must be carried out to prevent any recurrence of the deformity.

**Acquired Torticollis** is due either to "rheumatic" fibrositis following exposure to colds or draughts (the common stiff neck) or to reflex causes, such as inflammatory lesions of adjacent lymph glands or spinal caries of the cervical vertebræ. It is always essential that these causes should be eliminated before treatment is considered.

**Spasmodic Torticollis.**—This type occurs in adults and is a very obvious and distressing condition. The sternomastoid and trapezius on one side acting in conjunction with the posterior rotators on the other produce violent jerking movements of the head, which is suddenly and forcibly pulled into the typical torticollis position. At first some control can be exercised by the patient, but later movements are quite involuntary and may even spread to other muscles, such as those of the shoulder and face.

This condition is undoubtedly of organic origin, the lesion being either of the peripheral nerves or more probably of the midbrain. Roger and Pourtal found that five of their patients had lesions of either the pyramidal tract or the extrapyramidal system.

*Treatment.*—Sedative drugs and re-educative exercises may afford some relief, while fixation of the head in a plaster jacket may help. In severe cases operation should be advised; this consists in resection of the spinal accessory nerve of one side and of the posterior primary divisions of the first three cervical nerves on the other. This has given great relief in a certain number of patients.



FIG. 177

Torticollis, showing method of application of bandage after operation.

**Hysterical Torticollis.**—Habit spasms and certain jerking movements of the head are sometimes seen in hysterical young women. A careful analysis of these movements will show that they rarely conform exactly or consistently with those of spasmodic wry neck. Treatment is directed to the underlying neurosis.

### INFLAMMATORY CONDITIONS IN THE NECK

**CELLULITIS OF THE NECK (Ludwig's Angina).**—This is either a streptococcal infection having origin in some focus in the teeth, tongue, floor of the mouth, jaw, tonsils, larynx or pharynx, or it may be a complication of an acute infectious fever, *e.g.*, scarlet fever or diphtheria. The infection first passes to the submental or submaxillary lymphatic glands and then spreads in the tissue planes below the jaw. It is characterised by a brawny oedema in which pus forms slowly and in small amounts. If unchecked, infection spreads throughout the neck with grave symptoms of constitutional involvement. Eventually extension occurs into the mediastinum, leading to mediastinitis, pericarditis and empyema. Other complications which may usher in a fatal ending are septic venous thrombosis, pyæmia, meningitis and oedema of the glottis.

*The Clinical Picture* in the early stages shows a dusky red swelling beneath the jaw with brawny oedema. Although pus forms, it will rarely give fluctuation and pointing does not occur. The patient is in great pain and the swollen area is exquisitely tender. There are high fever, rapid pulse and rigors. Oedema of the glottis is an ever-present danger, which may at any time become urgent and demand a tracheotomy.

*Treatment.*—The original focus should be identified and dealt with. The local condition may be treated at first by hot dressings, short-wave therapy and sulphapyridine, but if the swelling is increasing, one or more incisions must be made. No inhalation anæsthetic is safe, and pentothal should be used. Several small incisions are better than one large one; they must go through the deep fascia and pus be sought for. Even if no pus is found they allow a profuse drainage of infected blood-stained serum. In severe cases the general condition of septicæmia is grave and demands active treatment.

(Boils, carbuncles and other infective processes will be found under their specific headings in other chapters.)

### INJURIES

**Cut-throat.**—Cut-throat may be suicidal or homicidal. Owing to the medico-legal aspect, exact observations of each case must be recorded. In attempting suicide by this method, the victim throws back his head, and in so doing renders the air passages more prominent and tenses the sternomastoid muscles, behind which the carotid vessels obtain some protection. A right-handed man begins the cut on the left side, draws the knife straight across the midline, and tends to



finish the cut in an upward direction on the right side. Unless he is very determined the cut becomes shallower as it progresses. A left-handed suicide produces his injuries in the reverse direction. In a homicidal case the extent and nature of the injury depends on the relative position of assailant and victim and on the hand used. Most attacks are made from behind and the cut is usually shallower at its commencement and deeper at the end, or it is of equal depth throughout. The wound is more severe than in suicidal cases and the carotid vessels are more likely to be severed. In some cases the knife has reached the vertebral column and even entered the inter-vertebral disc. In superficial cuts all important structures may escape injury, and only the anterior and external jugular veins be opened. If the platysma is divided, the wound retracts and hæmorrhage may be profuse, though easily controlled. In deeper injuries a rapidly fatal hæmorrhage ensues from a severed carotid vessel.

The factor which has most influence on treatment and prognosis is involvement of the air passages. If they escape the problem is simply that of general wound treatment. If they are affected, the condition becomes grave owing to the danger to respiration.

*The Symptoms and Treatment* depend on the site of injury.

**A. Above the Hyoid Bone.**—In this rare injury the extrinsic muscles of the tongue may be divided, the floor of the mouth opened, and the lingual and facial arteries and the hypoglossal nerve severed. The dangers are either immediate in that the damaged and possibly paralysed tongue may fall back and block the entrance to the larynx, or remote when the opening in the floor of the mouth makes infection of the wound certain.

**B. Through the Thyrohyoid Membrane.**—This is the common site of injury. The membrane is divided, the pharynx opened and the epiglottis injured. The lingual, facial and superior thyroid arteries and the hypoglossal nerve are likely to be cut. Respiration will be unaffected unless either the injured epiglottis or loose folds of mucous membrane obstruct the larynx, or blood trickles down into the trachea. Speech and swallowing are painful and difficult, and food will leak into the neck, rendering severe sepsis a probable sequela.

**C. At the Level of the Larynx.**—Wounds in this situation are seldom severe as the density of the cartilage prevents the knife entering deeply. The vocal cords may be affected and bleeding from the superior pole of the thyroid profuse. Speech is difficult and painful, and blood may enter the trachea.

**D. Through the Trachea.**—The narrowing of the air passage, which takes place at the origin of the trachea, allows the main vessels to swing in towards the middle line. Injuries in this position therefore tend to produce serious effects. Hæmorrhage may occur from the vessels of the thyroid gland and from the carotid and jugular trunks, and will prove rapidly fatal. Blood may enter the trachea and be aspirated into the lungs, and air be sucked into the great veins. The œsophagus and recurrent laryngeal nerves may be injured, and if the skin wound and the tracheal opening do not correspond, surgical emphysema results.

In all wounds of the neck the immediate dangers are hæmorrhage and suffocation. The later complications are sepsis in the planes of the neck and bronchopneumonia. Sepsis in the neck may be introduced by a dirty weapon, or come from the mouth, pharynx or air passages. The lungs are affected by aspiration of blood and food. The mortality is high in suicide cases, because these unfortunate people are usually in no state to combat a general or a pulmonary infection. Later sequelæ include tracheal and laryngeal stenosis and fistula from the œsophagus or air passages.

*Treatment.*—The general principles of wound treatment and arrest of hæmorrhage are applicable here. Excision of the damaged tissues is carefully performed and the wound cleansed, but although it may be sutured in layers, drainage *must* be provided. The wounds in the air passages will be treated as follows :—

1. Wounds through the thyrohyoid membrane and above the hyoid bone. The epiglottis is carefully sutured and the mucous membrane repaired, after which the various layers are sewn up with drainage. If respiration is embarrassed, a high tracheotomy is performed.

2. Through the larynx. The wound is sutured and a high tracheotomy performed.

3. Through the trachea. A tracheotomy tube is inserted in the tracheal wound and the opening sutured if necessary. The rest of the wound is sewn up in layers. Wounds of the œsophagus should be sutured and a drainage tube put down to the site. Difficulty in swallowing may be due to injury of the epiglottis and loss of sensation in the pharynx and larynx. If any trouble is threatened, an œsophageal tube should be used for feeding or a gastrostomy performed.

These patients are nursed in a semi-reclining posture, with the head well flexed on to the chest. The usual general remedies for shock and loss of blood are employed and a close watch kept for the earliest signs of sepsis, counter-openings for drainage being made if needed. Precautions must be taken to prevent suicidal patients from interfering with the dressings.

**Other Injuries in the Neck.**—Contusions of the skin of the neck have little significance, except from the medico-legal aspect. Pressure of the fingers and nails usually leaves marks which can be recognised, and a cord tied round the neck for strangling or hanging bruises the skin in a peculiar manner. Direct blows cause widespread bruising, which may be very extensive in “run-over” accidents.

The subcutaneous lesions may be restricted to hæmorrhage beneath the cervical fascia, but be sufficient to cause urgent dyspnœa and dysphagia. In the more severe injuries, the laryngeal cartilages and trachea may be bruised or torn, leading to immediate or delayed suffocation, and in all such injuries the danger of œdema of the glottis cannot be dismissed for forty-eight hours. Fracture of the cartilages, when calcified, occurs but rarely, and then in old people. The treatment of all subcutaneous injuries in the neck is expectant, a tracheotomy being performed at any time within the first few days if required.

## CYSTS IN THE NECK

These may be classified as follows :—

<b>A. In the Middle Line of Neck</b>	Developmental (Salivary)	{ Dermoids. Thyroglossal cysts. Sublingual ranula. Adenoma of thyroid isthmus. Subhyoid bursitis. Branchial cysts. Submaxillary retention cysts. Lymphatic cysts.
<b>B. In the Side of Neck</b>	Lymphatic	{ Cystic hygroma. Chronic abscess in glands. Cystic growth in glands.
	Vascular	{ Aneurysm. Venous cyst. Serosus cyst.
	{ Thyroid Parasitic Neoplastic. Aerocele. Pneumocele.	{ Cystic adenoma. Echinococcal.
<b>C. Anywhere in Neck</b>	Sebaceous cysts.	

**Dermoid Cysts.**—Sequestration dermoids are sometimes seen in the midline of the neck between the hyoid bone and the jaw. They are lined with squamous epithelium and contain soft pultaceous matter. They are seen in children, are of small size and are not attached to the skin or deep structures, but may project between the mylohyoid muscles and form a swelling in the floor of the mouth. They can be removed with ease.

**Sublingual Ranula.**—This will rarely push its way between the mylohyoid muscles and appear in the neck. A bilobed swelling is seen with a narrow channel between the buccal and the submental portions. They have all the characteristics of the ordinary ranula (p. 349) and should be dissected out.

**Lymphatic Cysts** (Hydrocele of the Neck).—These are unilocular cysts of moderate size occurring in the lower part of the neck and in the axilla. They are congenital in origin and are seen in children under the age of ten years as soft, lobulated, usually translucent, flabby cysts, which are liable to undergo recurrent attacks of mild inflammation, causing them to become more tense and a little painful. In the quiescent periods they give rise to no symptoms, but nevertheless should be removed.

**Cystic Hygroma.**—Much confusion has existed in the past owing to the vague nomenclature of lymphatic swellings in the neck, and the term "cystic hygroma" is used by some authors to cover all lymphatic cysts. It is better to limit it to a rare condition which is seen in infants, in whom there is an overgrowth of lymphatic tissue, resulting in a large swelling in the neck which is riddled with small cysts. This may stretch from one mastoid process to the other below

the jaw and down to the clavicles, axillæ and mediastinum. Attempts to remove them usually fail owing to their ramifications, but injection of the cysts with sodium morrhuate may succeed in causing a reduction of the swelling.

**Cystic Swellings of the Lymphatic Glands.**—These include acute and chronic abscess and the cystic degeneration of new growths. They are discussed in Chap. XVI.

**Subhyoid Bursa.**—A small bursa occupies the postero-inferior aspect of the hyoid bone, lying between it and the thyrohyoid membrane. When enlarged it may be mistaken for a thyroglossal cyst. It is firmly fixed to the hyoid, moves on swallowing and its long axis is transverse. If it is unsightly or painful it should be removed.



FIG. 178

A carotid body tumour. The common carotid artery is seen bisected and dividing into its two terminal branches.

**Aerocoeles.**—These are diverticula from the larynx or trachea and are not true cysts. They are resonant and reducible, and are popularly supposed to be common amongst trumpeters and the Moham-medan muezzins.

**Pneumatocoeles.**—These are herniæ of the apex of the lung into the supraclavicular triangle.

## MALIGNANT DISEASE IN THE NECK

In addition to growths of the ordinary structures in the neck there are two rare

varieties to be described, branchial carcinoma and tumours of the carotid body.

**Branchial Carcinoma** (Branchiogenetic Carcinoma) is a very rare condition seen in elderly men. It is a squamous-celled growth arising in a branchial-cleft cell inclusion or in a pre-existing branchial cyst. It forms a hard, rapidly growing mass between the sternomastoid muscle and the hyoid bone, becomes fixed to surrounding structures, and then infiltrates diffusely in the upper part of the neck. It is more compact and smooth than a fused mass of secondary malignant glands, and there may be the history of a pre-existing cyst which has suddenly taken on rapid and solid growth. It causes some pain but is otherwise symptomless, and metastasises quite late. Attempts at removal are usually unsuccessful, and radium or deep X-ray therapy should be tried.

**Carotid Body Tumours** are endotheliomata of two varieties. One is a slowly growing vascular tumour described as a perithelioma, and the other is a more malignant and very hard growth named the "potato tumour." They envelop the carotid arteries, the internal jugular vein, the vagus and sympathetic nerves, but in

spite of this they give no symptoms. The diagnosis is usually impossible except by microscopy. Treatment is excision which may have to include important vessels and nerves (Fig. 178). The dangers of cerebral anæmia are so real after this operation that it is better to try the effect of radium or deep X-ray therapy first.

## THE THYROID GLAND

*Anatomy.*—The thyroid gland consists of two lateral lobes and an isthmus. The lobes are conical with their bases downwards and measure about two inches in length, extending from the lower part of the ala of the thyroid cartilage above to the fifth ring of the trachea below. They are moulded to the larynx and trachea on their postero-internal surfaces, while superficially they are smooth and rounded. The isthmus unites the lateral lobes across the front of the trachea at the level of the second and third rings. The whole gland is enveloped in a fibrous sheath derived from the pre-tracheal layer of the deep cervical fascia, which is attached above to the thyroid cartilage. It is covered by the sternohyoid, sternothyroid and anterior belly of the omohyoid muscles. The thick, rounded, posterior border rests on the pharynx or œsophagus and the recurrent laryngeal nerve, and laterally overlaps the carotid sheath. The pyramidal lobe is not always present, being a narrow strip of thyroid tissue arising from the isthmus just to the left of the midline, running upwards along the trachea and thyroid cartilage. It may end in a fibrous cord attached to the hyoid bone—the suspensory ligament of the thyroid gland.

The superior thyroid artery is a branch of the external carotid and, running downwards, meets the apex of the lateral lobe, where it divides into two branches, an external branch passing down the lateral surface to anastomose with branches of the inferior thyroid artery, and an internal branch along the inner border which reaches the upper surface of the isthmus to anastomose with its fellow from the opposite side. The inferior thyroid artery is a branch of the thyroid axis trunk from the first part of the subclavian. It reaches the posterior aspect of the lower pole of the gland dividing into several branches which enter it along its postero-lateral border. Among these branches the recurrent laryngeal nerve ascends to the larynx. The thyroidea ima artery is an inconstant vessel which, arising from the aortic arch or from the innominate artery, reaches the inferior aspect of the isthmus. The inferior and middle thyroid veins collect blood from the lower poles, the left joining the left innominate vein and the right entering one or other innominate vein or the junction between the two. The nerves are derived from the sympathetic plexuses which accompany the arteries, and the lymphatics enter the pre-tracheal and inferior deep cervical glands.

The thyroid gland develops from a median and two lateral buds. The median one arises from the fused ventral ends of the second branchial clefts and grows downward in front of the larynx. It forms the isthmus and the major part of the lateral lobes. The lateral buds are developed from the fourth clefts and form thin caps to the median growth, thus completing the lateral lobes. The track of the developing median bud stretches from the foramen cæcum at the back of the tongue down to the isthmus, thus constituting the thyroglossal duct.

*Method of Examination.*—The patient should be placed in a semi-reclining position with the neck partly flexed and so supported that the muscles are completely relaxed. Difficult cases should be examined from behind so that

the thyroid can be pressed back against the transverse processes by the examining fingers. Thyroid swellings are recognised by their upward movement on swallowing, and as some patients find it difficult to swallow at will, they should be given some water to drink if any doubt exists. A few other swellings move on deglutition, but are so rare that they may be disregarded. The extent and consistence of any swelling are noted and any prolongation into the mediastinum can be mapped out by percussion and by X-rays, which will show the tracheal displacement and possibly a shadow of the swelling. The rate, volume and regularity of the pulse, and the condition of the heart muscle are next investigated. The outstretched fingers are examined for tremors, the eyes for the signs associated with exophthalmic goitre, and finally the basal metabolic rate is estimated. The metabolism of the body may be expressed in terms of heat output, and under conditions of muscular rest and fasting the figure is constant. This constant is known as the "basal metabolic rate" (hereinafter termed the B.M.R.) which may be defined as the number of calories produced per square metre of body surface per hour under standard conditions. Clinically this is estimated indirectly by the amount of oxygen absorbed and carbon dioxide produced in a given time. It is known that the B.M.R. is affected by changes in thyroid activity. It is plotted from a normal constant of zero, plus figures denoting hyperthyroidism and minus figures the reverse. A variation of from  $-10$  to  $+10$  is regarded as being within normal limits.

**Anomalies in Form.**—Accessory thyroid bodies may be found in the deeper areas of the neck, and are developed as offshoots from the median thyroglossal duct. The pyramidal lobe is a persistence of the lower end of the thyroglossal duct, while thyroglossal cysts and sinuses arise in the upper end of the duct.

**Anomalies in Function.**—The absence or deficiency of thyroid secretion in children leads to a form of dwarfism named **cretinism**. Growth is stunted, body fat is increased, particularly over the shoulders, the face is pale, flabby and expressionless, mental development is backward and the children are unclean in habits. If it is recognised before two years of age, thyroid medication will permit the development of an almost normal child, but the later the diagnosis is made the less marked is the improvement and the worse the end result.

Hypothyroidism in adults is produced by the removal of the thyroid gland or by its destruction from disease. The condition is called **myxœdema** in which there is an imperfect removal of mucin from the body, which becomes fat and heavy. The face is white and waxy, the expression dull and vacant, the skin dry and the hair falls out. The tongue is enlarged and sore, and the voice altered. There is a slow pulse, a subnormal temperature and a dulling of all mental faculties. Sexual power and desire are lost. The administration of thyroid extract restores these patients to normal in a wonderfully short time.

Hyperthyroidism is a condition exemplified by exophthalmic goitre and toxic adenoma, under which headings it will be discussed.

### ACUTE THYROIDITIS

This is a very rare disease. Joll reports two cases in over 2000 goitre admissions, and only two patients of this type have been seen in St Mary's Hospital during the last twenty years. There is some

evidence to suggest that a goitrous gland is more susceptible to infection than a normal one. Acute thyroiditis may follow acute infections of the mouth, fauces, pharynx and neck, acute infectious fevers or result from embolism in pyæmia. In almost every example organisms are either blood or lymph borne. Two types are seen, suppurative and non-suppurative.

**Suppurative Thyroiditis** starts abruptly with pain and throbbing, either the whole gland or one lobe becoming swollen and tender. High fever and rigors follow and are rapidly succeeded by difficulty in swallowing, breathing and talking. It is an exceedingly dangerous disease owing to the risk of spread to the mediastinum. Treatment consists in early and free incisions which must provide adequate drainage.

**Non-suppurative Thyroiditis** has a more gradual onset, its symptoms are less severe and after a few days (usually seven to ten) resolution occurs and the gland returns to normal.

### CHRONIC THYROIDITIS

(Chronic pyogenic, tuberculous and syphilitic disease are exceedingly rare and merit no description here.

**Reidel's Disease.**—Ligneous thyroiditis or woody thyroid is uncommon, but is more frequently recognised as its clinical picture and pathological appearance are more widely understood. It is characterised by a dense sclerosis which destroys the gland substance; moreover it shows a marked tendency to spread outside the capsule and involve neighbouring structures, such as the carotid sheath, infrahyoid muscles, trachea and œsophagus. Further it so invades the tissue planes that normal structures become impossible to define exactly. The disease usually starts in the lower pole, either upon its surface, destroying the gland by strangulation, or within its substance, replacing the vesicles by infiltration. It may spread to the whole gland or one lobe may remain unaffected.

Nothing is known of the causation or nature of the disease beyond the fact that it is inflammatory in type and definitely not neoplastic.

**Clinical Picture.**—The sexes are affected almost equally during the years of active adult life. Pain and dyspnoea are the chief symptoms, the latter being severe out of all proportion to the size of the swelling. Dysphagia and interference with the voice are commonly met with. The swelling is not of great size, unless the disease has arisen in a previously goitrous gland, its surface is smooth and regular and the regional lymph glands are not involved. The striking feature is its stony hardness, in which it excels even a scirrhus carcinoma. In spite of extensive destruction of gland tissue, signs of hypothyroidism are not seen.

**Treatment** is directed solely to relief of pressure upon the trachea and œsophagus (Joll). An operation directed to this end is fraught with both difficulty and danger, but it is the only procedure we have to offer. Radiotherapy can do no good.



**Lymphadenoid Goitre.**—Although many authorities regard this as an early stage of Reidel's disease, there are strong grounds for the belief that it is a clinical entity. It consists in a diffuse infiltration of the gland with lymphocytes in women over 45 years of age. The gland becomes uniformly enlarged and, although fibrosis occurs later, there is never the same degree of hardness as in ligneous thyroiditis. Most of these patients eventually show signs of myxœdema.

*Treatment.*—If a correct diagnosis is made, surgical treatment is contraindicated. When myxœdema threatens, appropriate thyroid medication will be required.

### PHYSIOLOGICAL HYPERPLASIA OF PUBERTY

A slight increase in size of the thyroid occurs temporarily at puberty, each menstrual period and during pregnancy. A more obvious and persistent enlargement is commonly found in girls about the age of puberty. It is noticed first at the age of about 14 years and continues for periods varying from eighteen months to three years. A similar condition occurs rarely in boys. The swelling is regular and homogeneous and consists in a true hyperplasia of normal thyroid tissue. It is due to an attempt by the thyroid to make good a deficiency in secretion of one or more members of the endocrine group, which fail to respond to the increased demand that puberty makes upon them. The swelling is symptomless and slowly disappears, leaving no trace of thyroid disease; rarely, however, subsidence of the hyperplasia unmasks an adenoma. No treatment is needed, the only important thing being to reassure the parents that the condition is temporary.

### SIMPLE GOITRE

Joll classifies simple goitre thus :—

- |                      |                            |
|----------------------|----------------------------|
| 1. Parenchymatous    | } Diffuse or symmetrical.  |
| 2. Colloid           |                            |
| 3. Nodular . . . . . | { (a) Diffuse or multiple. |
|                      | { (b) Localised or single. |

**Etiology.**—Simple goitre is either endemic or sporadic. Nothing is known of the cause of sporadic cases, which occur under conditions quite antagonistic to the development of endemic goitre. The etiology of the latter is imperfectly understood and we are ignorant of any specific factors leading to the variation in type. It is instructive to consider some of the facts and theories which command serious attention.

1. **DISTRIBUTION.**—Endemic goitre occurs in many parts of the world. The "goitrous" areas in Great Britain are the south-west counties of England, Hereford, Warwickshire, Gloucestershire, Derbyshire, Cheshire and Nottingham, parts of Wales, and a small area of Scotland; in Europe, the whole Alpine range from Savoy to Austria, the plains of Northern Italy and the Pyrenees; in America, the mountain districts of the Pacific seaboard and the plains around the Great Lakes; in the Himalayas, in Egypt and in New Zealand.



2. **IODINE DEFICIENCY.**—The thyroid gland is associated with iodine metabolism and in many districts the prevalence of goitre corresponds to an iodine deficiency in soil, water and food. The successful use of iodine as a prophylactic agent in the prevention of goitre is a strong argument in favour of this theory. Excellent results have followed the experiment in America of adding a trace of iodine twice monthly to the communal water supply, and also in Switzerland where widespread propaganda urges the inhabitants of the mountain valleys to use iodised salt, tea and chocolate.

3. **WATER CONTAMINATION.**—MacCarrison has proved conclusively that goitre can be produced by giving animals or humans water from known "goitrous springs." He has shown that if such water is filtered, no ill-effects are experienced, but that a solution of the scrapings from the filter candles does produce goitre. Further he quotes a military school in the Punjab in which the incidence of goitre was between 60 and 80 per cent. until in 1918 a new water supply was installed, after which the rate dropped to 2.2 per cent. within three years. Instructive as this work is, it does not prove that water pollution is an active etiological factor in goitre production throughout the world.

4. **INFECTION.**—There is no evidence to uphold theories that a specific living organism is responsible.

5. **HEREDITY** undoubtedly plays a part. Many goitrous districts are composed of isolated communities in whom intermarriage is unavoidably prevalent. Endemic goitre has a high incidence in children of goitrous parents, and if this inbreeding is continued from one generation to another, goitre becomes an established characteristic in the children.

6. **OTHER CONTRIBUTORY FACTORS** are a diet having an excess of protein and calcium, unhygienic condition of living and possibly lack of sunlight. Finally MacCarrison suggests that goitre may be due not only to a deficiency of iodine and other substances, but also to an inability of the tissues to utilise them when present in normal amounts.

It cannot be said that any one theory so far satisfies critical analysis.

### PARENCHYMATOUS GOITRE

*Etiology.*—Joll defines this type of goitre as one "due to an increase in the epithelial elements without any appreciable colloid accumulation." It is found in areas of high endemicity, *e.g.*, the mountain valleys of Switzerland, where iodine deficiency and water pollution are prevalent. It is uncommon in this country. It occurs in children and adolescents of both sexes and may be present as a congenital lesion. It is not usually seen after twenty years because by that time it is likely to have become a colloid or nodular goitre.

*Pathology.*—The change affects the whole thyroid, though one lobe may be larger than the other. The gland is enlarged to moderate size and is firm and vascular; its surface is but slightly lobulated. Essentially the pathological process is an overgrowth of epithelial elements,

the vesicles being small and irregular. The colloid and iodine content of the gland is reduced.



FIG. 179

An elderly woman with a huge colloid goitre.

*Symptoms.*—At first there is a symptomless swelling readily identified as affecting the whole thyroid gland. It has a smooth lobulated surface, is solid, elastic and homogeneous. Later compression of the trachea causes dyspnoea and stridor and in the later stages signs of dysphagia and myxoedema may occur.

*Treatment* is needed only to relieve pressure and for cosmetic reasons. A bilateral partial thyroidectomy should be performed.

#### COLLOID GOITRE

*Etiology.* — Diffuse colloid goitre occurs in areas of low endemicity, *e.g.*, England and Wales and the region of the Great Lakes of America. It is seen in both sexes commonly between puberty and 30 years, though in some parts it may occur at an earlier age.

*Pathology.*—The whole gland is affected, being enlarged often to a considerable size (Fig. 179). Its surface shows marked lobulation. On section it shows a honey-combed appearance (Fig. 180) and sticky colloid oozes from it. Microscopically there are seen greatly distended vesicles lined by flattened cells and full of colloid. The iodine content is much in excess of normal, though actually it is less in proportion to the size of the goitre.

Many transitional types between this and nodular goitre will be seen, the cut surface showing increasing degrees of lobulation, until the gland seems to be filled with numerous encapsuled swellings.

*Symptoms.*—Retrosternal prolongations are common in this



FIG. 180

Colloid goitre showing general appearance and cross-section.

type of goitre, which also tends to spread behind the trachea and encircle it. Compression of the trachea with dyspnoea is therefore earlier in its appearance than in the parenchymatous form. Later many of these patients exhibit signs of a moderate degree of thyrotoxicosis.

*Treatment* is directed to relief of pressure, improvement of the patient's appearance and prevention of thyrotoxicosis.

### NODULAR GOITRE

**Generalised Type.**—Although histologically incorrect the term "multiple adenomatous goitre" graphically described the appearance of this, the commonest of all endemic goitres. It occurs in all areas in patients of both sexes from the age of 30 onwards, whilst in long-established goitrous localities it may be seen in young subjects.

*Pathology.*—This goitre often grows to great size and as its name implies has a markedly lobulated surface with large veins coursing over it. Unlike other varieties it is frequently asymmetrical, although changes are present throughout the gland. Its cut surface presents a picture of multiple "adenomata" surrounded by fibrous septa. These masses may be more or less uniform in size, but more probably vary greatly. The adenomata are pale pink in colour and show the semi-translucent appearance due to colloid. Degenerative changes will be seen in older lesions, hæmorrhage and cyst formation being common. Microscopical appearances vary widely from greatly distended vesicles with flattened epithelium to solid colloid-free adenomatous structures. Some of the vesicles exhibit epithelial hyperplasia to such an extent that the picture may closely resemble that found in primary thyrotoxicosis.

The clinical picture and treatment are similar to those of colloid goitre.

**Localised Type.**—Great controversy still ranges around the exact pathological status of the single localised encapsuled swelling appearing in an otherwise normal thyroid gland. Joll includes them amongst the nodular goitres. Such tumours do occur without any change, naked-eye or microscopic, in the rest of the gland. For this reason I prefer to classify them as true neoplasms, and they will be described later (p. 375).

### THYROTOXICOSIS

#### PRIMARY THYROTOXICOSIS

Exophthalmic goitre or Graves's disease is a condition in which thyrotoxic symptoms are due to changes affecting a previously normal gland. It is believed that this disease does not originate in the thyroid gland, but typical changes are produced in it which lead to a characteristic clinical picture. It is generally held that the toxic substance circulating in the blood is a perverted thyroid secretion, but Joll believes that in addition there must be also an element of hyperthyroidism, i.e., an excess of thyroxine in circulation. We remain in ignorance of

the basic causes of this disease, but certain facts and theories repay consideration.

*Etiology.*—1. Generally speaking, this disease is uncommon in most endemic goitre districts, but this is not true of this country. Here the incidence of the two conditions is high in goitrous districts, although Graves's disease is distributed widely throughout the whole country.

2. Women are more frequently affected than men in the ratio of 10:1 but after the age of 50 this falls to 6:1. Nulliparous women are more susceptible than their parous sisters.

3. The highest age incidence is between 25 and 45 years; a number of cases occur in girls between puberty and 24 years; after 45 years there is a sharp fall.

4. Causation. *A.* Heredity as far as we know plays no part in this disease. *B.* As some of the symptoms can be produced by stimulation of the sympathetic nervous system, it has been suggested that an imbalance between the two sides of this system may be the underlying cause, but the theory is untenable. *C.* Other workers seek to incriminate both adrenal and thymus glands, but in neither case can the argument be upheld. The thymus is considerably enlarged in about 21 per cent. of cases of Graves's disease, but the exact nature of this association is unknown (Fig. 181). *D.* Focal sepsis especially in the fauces and pharynx and acute infectious fevers such as influenza undoubtedly lead to exophthalmic goitre. The former is exemplified by a patient suffering from acute streptococcal tonsillitis who developed a severe attack of thyrotoxicosis with a high B.M.R. and who rapidly recovered after tonsillectomy. *E.* Psychological factors, anxiety, worry, sexual maladjustments and emotional disturbances



FIG. 181

A specimen showing the appearance of the thyroid in exophthalmic goitre with an enlarged persistent thymus

ances appear in the histories of so many patients that this association cannot be merely coincidental. Joll regards this aspect as being overrated and observes that most patients will give a story of psychic trauma. I feel that the evidence, though circumstantial, is too strong to ignore. In some patients at least it is probably the strongest etiological factor.

*Pathology.*—Iodine medication and radiotherapy produce marked changes in the thyroid gland in Graves's disease and the following description is of the untreated gland.

1. Naked-eye appearance. The degree of thyroid enlargement varies greatly, but it is never so pronounced as in simple goitre; indeed

it may be quite trivial. It is necessary to emphasise, however, that there is always some enlargement. It is paler, smoother, more solid and compact than normal, and there is a marked increase in vascularity out of all proportion to its size. On section the surface is uniform in colour and consistence, being pale pink, of solid appearance and showing no suggestion of colloid (Fig. 181).

2. Microscopically, the acini are irregular and the epithelium undergoes marked hyperplasia. The cells are several layers deep and many are columnar in type. The vesicles become full of cells and little or no colloid can be seen, while in some sections the acini are completely solid.

It is right to add that the histology of primary thyrotoxicosis varies considerably, and the above description, though typical, does not cover the whole ground. In some cases little or no epithelial hyperplasia can be found and colloid appears to be normal in amount.

Iodine medication and X-rays both produce greatly increased fibrosis and the former leads to a definite increase of colloid in the gland.

*Symptoms.*—Graves's disease is rather more chronic than is frequently taught. Dunhill has pointed out how large a percentage of his patients come to him with a history of five to ten years. Untreated it tends to pass through a cycle of changes of exacerbation and improvement, and at one time an attempt was made by Plummer to utilise the periodicity as an indication of prognosis and of the most favourable time for operation. The behaviour of these patients is too capricious for any such classification. A few cases are of a rapid fulminating type and die within a few weeks. On the other hand, very few patients recover spontaneously.

Symptoms may be grouped as :—

A. Pressure.

C. Ophthalmic.

B. Toxic { Nervous.  
Cardiac.  
Alimentary.

D. General { Loss of weight.  
Menstrual disorders.  
Metabolic changes.

A. PRESSURE SYMPTOMS are unusual because the thyroid rarely enlarges to any size. In some cases there may be dyspnoea (Fig. 182).

B. TOXIC SYMPTOMS.—1. Nervous. These patients are in a highly nervous, excitable state, reduced to a fever of apprehension by the approach of a stranger or of a doctor to examine them. In the early stages they find that work previously easy cannot be faced, and small difficulties cheerfully surmounted before now become impossible, the effort leaving them exhausted with a mental fear of impending disaster. The slightest strain upsets them and they are subject to waves of cutaneous vasodilatation, in which a red flush spreads from the face



FIG. 182

An unusually large swelling in exophthalmic goitre causing dyspnoea.

to neck and chest. Later the degree of nervous instability increases and they suffer from alternate fits of great excitability and depression. In the more severe cases either acute mania or melancholia precedes a fatal issue.

2. **Cardiac.** One of the earliest symptoms is a feeling of palpitation with a rapid pulse-rate due to toxic myocardial degeneration. As time goes on the heart dilates and auricular fibrillation occurs. Many patients are diagnosed as having valvular disease or paroxysmal tachycardia without any thought being given to an underlying thyrotoxicosis. An important lesson emerges, viz., in every case of disordered action of the heart which presents no previous history of illness likely to affect the heart and having no clearly defined cause, the possibility of thyrotoxicosis must always be considered.

3. Vomiting and diarrhoea are usually seen only in the later stages or in fulminating cases. Glycosuria is also a late symptom. They should be regarded as indications that treatment is urgently needed if the patient is to be saved.

**C. OPHTHALMIC.**—Protrusion of the eyes from which the disease takes one of its names is an early and characteristic feature (Fig. 183). It is frequently the reason for which a patient seeks advice.

**D. GENERAL.**—(1) Loss of weight is universal and its arrest and subsequent gain is the most important indication of response to treatment. (2) Women usually

suffer from a disturbance of menstruation, varying from complete amenorrhœa to irregular periods. (3) The B.M.R. is always raised and affords a guide to the degree of toxicity. Its importance must not be exaggerated and should be considered only in association with weight and response to medical treatment in assessing prognosis and the need for surgical intervention.

**Signs.**—**A. THYROID ENLARGEMENT.**—The slight or moderate enlargement affects the whole gland but not necessarily equally. It is smooth, firm and regular. In the later stages or after iodine medication the gland becomes much harder.

**B. MUSCULAR TREMOR** is constantly found. It is demonstrated by asking patients to hold out their hands with fingers separated.



FIG. 183

A very typical appearance in exophthalmic goitre, the long thin neck, the moderate enlargement of the gland and the staring eyes.

*C. TACHYCARDIA* is present in every patient even in the early stages. When taken in conjunction with loss or gain in weight, it forms a most valuable gauge of the degree of toxicity. Owing to its susceptibility to emotional influences, so prominent a feature of this disease, the pulse-rate during sleep is more reliable than at other times. It may rise to 180 beats per minute in severe types, 120 to 140 in cases of moderate severity and 100 in mild forms. During auricular fibrillation much irregularity in the pulse may be expected.

*D. EXOPHTHALMOS* is by no means constant. It may be absent throughout, occur either early or late, be slight or marked and affords no guide as to the severity of the toxicosis. Its cause is unknown; one theory postulates that it is due to overaction of Müller's unstriated muscle at the back of the eyeball. Doubt is thrown upon the very existence of this muscle; if present, it could not push the eye forward. Overaction of the sympathetic is suggested as an explanation, but resection of the cervical sympathetic produces little or no improvement in the ptosis.

Exophthalmos is important for two reasons. First, when severe it may threaten the integrity of the eye because of corneal ulceration and panophthalmitis. Secondly, it may be permanent if operation upon the thyroid is delayed too long.

*E. OCULAR SIGNS.*—1. Von Graefe's sign is demonstrated by instructing the patient to keep the head still and look up. She is then asked to look down to the floor, when the upper lid is seen to lag behind the eyeball and come down in a series of little jerks instead of moving smoothly and simultaneously with the globe.

2. Moebius' sign is the inability of the eyes to converge on near accommodation.

3. Stellwag's sign consists in diminished blinking with imperfect closure of the lids.

4. Dalrymple's sign is a rather fixed stare due to retraction of the upper lid, even when exophthalmos is absent.

5. Joffroy's sign is the absence of wrinkling of the forehead when the patient looks up. This is not constant.

*Treatment* falls into three groups.

*A. MEDICAL.*—*Rest* in bed is an essential preliminary to other forms of treatment. It leads to a fall in pulse-rate and B.M.R. and to an increase in weight. The time thus spent depends entirely upon the progress of each individual.

*Iodine Medication.*—Iodine is administered in the form of Lugol's mixture, which is a 5 per cent. solution of iodine in a 10 per cent. aqueous dilution of potassium iodide. It is given in 5-minim doses three times a day and the dose increased daily up to 10 minims thrice daily. Its effect is rapid and striking, the pulse-rate falls, the general condition improves and the B.M.R. drops. Its maximum effect is reached between the tenth and twenty-first day. Its continued use beyond the fourth week leads to an increase in the size of the vesicles, which become packed tight with colloid, and the gland is much harder as a result. Such misuse may produce a return of symptoms more severe than before. Iodine is therefore IN NO SENSE CURATIVE and there



is an ideal time for its use. A second course at a later date is not usually so effective.

Other drugs have little value except for the control of auricular fibrillation (see below).

**B. RADIOLOGICAL.**—Improvement follows X-ray therapy in many cases, but it is not curative except in a few patients and relapses are to be expected. It has definite indications as will be seen later.

**C. SURGICAL.**—Subtotal thyroidectomy, in which seven-eighths of the gland is removed, is the method of choice.

In preparation the patient is kept in bed on a light diet with plenty of fluids. In moderate cases the operation is fixed for the twelfth day following the beginning of iodine treatment, which is continued for ten days after operation. In the severe cases the iodine is withheld until such time as the patient has improved to such a point at which it can confidently be expected to achieve its maximum effect and bring the patient within the limits of operative safety. Anæsthesia may be avertin and gas-oxygen-ether, local infiltration of the skin together with cervical plexus block or by a simple straightforward inhalation method. Technique is directed towards adequate access, gentleness of handling and removal of seven-eighths of the gland, the parts left being the posterior area of the capsule and a thin slice of each lateral lobe. This ensures an adequate residue of thyroid tissue, preserves the parathyroids and prevents injury to either recurrent laryngeal nerve. Great care is taken in accurate suturing of both platysma and skin, the wound being drained for twenty-four hours.

*Application of Alternative Methods.*—Every patient must be judged upon the individual clinical picture. In few other diseases is the decision of when to operate and the pre-operative preparation so important. In every case a routine examination will search for focal sepsis or intercurrent disease and appropriate therapy be directed to each. It is advantageous to discuss treatment by dividing patients into three groups.

1. Mild group. These people have mild symptoms, a pulse-rate not above 100, a B.M.R. not exceeding +25 and no great loss of weight. They should be put to bed and any underlying psychological cause sought for and treated. X-ray therapy is of value but iodine must not be given. If marked improvement is not evident within six to eight weeks, operation should be advised.

2. Moderate group, into which the majority fall, is the ideal surgical one and no time should be lost in preparing for operation.

3. Severe group. Patients are too ill for operation and every effort must be made to prepare them for it as quickly as possible. Auricular fibrillation is an indication for, rather than against, surgery. In these cases Joll advises that 1 drachm of tincture of digitalis should be given daily until compensation is restored; he substitutes strophanthin intramuscularly if digitalis causes vomiting. Other workers prefer quinidine. Some severe cases fail to respond to weeks of medical treatment. They should be dealt with by a series of arterial ligatures, first one and then the other superior thyroid artery being tied under local anæsthesia in bed, and rarely one or both inferior vessels



may need to be tied also. Full doses of Lugol's iodine will be started as soon as improvement warrants operation.

Results have improved remarkably since 1918, before which time the mortality was high. Dunhill and Joll have shown how few cases need be despaired of, but they would be the last to deny how grave is the anxiety associated with the care of severely ill thyrotoxic patients. Thanks to their surgical work and teaching and to the introduction of iodine medication prognosis is now favourable in almost all cases. The result of successful treatment is the return to normal health, the last symptom to disappear frequently being exophthalmos, which may be present for several years, or indeed for ever.

### SECONDARY THYROTOXICOSIS

Dunhill and others regard thyrotoxicosis as one disease with a wide range of variations in severity and manifestations. Joll, though in general agreement, prefers to retain the classification of "Primary" and "Secondary" thyrotoxicosis.

The name by which the secondary type is known usually in this country is Plummer's "Toxic Adenoma," based upon the observation that such patients frequently had one large adenoma; in fact almost every case is suffering from the multiple adenomatosis of a nodular goitre.

Secondary thyrotoxicosis is best defined as a condition in which thyrotoxic symptoms are engrafted upon a long-standing goitre. It usually follows the nodular type of simple goitre and one variety results from excess medication with iodine.

The clinical picture is in many ways similar to that of the primary variety. Most of the symptoms and signs are milder in the secondary type; but myocardial changes may be much more marked.

Treatment consists in a partial thyroidectomy and in some cases only one lobe needs attention.

## NEW GROWTHS

### ADENOMA

It has been shown that the existence of a true adenoma is denied by many observers, who regard it merely as an example of the localised variety of nodular goitre. Such isolated adenomata do occur, however, in an otherwise normal gland and it seems right to classify them among the new growths.

It occurs at any age after puberty, being most common between 25 and 45 years. It forms a localised, round, smooth encapsuled swelling. Two pathological types are described, foetal and cystic.

The foetal adenoma is solid and more compact and firm than normal thyroid tissue, consisting of tubular acini with no lumen and no colloid. The cystic adenoma is soft and the acini irregular in shape and size, some fusing with their neighbours to form minute cysts, which by a process of coalescence produce large cysts visible to the eye. Their cut surface shows pale solid areas with cysts containing glairy fluid

discoloured by altered blood (Fig. 184). If untreated, adenomata may undergo certain modifications; they may become (1) the site of an intracystic hæmorrhage, (2) infected, (3) calcified or (4) malignant.

*Clinically* they produce a localised swelling in the neck either in the isthmus or lateral lobes. Those arising in the lower pole may grow down into the superior mediastinum, in which case the swelling above the clavicle may be somewhat diffuse. Pressure symptoms are absent in most cases. A single adenoma in the neck displaces the trachea but does not cause dyspnoea: bilateral growths, however, may compress the trachea between them and eventually cause difficulty in breathing. When they are situated in the superior mediastinum there is no room for tracheal displacement and dyspnoea is an early symptom. Sudden hæmorrhage into a cyst will usually produce urgent and alarming shortness of breath. Rarely a calcified adenoma may press on a recurrent laryngeal nerve, and cause hoarseness of the voice. In the majority of patients the adenoma attracts attention only by the disfigurement of the neck.

*Treatment* is removal. Small adenomata can be enucleated, whilst others are more satisfactorily dealt with by resection-enucleation in which a small area of gland tissue is removed with the tumour.

So-called toxic adenoma is referred to under secondary thyrotoxicosis.



FIG. 184

An adenoma of the upper pole of the thyroid gland.

## CARCINOMA

This may arise spontaneously in a normal thyroid gland, in pre-existing parenchymatous and colloid goitres, or in an adenoma. It is either the polygonal-celled carcinoma simplex type or an adenocarcinoma with acini containing pseudo-colloid. These growths are among the rarer types of carcinoma, are more common in men and occur late in life. They spread throughout the gland tissue, infiltrate the capsule and become fixed to surrounding structures, of which the recurrent laryngeal nerves are early affected. In metastasising, which they do extensively and quickly, a marked predilection is shown for bone, in which pulsating tumours are produced. There is a particular type of thyroid growth, the thyro-adenoma malignum, in which the primary nodule cannot be detected and yet widespread bony metastases occur, in which the tissue appears microscopically to be, and can function as, normal thyroid. A specialised, but very rare, type of carcinoma occurs in young males during adolescence. It does not appear to be highly malignant, but local recurrence will occur unless the whole gland is removed.

If the carcinoma starts in a pre-existing goitre, there is a sudden increase in its size. On the other hand, there may be enlargement of

a previously normal gland. The swelling is very hard, irregular and nodular, and rigidly fixed to surrounding structures. Hoarseness comes on when the recurrent nerves are affected, and stridor and dyspnoea result from tracheal compression. Later the growth ulcerates through the trachea and a profuse blood-stained sputum appears. As the growth spreads along the trachea, respiratory obstruction becomes most distressing, and is marked by violent attacks of coughing. Pain is a variable symptom. Death occurs from suffocation, broncho-pneumonia or secondary hæmorrhage.

*Treatment* by radical thyroidectomy is rarely possible owing to the extent of the growth when first seen. Threatened suffocation may demand a tracheotomy, but this inevitably means the beginning of the end from broncho-pneumonia. Many conflicting opinions are heard concerning radiotherapy. X-rays appear to be useless, but some improvement can be expected from the implantation of radium or radon seeds.

R. M. HANDFIELD-JONES.

## CHAPTER XX

### THE EAR

**SURGICAL ANATOMY.**—The auricle has a framework of cartilage covered by a thin layer of skin. In suturing wounds of the auricle, the stitches should not be passed through the cartilage but should include only the skin, so that a line of sutures on the front and back may be necessary. The lobule containing no cartilage is more pliable.

The external auditory meatus, partly cartilaginous and partly osseous, is lined with skin and closed at the inner end by the tympanic membrane.



FIG. 185

A cross-section of the outer and middle ear showing the tympanic membrane and the ossicles.

The outer cartilaginous meatus is about a centimetre in length. The cartilage is defective above and also interrupted by the transverse fissures of Santorini which are filled by fibrous tissue and blood vessels. The osseous meatus, about twice as long, terminates in a ridge which has a groove on its free border for the attachment of the tympanic membrane (Fig. 185). In the upper part there is a hiatus in the ridge, the notch of Rivini, the edge of which gives attachment to Shrapnell's membrane. The meatus is directed inwards and slightly forwards, and in the outer half upwards, so that to straighten the meatus for examination or syringing the auricle should be drawn backwards and upwards. In early life the annulus tympanicus is near the surface and there is no osseous meatus, which lengthens the meatus as it develops.

Consequently in small children the tympanic membrane is very near the opening of the external auditory meatus, and the auricle need only be drawn backwards for examination of the membrane. The skin of the cartilaginous meatus contains sebaceous and ceruminous glands, which latter are modified sweat glands.

The tympanic membrane lies at an angle of  $55^{\circ}$  with the horizontal plane. Its outermost part is above and behind and its innermost below and in front. It is composed of three layers: an outer epithelial layer continuous with the skin, an inner one continuous with the mucous lining of the tympanum and a middle fibrous layer composed of circular and radiating fibres. In this way the *membrana tensa* is formed. The fibrous layer is absent in the portion which occupies the notch of Rivini and thus the *membrana flaccida*, or Shrapnell's membrane, is formed. The long process

or manubrium of the malleus lies in the tympanic membrane and passes upwards and forwards. Its tip is fixed to the membrane at the umbo, and above its short process the malleus is not so attached. The important landmarks on inspection with mirror and speculum are the manubrium, with the cone of light spreading downwards and forwards from its tip at the umbo to the margin, the short process with a fold stretching backwards and forwards to either extremity of the notch of Rivini and Shrapnell's membrane above these folds. If the membrane is thin the long process of the incus may be visible behind the handle of the malleus.

The outer wall of the tympanum consists mainly of the tympanic membrane (Fig. 185), but above that is an overhanging portion of the squamous portion of the temporal bone. In this way the attic, which contains the head of the malleus and the body of the incus, is formed. It is to a great extent shut off from the lower part of the tympanum, and lies above the level of a line joining the two tympanic spines which bound the notch of Rivini. The attic communicates posteriorly with the mastoid antrum by the *aditus ad antrum*. The horizontal semicircular canal forms a slight prominence on the inner and lower walls of the aditus. The facial nerve, after passing backwards in the aqueductus Fallopii on the inner wall of the middle ear above the oval window, turns downwards and outwards to the stylomastoid foramen. It is therefore below and in front of the mastoid antrum. In the infant the mastoid process has not developed, so that the antrum lies higher, above rather than behind the middle ear, and the facial nerve escapes from the stylomastoid foramen beneath the skin, unprotected by the mastoid process. The nerve may then be divided by an incision incautiously carried down to the bone.

The external aspect of the mastoid gives no information in regard to its internal structure, the depth of the antrum, the position of the sigmoid sinus or the level of the middle fossa. On exposing the mastoid process, however, its borders should be defined and, in addition, the posterior edge of the osseous meatus, the supramastoid crest or *linea temporalis*, which is the continuation backwards of the upper border of the zygoma, the spine of Henle and the suprameatal triangle. The suprameatal triangle is formed by the posterior root of the zygoma above, the upper and posterior segment of the osseous meatus below, and an imaginary perpendicular line uniting these two extending from the most posterior point on the external osseous meatus to the zygomatic root. The surface of the triangle is marked by numerous foramina for blood vessels and in the infant these markings are very conspicuous. The spot thus indicated is a fairly constant guide to the level of the antrum.

The internal structure of the mastoid process is very variable. In about 40 per cent. of skulls the air cells are fully developed, and in 20 per cent. the bone is diploetic, while in the remainder either the tip and posterior part are diploetic and the rest pneumatic, or the bone is sclerotic. The ivory hard sclerotic bone is commonly associated with chronic suppuration in the middle ear and antrum, and was thought to result from that process, but it may occur independently of any obvious pathological change. It is probable that it is a factor which tends to make an attack of otitis media chronic, and its occurrence has been explained by inflammatory changes which occur very early during life and prevent the pneumatic cells from being developed. When fully developed the pneumatic cells are found in the following groups :—

1. Zygomatic. The cells of Kirschner in the posterior root of the zygoma.

2. Retrofacial. Cells on the posterior meatal wall running along the aqueduct. They may extend behind the horizontal canal into the petrous beneath the labyrinth and also beneath the middle ear as peribulbar cells in close relation to the jugular bulb.
3. Apical cells. There is often one large cell near the tip of the mastoid process.
4. Marginal cells. These are in the posterior part of the mastoid process lying around and behind the lateral sinus, from which they are separated by a plate of dense bone. The mastoid emissary vein runs through them and they extend upwards into a group of cells lying between the lateral sinus and the dura of the middle fossa.

All these groups of cells must be followed out in the operation for acute suppuration in the mastoid process (p. 396).

The extent to which the lateral sinus cuts into the inner surface of the mastoid process and approaches the posterior wall of the osseous meatus is also very variable. It may lie as much as an inch behind the meatus, while at other times it lies so close behind that the exposure of the antrum is extremely difficult, and this has to be approached by working backwards from the attic. The middle fossa may be low and overhang the outer side of the antrum, and this anatomical formation is commonly associated with a forward-lying sinus, thus limiting the field of operation still further.

The deep cervical fascia is attached to the mastoid process, so that if pus perforates the inner plate of the mastoid process it may track deeply along the posterior belly of the digastric or down the neck (Bezold's mastoiditis).

*Examination of the Ear.*—The symptoms of which complaint may be made are deafness, tinnitus, pain, vertigo and discharge, but before any further investigation of these is made, the ear should be inspected and wax, pus or debris removed, for until this is done any other examination is futile. The fluid for syringing should be comfortably warm and the patient must always be sitting, lest vertigo be induced. The meatus is dried with cotton-wool held in angular forceps and then inspected. Unusual and misleading appearances are frequently due to failure to cleanse the meatus before examining with mirror and speculum. Several sizes of speculum should be available. The light is reflected on the auricle and any external abnormality noted. The speculum held by the forefinger and thumb of the left hand is warmed and inserted into the meatus, which is straightened by drawing the auricle upwards and backwards with middle and ring fingers. The condition of the meatus, the colour of the membrane, the presence of the light reflex, the inclination of the malleus, the prominence of its short process and of the anterior and posterior folds, the presence of perforations, patches of calcification or of pulsation in any part are points to be noted. Swelling or exostoses of the meatus may prevent a satisfactory view from being obtained. A magnified image is obtained by using Siegle's speculum which enables the air pressure in the meatus to be varied. Thus mobility of the membrane and ossicles may be tested and the position of an otherwise invisible perforation be detected (Fig. 186).

If compression of air in the Siegle's speculum produces nystagmus in the presence of a perforation, a fistula of the external canal, transmitting increased pressure to the labyrinth, may be present, but cannot be diagnosed with absolute certainty. To test the hearing or cochlear portion of the labyrinth the voice is the most useful standard, as it is the most important

sound which has to be appreciated in ordinary life. The distances at which whispered and conversational voices can be heard are recorded. The examiner

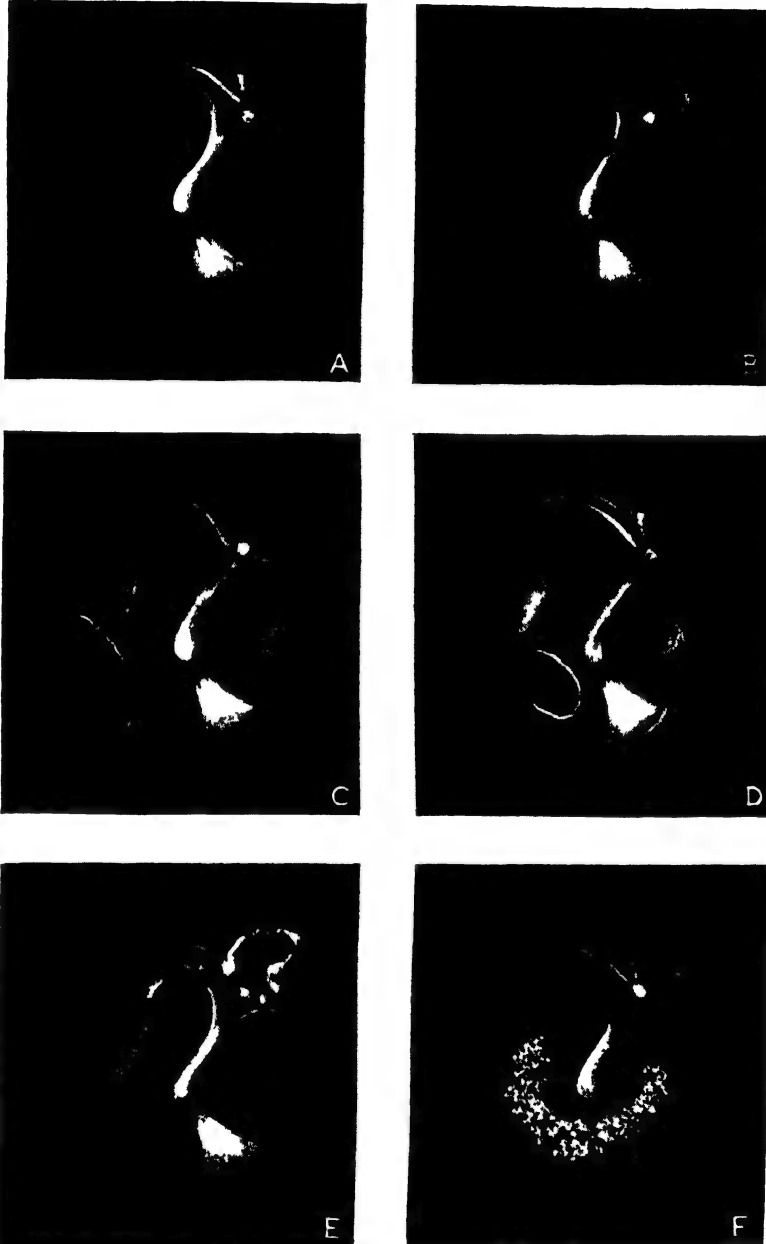


FIG. 186

The various appearances of the tympanic membrane as seen through an aural speculum. A, normal; B and E, attic perforations; C, a marginal and D, a central perforation; and F, calcareous thickening of the membrane.

must be careful to maintain the loudness of his speech for testing at a constant level. The watch is an unsatisfactory instrument for testing as the tick is high-pitched and only tests the high tones, which are not of practical importance. Hearing for these tones may be much reduced or lost without

causing much difficulty in hearing the spoken voice. For scientific record of hearing an audiometer must be used. This tests the hearing over a wide range of tones, and the hearing capacity for the different parts of the scale can be recorded as a graph. This can be done with less precision but sufficiently accurately for practical diagnosis with a set of tuning forks of different pitch. Generally, it is found that in disease of the middle ear or obstructive deafness the lower part of the scale is affected, whilst in disease of the internal ear or auditory nerve, or perceptive deafness, the higher notes are lost. A further method of determining the affected portion of the auditory apparatus is by testing bone conduction. The base of a fork is applied to the mastoid process and, if the time of hearing is shortened as compared with the capacity of the examiner, the deafness is in the cochlea or the auditory nerve. If the time of hearing is prolonged, then the deafness is of the obstructive type in the middle ear. This is Schwabach's test. Absolute bone conduction can be determined by performing this test with the meatus closed, so that obstructive deafness is produced. No pathological condition produces an increase of bone conduction beyond the absolute bone conduction thus determined, although it may appear to be relatively increased with the meatus open. In Weber's test the fork is applied in the middle line of the forehead, and will be heard better in the deaf ear in middle ear deafness, and better in the good ear in nerve deafness.

Rinne's test depends upon a comparison between the capacity for hearing a fork by air and bone. If the air conduction exceeds the bony, as in nerve deafness, then Rinne's test is positive. If the bone conduction exceeds the air conduction, as it may do in middle ear deafness, then the test is negative. Normally, air conduction exceeds bone conduction and the test is positive.

The condition of the Eustachian tube, which may be obstructed or contain mucus, is investigated by inflation. The mouth is shut, the nostrils closed by the thumb and first finger and a forcible attempt made at expiration (Valsalva's experiment). Politzer's method is better. The patient takes a sip of water and holds it in the mouth. The nozzle of a Politzer's bag is inserted into one nostril and then both are closed so that no air can escape. The patient is told to swallow, and at the moment of swallowing the bag is compressed and the air is driven up into the tympanum. The mouth must be kept tightly closed during the act of swallowing. It is generally believed that the orifice of the Eustachian tube is opened during swallowing by contractions of the tensor palati.

A more certain way of inflating the tympanum is by the Eustachian catheter. This has a curved beak to fit into the orifice of the Eustachian tube and near the proximal end is a ring, whose plane coincides with that of the beak, and so indicates its position when concealed. Before passing the catheter through the nose, some 5 per cent. cocaine may be applied, if there is a sensitive spur low down on the nasal septum, but the application is not necessary as a routine. The catheter is introduced along the floor of the nose until it reaches the posterior pharyngeal wall. The point is turned outwards into the fossa of Rosenmüller, and as it is gently withdrawn it slips over the posterior lip into the mouth of the tube. It is generally possible with practice to turn the tip of the catheter into the tube as soon as the soft palate has been passed. A bag is then attached to the catheter and air blown gently into the Eustachian tube. An auscultation tube connecting the ear of the patient and the surgeon will indicate by the sound, which normally is of a dry blowing character, whether air is passing into the tympanum. Bubbling sounds indicate catarrh, and if there is a perforation of the tympanic membrane, a whistling sound is heard close to the ear of the observer. The hearing should be tested before and after inflation.



The functional activity of the vestibular portion of the labyrinth can be tested by caloric and rotation tests. By irrigating the ear with hot or cold water, convection currents are set up in the intact membranous labyrinth and nystagmus is produced. Rotation of the patient with the head in varying positions will produce nystagmus of varying character and duration. By the caloric method each ear is tested separately. Rotation affects both ears simultaneously, but in opposite directions. Just as compensation makes the investigation of defective cerebral function difficult, so it introduces great difficulty into the interpretation of these tests, however carefully performed.

Bacteriological examination of discharge from the ear frequently gives important information. In acute cases the hæmolytic streptococcus, *S. viridans*, *Pneumococcus*, *M. catarrhalis*, *B. influenzae*, and less commonly, *S. aureus* may be found in discharge from the middle ear. In chronic discharges, which are usually fœtid from the decomposition of epithelium by the *B. butyricus*, there are a number of saprophytic organisms, and, in addition to the ordinary pyogenic organisms, *B. tuberculosis*, the Klebs-Loeffler bacillus and *B. pyocyaneus*, all pathogenic, may be found. It is difficult to eradicate the infection of a diphtheria carrier from the ear, and there is good reason also to believe that the infection of scarlet fever may be carried in a chronic aural discharge. The acute otitis media which frequently occurs during an attack of diphtheria, scarlet fever or typhoid fever is usually streptococcal. Again, the septicæmia, which results from septic lateral sinus thrombosis caused by a chronic otorrhœa, is always streptococcal, although the aural discharge contains a mixed infection of various organisms.

In discharge coming from an eczematous lesion of the external auditory meatus the *Pityrosporon* of *Malassez* may be found, and with it a secondary infection of *staphylococcus albus*. In this manner the field is prepared for further infection of the skin by the *S. aureus* or the streptococcus, producing respectively either boils or fissures at the orifice of the meatus. A membrane, resembling damp blotting paper covered with black or yellow spots, associated with itching and discharge from the external ear, should cause otomycosis to be suspected. This is caused by the *Aspergillus niger*, *A. fumigatus* or *A. flavus*. If aspergillosis is suspected a piece of the membrane should be teased in dilute liquor potassæ and examined with the microscope. In *A. flavus* the sporangium may either be yellow or green, in the others it is grey or black. *Penicillium glaucum* has also been found.

## DISEASES OF EXTERNAL EAR

**The Pinna** is subject to the diseases which affect the skin in other parts. Thus eczema, as mentioned, is common and should be treated as it would be elsewhere, but it may be kept from healing by a chronic discharge from the middle ear and treatment must include measures to arrest this chronic otorrhœa. Tumours which affect the skin elsewhere also affect the skin of the ear, so that sebaceous cysts, rodent ulcer and carcinoma are not uncommon. Hæmatoma auris, an effusion of blood between the cartilage and perichondrium, producing a purple swelling of the auricle, is said to be common among the insane, and it occurs among boxers and footballers as a result of injury. The hæmatoma does not usually suppurate, but in a few cases some deformity of the auricle results.

### INCREASED SECRETION OF CERUMEN

Wax in the external auditory meatus may take place rapidly in response to some irritant, so that a collection is formed. The precise mechanism by which the amount normally secreted is removed from the meatus is not known with certainty. Probably the movements of the jaw cause it to be gradually expelled. If it collects into a plug, causing deafness, or reflex symptoms, such as cough, it should be removed by syringing with warm water (Fig. 187). If the wax is too

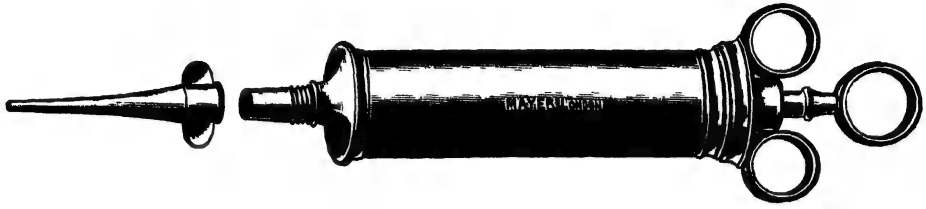


FIG. 187

An aural syringe. (*Mayer & Phelps.*)

hard for this, it may be first softened by instilling olive oil or a strong solution of bicarbonate of soda. The introduction of water into the ear often determines the sudden onset of deafness from this cause, by causing the cerumen to swell and produce complete obstruction.

### FURUNCULOSIS

Furunculosis is common in the external auditory meatus, and its bacteriology has been mentioned. It may be associated with boils elsewhere, but more often this is not the case, and it may be the first indication of diabetes. Pain is very severe on account of the unyielding texture of the tissues, and the ear is tender on manipulation. The swelling of the meatus may cause obstructive deafness. If the boil is situated on the posterior wall, swelling behind the ear will simulate periostitis of the mastoid process, but the groove behind the auricle is obliterated by a furuncle in this position. Diagnosis is occasionally extremely difficult, in which case the presence of staphylococcus aureus in the discharge is in favour of the diagnosis of furunculosis, but this is not entirely reliable, and a radiogram of the mastoid process may be of more help. A fistula from the mastoid process opening on the posterior meatal wall occasionally simulates a furuncle, if there are granulations at the orifice. In rare cases the pus from a furuncle may reach the tympanum through the notch of Rivini and cause otitis media. Incision of a furuncle is not usually required. Hot applications and gentle cleansing only are necessary. For recurrent boils vaccine treatment is useful. The antigen employed should be injected intradermally, rather than subcutaneously, in order to desensitise the skin.

### DIFFUSE EXTERNAL OTITIS

This condition, as mentioned in connection with bacteriology, is common. As local treatment, syringing with a dilute lotion of liquor carbonis detergens or liquor picis is useful, or a cream of zinc or calamine may be applied sparingly. In cases of aspergilliosis, after cleansing the ear a solution of perchloride or biniodide of mercury in strong alcohol produces a rapid cure. Oily applications or ointments should not be applied to relieve the itching, as they encourage the growth of the fungus.

### OSTEOMA OF THE MEATUS

This may take the form of either exostosis or a diffuse hyperostosis. Exostoses are frequently multiple and sometimes symmetrical in both ears. Virchow attributed their origin to some abnormality of development in the annulus tympanicus. Frequent sea bathing has also been suggested as a possible cause. Exostoses may cause obstructive deafness, and less commonly tinnitus and vertigo. They should not be removed unless causing symptoms. If pedunculated they can be removed through the meatus, but if sessile, as is usually the case, the auricle must be turned forward and the tumours removed with mallet and chisel, taking great care to avoid damage to the tympanic membrane. When a chronic discharge from the tympanum is associated with exostoses, they are a source of danger, as they obstruct the drainage from an area of caries, and in this case the radical mastoid operation is indicated to remove them and cure the discharge from the middle ear.

### INJURIES AND FOREIGN BODIES

Injuries to the external auditory meatus may be caused by foreign bodies, and are still more likely to result from attempts at removal. A foreign body of itself causes little damage and may be left alone temporarily, unless it is touching the tympanic membrane when severe pain will be produced. On the other hand, if a smooth round object is seized with forceps, it is more likely to slip further in than to be held firmly, and the instrument will probably injure the walls of the meatus. The meatus should be examined with a speculum and, if a chink is present between the foreign body and the meatal wall, a hook may be passed through and the foreign body drawn out, or a jet of warm water directed into the chink with a powerful syringe will wash the foreign body out of the ear. If there is no space to allow either of these manoeuvres, it is necessary to turn the auricle forwards and extract the foreign body through an incision in the posterior meatal wall. The irritation of an aural discharge sometimes explains why a child introduces the foreign body. The foreign body may then obstruct drainage, and cause severe symptoms. It may find its way through a perforation into the middle ear, and even into the mastoid antrum. In these circumstances the child is in a dangerous position, and if the foreign body is not extracted skilfully a septic wound or meningitis may result. Injuries may also be caused by indirect violence, especially by a blow or an

explosion. In such cases the tympanic membrane is more likely to suffer. A blow causes a linear split which becomes an oval or round perforation. The edges bleed, but if the ear is kept dry and all syringing avoided, healing usually takes place without suppuration. If an explosion, such as the bursting of a shell, ruptures the membrane, the edges are usually found to be everted, and almost the entire membrane may be blown out in this way. The bleeding may be profuse, and the temptation to syringe out the ear must be resisted. Rupture of the membrane seems to protect the labyrinth, but if the explosion does not rupture the membrane and only produces some punctate hæmorrhages, the labyrinth may be concussed and a high degree of internal ear deafness may follow. Recovery is more likely from deafness produced thus than from the deafness caused by long exposure to the sound of gun fire without any visible change in the tympanic membrane.

### DEAFNESS

In addition to the causes of deafness already indicated, a chronic exudative catarrh of the middle ear with secondary adhesive processes is common. It can be relieved by treatment with the Eustachian catheter.

It must be distinguished from **otosclerosis**, which in its early stages has also the characters of a middle ear deafness, but there are no gross changes in the tympanic membrane and there is no improvement in hearing after inflating the tympanum by the Eustachian tube, which is dry and clear. Otosclerosis occurs usually in young women. The underlying pathological process is an absorption of bone in the capsule of the labyrinth, followed by the deposition of new spongy bone, which in its turn becomes compact. If the amount of new bone deposited is excessive, exostoses eventually are formed. During the process of absorption, the cartilages lining the oval window and the footplate of the stapes disappear and are replaced by bone, so that the stapes becomes fixed in the oval window. Deafness is produced thus, and this form of fixation of the stapes is associated with distressing tinnitus, far more severe than that caused by catarrhal otitis media. **Paracusis Willisii**—the patient seeming to hear better in a noise—is a frequent symptom. The pathogenesis is unknown, but the disease is aggravated by pregnancy, parturition and lactation, and as there is in addition a hereditary element in its incidence, the question of marriage in sufferers from this condition requires serious consideration. A variety of drugs has been tried, and it is said that parathyroid extract is sometimes beneficial, but it is doubtful whether any treatment is of avail, except that directed to the improvement of the anæmia which often accompanies the disease. For the tinnitus hydrobromic acid is usually recommended. The mixed bromides often act better, and sometimes small doses of calomel (gr.  $\frac{1}{4}$ ) are useful. Extraction of the stapes has always failed.

The various forms of otitis media produce different degrees of hardness of hearing, but there is never total deafness unless the cochlea or the auditory nerve is destroyed by injury or disease.

The most severe forms of deafness are caused by disease which affects these two structures. Clinically it is difficult to distinguish between the two, except by eliciting a history of labyrinthitis with vertigo, vomiting and nystagmus, or by electrical stimulation of the auditory nerve. If the VIIIth nerve responds to electrical stimulation the lesion must be confined to the labyrinth, but the patient will not sway or fall if the function of the nerve has been destroyed. Thus the deafness which results from a non-suppurative effusion into the labyrinth in mumps may begin with an attack of vomiting, vertigo and nystagmus, locating the lesion in the labyrinth. This form of deafness is usually unilateral, but both ears may be affected. The deafness caused by cerebrospinal meningitis or congenital syphilis is usually bilateral, and if the patient is attacked early in life, deaf mutism may be the result. Unilateral deafness of the internal ear should give rise to the suspicion of acquired syphilis or a tumour of the auditory nerve. Toxic deafness, such as that caused by quinine or malaria, is usually bilateral and permanent, but in typhoid fever it is transient. It has been suggested that congenital deafness may be caused by quinine administered to the mother during pregnancy, either as an abortifacient or for other reasons. Such deafness would cause mutism and caution against such risk is evident. Senile deafness, from degeneration of the auditory nerve, is also usually bilateral and is not accompanied by tinnitus. The presence of this symptom is commonly due to an accompanying arteriosclerosis. In all old people the ligamentum spirale shows signs of atrophy, and thus the basilar membrane in the lower whorl of the cochlea loses its tension, causing a loss of hearing for high notes.

## DISEASES OF MIDDLE EAR

### ACUTE OTTIS MEDIA

Acute otitis media is often preceded merely by an ordinary sore throat or nasal catarrh, but the common cause is an attack of influenza. The exanthems are also an important cause, especially scarlet fever, measles and diphtheria. It occurs occasionally during the course of typhoid fever. The pathogenic organisms are usually either the hæmolytic streptococcus pyogenes, which is the infective organism in scarlet fever, the *S. viridans*, or the pneumococcus. The pneumococcus mucosus (Type III. pneumococcus) is especially dangerous, for although at first the symptoms are mild it may cause meningitis. *S. aureus* is occasionally found in pure culture. Other organisms are rare. *S. pyogenes* is the most important on account of the bone destruction and liability to intracranial complications associated with it. The wide straight Eustachian tube of infants favours the occurrence of otitis media, and in children the presence of adenoids is a common predisposing cause. It is also liable to occur as a complication after operation on the nose or nasopharynx. Diabetics are liable to it, as to other forms of suppuration.

In the early stage there is hyperæmia of the mucous membrane, followed by a serous exudation often accompanied in influenzal cases by small hæmorrhages. The exudate soon becomes purulent and the tympanum contains at first serum, then mucus or mucopus. The inflammation may be limited to the tympanum, but it usually extends to the mastoid antrum and all the spaces connected with the middle ear.

*Clinical Picture.*—Pain and deafness are the most prominent symptoms. Tinnitus of a pulsating character may be present, though it is not distressing. The pain is often severe, worse at night, and radiates over the side of the head and down the neck. There may be some tenderness over the mastoid process. The pain is sometimes quite slight, especially in scarlet fever and diphtheria. On examination at an early stage the tympanic membrane is seen to be flushed and shows a network of dilated vessels, but the outline of the malleus is still visible. In a few hours the membrane is seen to be bulging and bright red in the posterosuperior quadrant. A little later its whole surface is uniformly red and bulging, so that all the normal landmarks, including the malleus, are obscured. In influenza there are often blebs on the surface of the membrane containing blood-stained serum. If the membrane is not incised, a yellow spot appears, most commonly in the posterior quadrant, where rupture takes place and pus is discharged into the meatus. The inflammation may be limited to Shrapnell's membrane, and resolution begin at any stage before rupture occurs. The pain is usually relieved instantly by rupture or incision of the membrane, but this is not invariable and persistence of pain does not necessarily indicate retention of pus in the mastoid process, although it is a suspicious symptom. In infants, in whom the membrane is thick, it may be bulging, but dull white or grey and not red as in older patients. At the onset there is usually, but not invariably, a rise of temperature, most pronounced in children, in whom it may reach 103° or 104° F.

*Treatment.*—The pain is best relieved by instillations of glycerin of carbolic acid into the meatus and the application of moist heat to the side of the head. If the membrane is seen to be swollen and congested and there is any sign of bulging, especially in the posterosuperior quadrant, it should be incised through the most prominent part. Nitrous oxide gas or a short general anæsthetic is preferable to local anæsthesia, as the pain of incision is severe though momentary. The exact site of the incision should be decided and the speculum kept in position while the gas is administered. Keeping the eye on the field the whole time, a free vertical incision is made with a paracentesis knife. There is thus no risk of mistaking the situation of the membrane and of injuring the inflamed posterior wall of the meatus. A wick of gauze left in the meatus assists drainage and the ear should be covered with a large dressing. It is better to syringe the meatus four or six hourly with a dilute warm solution of biniodide of mercury than to allow the discharge to stagnate in the meatus, though some surgeons consider syringing to be harmful. In the majority of cases resolution follows with healing of the perforation and restoration of hearing. Politzerisation is often necessary to restore the hearing fully. In a few cases

intratympanic adhesions, especially around the stapes, may cause permanent deafness.

In spite of incision of the membrane or spontaneous rupture, the febrile symptoms may persist and the discharge continue undiminished or only slightly diminished in quantity. Such symptoms, especially if accompanied by persistent bulging in the posterosuperior quadrant of the membrane and tenderness at the tip of the mastoid, indicate retention of pus in the mastoid process. The mastoid antrum and cells should then be opened by the simple mastoid operation without waiting for swelling or periostitis of the mastoid process to appear. In this way the tympanum is freed from the pus reaching it from the mastoid process, the hearing saved, and at the same time the patient is placed in comparative safety.

*Complications.*—During an attack of acute otitis media the patient must be watched for any signs of irritation of the labyrinth, of meningitis, or of infection of the great venous sinuses. Cerebral or cerebellar abscess may occur, but they are rare complications of acute otitis.

It is, however, not uncommon for a collection of pus to form between the roof of the antrum and the dura mater or around the lateral sinus. Extradural abscess in the middle fossa is less common than perisinus abscess. They present no characteristic symptoms, except an unusual quantity of discharge and pain radiating towards the vertex. They are usually discovered during the course of a mastoid operation.

A rare situation for extradural abscess is at the apex of the petrous. In addition to general symptoms, such an abscess is liable to cause paralysis of the VIth nerve, shown by an external rectus palsy, and neuralgia in the first or first and second divisions of the Vth nerve (syndrome of Gradenigo). The prognosis of an abscess in this situation is grave, but the symptoms may be produced by a serous effusion, in which case the patient can recover without operation. A leucocyte count is a useful aid in distinguishing between the two conditions.

### CHRONIC SUPPURATION OF THE MIDDLE EAR

The majority of cases of chronic infection follow an attack of untreated acute suppuration which has never resolved. A few, however, arise insidiously and are tubercular in nature, and in some others a small perforation is situated in Shrapnell's membrane above the short process of the malleus. In these there is no history of acute otitis, but the membrane atrophies and finally breaks down with chronic attic suppuration as a secondary result.

Deafness and discharge from the ear are the only symptoms in the majority of cases, which explains the neglect of the aural condition, but headache and attacks of pain both in the ear itself and in the temporal region are common symptoms demanding relief. Examination after cleansing the meatus shows in every case a perforation which is usually central, so that its edge does not reach the marginal attachment to the tympanic ring. Such a perforation may be small or large, circular, oval or crescentic, and there may be adhesions



between its edge and the inner wall of the tympanum. Unless this condition, which would obstruct drainage, is present, such central perforations are innocuous and are not associated with caries of the middle ear, but only with a mucopurulent catarrh of the tympanic mucous membrane.

Marginal perforations, in which part of the circumference is formed by the bone at the periphery of the membrane (Fig. 168, E), indicate caries of the adjacent bone and of the ossicles. Many are situated either in Shrapnell's membrane or the posterosuperior quadrant of the membrana tensa or sometimes in both, and thus lead directly into the attic, aditus and antrum. The epidermis of the meatus can thus spread directly through the perforation along a smooth surface into the antrotympanic cavity. Proliferation of this epidermis, which becomes exfoliated in successive layers, forms a compact mass of squamous cells, cholesterin and pus which is capable of reaching a large size and eroding the bone deeply and widely. In this way a *cholesteatoma* is commonly formed, though in rare cases it arises as a primary inclusion cyst of the epidermis. Marginal perforations, therefore, indicate caries of the walls of the antrum and middle ear and also of the ossicles. The incus, having a poor blood supply, is affected before the malleus and often disappears. The stapes is affected least. At the same time the cellular bone surrounding the labyrinth and the antrum undergoes a proliferative osteosclerosis whereby the diploic or pneumatic structure becomes replaced by dense compact bone. This tends to limit the advance of the suppuration, but also to render it chronic. On the other hand, acute suppuration occurring in a compact type of temporal bone is liable to become chronic. The compact structure may be congenital and is then the predisposing cause and not the effect of the chronic suppuration.

Granulations bathed in pus may be seen attached to the underlying carious bone and protruding through the perforation. They may reach a large size, become covered with squamous epithelium and fibrous in structure. An aural polypus thus formed often fills the meatus and resembles a fibroma. The attachment is usually to a carious area of the tympanic ring, but it may be to an ossicle, to the promontory or even to the edge of the perforation in the tympanic membrane. For this reason, after simple removal, an aural polypus almost always recurs from the same underlying area of caries.

In cases of central perforation it is only necessary to keep the ear clean by syringing and to dry it by instilling rectified spirit (50 to 70 per cent.), and in most cases healing takes place. It is a dangerous condition only if the edge of the perforation is adherent to the inner wall of the tympanum.

Marginal perforations, accompanied by a foul discharge and cholesteatoma, indicate a progressive destruction of bone for which the radical mastoid operation is required.

### TUBERCULOSIS OF THE EAR

Primary tuberculosis of the ear occurs in infants and young children from infection by milk via the Eustachian tube, which may itself be



affected. A blood-borne infection is also possible. When facial palsy and enlargement of the cervical glands accompany the otitis the diagnosis is almost certain from clinical observation only, but it may easily be missed unless the aural discharge is examined for the presence of tubercle bacilli. A mixed infection soon supervenes.

In a few phthisical patients a tuberculous otitis media develops and must be distinguished from those ordinary forms of middle-ear disease which may, and often do, attack such patients. The onset of this tuberculous lesion is insidious and painless, resembling a mild catarrhal otitis. It may heal, but commonly there is a spontaneous perforation of the tympanic membrane. Incision is unnecessary as there is no pain, and it merely serves to introduce secondary infection. The perforation is sometimes multiple and bacilli may be found in abundance; in other cases they are so scarce that it may be necessary to inoculate a guinea-pig to establish the diagnosis.

### **SYPHILIS OF THE EAR**

Chronic suppuration in the middle ear is occasionally due to syphilis and its true nature is likely to be overlooked owing to its resemblance to the more common forms of otitis media. Such patients will not improve without specific treatment and the wound produced by a mastoid operation becomes indolent and sloughy, and makes no progress towards healing until appropriate treatment is adopted.

The internal ear may be affected by syphilis with or without chronic suppuration in the middle ear, and produces a high degree of deafness, generally unilateral, affecting especially the high tones with loss of bone conduction. There may be tinnitus and very likely vertigo.

The ear is affected in about 10 per cent. of cases of congenital syphilis. Both the middle and internal ear are likely to be attacked; in the latter lymphatic infiltration and miliary gummata exercise a devastating effect with the production of severe or total deafness. Although it may appear much later, it usually does so between the ages of 10 and 20 and may be accompanied by interstitial keratitis and Hutchinson's teeth. The cerebrospinal fluid as well as the blood should be subjected to the Wassermann and Kahn tests.

### **MALIGNANT DISEASE OF THE EAR**

In addition to the epithelial tumours of the pinna, malignant disease may originate in the deep part of the external auditory meatus or in the middle ear. Squamous-celled carcinoma is the usual form but sarcoma and hæmangio-endothelioma also occur. There is usually a long history of pre-existing chronic suppuration in the middle ear, but this is not necessarily so. If in addition to deafness and discharge, pain and bleeding supervene, or there is rapid recurrence after removal of polypi or granulations, suspicion of malignant disease should be aroused and a biopsy performed. As these tumours tend to extend forward into the parotid region around the temporomandibular joint, swelling and pain on chewing are characteristic though late symptoms.

Invasion of the meatus may lead to a mistaken diagnosis of eczema or furunculosis. The disease spreads mainly through the temporal bone and in the parotid region, and penetrates the dura only late in its course, but the facial nerve is certain to be paralysed sooner or later. Radiation gives disappointing results and the best treatment is by a wide excision of the auricle, external meatus and mastoid area of the temporal bone.

### COMPLICATIONS OF MIDDLE-EAR DISEASE

The complications which are liable to arise from either acute or chronic middle-ear disease in addition to labyrinthitis are septicæmia or pyæmia from lateral sinus thrombosis, meningitis, encephalitis and abscess of the brain. Venous infections may follow either acute or chronic suppuration, although it is more common in the chronic type. The converse holds true of meningitis and encephalitis, while abscess is more commonly seen with chronic otitis.

#### LATERAL SINUS THROMBOSIS

The most common extension of infection is towards the knee of the lateral sinus, which projects forwards into the mastoid process to a variable extent, so that the venous wall easily becomes adherent to the bone and infected. This infection may cause a septic thrombus to form on the wall of the vessel, and the blood stream can carry away septic particles producing pyæmia. In these circumstances the infected sloughing condition of the sinus wall is obvious as soon as it is exposed. The bone lesion is a chronic one, almost invariably associated with cholesteatoma. In acute mastoiditis the thrombosis is apt to arise by infection of the small tributaries in the bone and to spread by intravenous extension into the sinus. In these circumstances, the wall of the sinus appears normal on exposure, although it may contain an infected thrombus. In the same way primary thrombosis of the jugular bulb may occur rather by its proximity to the middle ear or from the intravenous extension just mentioned than from actual mastoid disease. It is frequent also for a thrombus in the sigmoid sinus to extend downwards into or even beyond the bulb, and backwards towards the torcula Herophili.

Mastoid disease accompanied by a persistently swinging temperature and especially by rigors, is the usual indication that sinus thrombosis has occurred. The symptoms may, however, be more meningeal in character, and occasionally the simulation of typhoid fever is so close that the aural condition is overlooked. The patient is flushed, has a dry tongue and rapid pulse. A blood culture may show a streptococcal infection of the blood, but it is often sterile. A hæmolytic blood infection may cause a serious lowering of the percentage of hæmoglobin in the blood, demanding occasionally a blood transfusion. The clot may be either mural or obstructive and fill the vessel. In the latter case optic neuritis is frequently observed. If the disease is not controlled, the entrance of septic particles into the circulation will

either cause pyæmia, abscesses forming especially in the lungs, joints, serous cavities and buttocks, or the patient die of septicæmia.

As soon as the condition is recognised, often by the occurrence of a rigor, the mastoid process should be explored and the lateral sinus exposed freely. It is incised and the clot turned out until the ends bleed. It may be necessary to remove bone and incise the sinus far back before bleeding occurs. If the clot is mural or in the dome of the bulb the sinus will bleed when incised. Bleeding should be controlled by inserting strips of gauze between the skull and the sinus, so that its walls are pressed together. If there is no bleeding from the lower end, or if signs of pyæmia have appeared, the jugular vein should be tied. The best plan is to tie off the common facial vein, then to divide the jugular vein low down between ligatures and strip it up. The skin wound is partially closed and the upper end of the vein left hanging out of the upper corner. The ligature can be removed the following day. In this way the vein and bulb are drained and the vein completely cut off from the general circulation. In dressing the patient afterwards the packing used to obliterate the sinus should be removed gradually. By removing an inch or so each day it can be eventually removed without any bleeding. If the patient is anæsthetised and too much removed at once, bleeding is likely to be started again.

Sulphapyridine is of great value in treatment.

#### OTTIC MENINGITIS

Headache, vomiting, rise of temperature and rapid pulse in the presence of otitis media, either acute or chronic, should raise the suspicion of meningitis. Confirmatory signs are those of Kernig and Brudzinski. The former is difficulty in extending the lower limb in the sitting position because of pain; the latter is reflex flexion of the lower limbs, which are drawn towards the pelvis in response to passive flexion forward of the head upon the neck. This meningitis may vary from irritation with an excess of cerebrospinal fluid to a fully developed septic meningitis in which the fluid contains pus cells and living organisms. Intermediate in severity is aseptic meningitis, in which the cerebrospinal fluid is clear or turbid, for it does not become opalescent until it contains 150 to 200 cells per c.mm. and is free from living organisms.

Lumbar puncture should be performed if it is fairly certain that there is no abscess in the brain, in which case withdrawal of even small quantities is highly dangerous, causing either sudden death or rupture of the abscess. Glucose may be absent or diminished, protein is enormously increased, but the most important points in both diagnosis and prognosis concern the cell count and the chloride content. A cell count above 5 per c.mm. is indicative of a commencing meningitis. In septic meningitis polymorphonuclear cells predominate; an excess of lymphocytes suggests a chronic protective reaction as in brain abscess or tuberculous meningitis. The concentration of chlorides in the cerebrospinal fluid is normally above that in the blood, but in meningitis

the barrier breaks down and the concentrations tend to approximate. A fall of chlorides in the fluid to 680 mg., that is 0.68 per cent., is a bad prognostic sign. Bacteriological examination should be made both of the aural discharge and the cerebrospinal fluid. The local irritant to the meninges or focus of infection should be eliminated by an appropriate mastoid operation, either limited to the cells and antrum in acute otitis or the radical procedure in chronic lesions. Cerebrospinal fluid is withdrawn on alternate days, observation being kept upon the cell count and chloride concentration. Attempts at drainage or lavage of the meninges are useless and in fact harmful. Prognosis has been much improved by the use of sulphapyridine, but chemotherapy cannot succeed unless the primary focus of suppuration has been thoroughly eliminated.

### OTOGENIC ENCEPHALITIS

The inflammatory complications of otitis media within the cranium are not necessarily suppurative, though the early stage of a brain abscess is an encephalitis which ends in pus formation. If this encephalitis is of a mild character it may produce only slight symptoms of raised intracranial pressure such as headache and an attack of vomiting, and localising signs such as transient aphasia or an extensor plantar response. More severe encephalitis of a hæmorrhagic type may even be fatal without producing pus either local (abscess) or general (meningo-encephalitis); this type usually occurs in acute otitis.

The recognition of otogenic encephalitis is important because if it is mistaken for abscess a futile and harmful exploration of the brain may be undertaken. The patient is febrile and has those signs and symptoms suggestive of an abscess. The cerebrospinal fluid, however, is clear and sterile, but may contain a small number of cells. If the dura is incised the swollen brain bulges through the opening but no abscess can be discovered.

### OTOGENIC INTRACRANIAL ABSCESS

Most intracranial abscesses arise as a complication of chronic suppuration in the middle ear and mastoid antrum, generally associated with cholesteatoma. The usual situations are the temporal lobe and cerebellum, about twice as often in the former as the latter. Abscesses of otitic origin in other parts of the brain such as the occipital lobe are rare.

If infection spreads slowly through the tegmen of the middle ear or antrum the meningeal spaces are sealed off, the cerebral tissue becomes inflamed and finally an abscess is formed. In this case it may have a stalk connecting it with the primary focus in the ear, and along this stalk it can be drained. Such a track is not always present, however, and the abscess is separated from the tegmen by a layer of macroscopically intact brain tissue. In this type suppuration results from breaking down of infected clot in a thrombosed vein. A chronic abscess develops a thick protective wall and may become so encysted that it can be shelled out of the brain.

An abscess in the temporal lobe may rupture into the lateral ventricle, but this accident is sometimes prevented by the obliteration of the descending horn by pressure of the abscess. Lumbar puncture, however, may cause such a rupture. A cerebellar abscess is likely to compress the brain stem and so cause distension of the ventricles, thus producing an internal hydrocephalus above the tentorium and increasing still further the symptoms of compression. Pressure below the tentorium causes herniation of the brain stem into the foramen magnum. This makes lumbar puncture in cerebellar abscess very dangerous owing to the risk of respiratory failure from sudden alteration in pressure.

**Cerebral Abscess.**—General symptoms are headache, giddiness, vomiting and slow cerebation. Usually the temperature is subnormal and the pulse slow because the effects of compression outweigh those of septic absorption, but this is not necessarily the case, and both

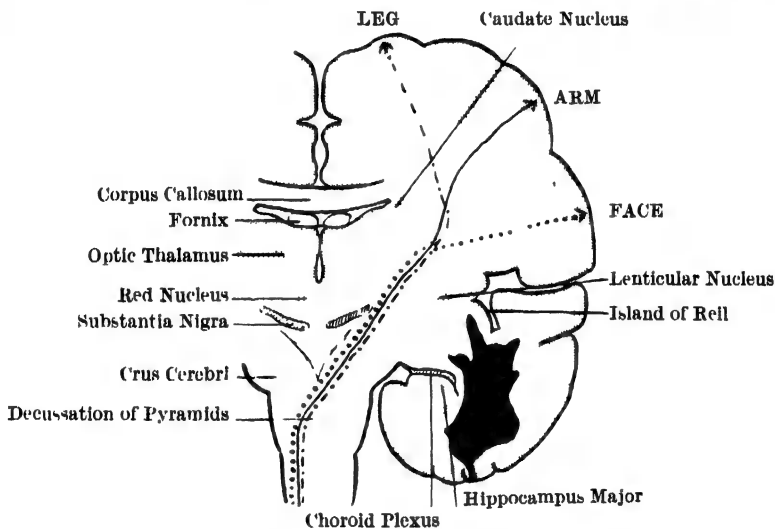


FIG. 188

Cerebral abscess (in black) showing directions of spread and structures likely to be compressed.

temperature and pulse may be raised in the early stages. Optic neuritis is frequent, especially in subtentorial lesions, but it has no localising value. Lumbar puncture should be avoided for the reasons stated, but if a small quantity of cerebrospinal fluid is examined it will be found to have a normal chloride and glucose content with a low or slightly raised cell count due to a localised meningitis. If the abscess is leaking into the ventricle or meningeal space, the fluid will be turbid.

There may be no localising symptoms, but in temporal lobe abscess pressure upwards upon the pyramidal tract may cause a contralateral weakness of the facial muscles. Pressure exerted more directly inwards may cause a contralateral hemiplegia, in which the lower limb is most affected, the fibres to the leg being most exposed to pressure (Fig. 188). A fixed dilated pupil on the same side from pressure upon the oculomotor nerve is also a common sign in this condition. In right-handed persons abscess in the left temporal lobe might produce

aphasia, but this is not common. It should be tested by asking the patient to name a number of common objects such as pen, pencil or watch, and it may be necessary to use several things, as otherwise a mild degree of aphasia may escape detection.

**Cerebellar Abscess.**—In this area there may be no localising signs; the reasons for this is explained in Fig. 189. If present signs are due to a homolateral loss of postural tone, shown by ataxy of the arm and leg on the same side as the lesion. There is often nystagmus, but this may be of labyrinthine origin. In this latter the coarse movement is to the opposite side and is transient, whereas in a cerebellar abscess a similar coarse movement is to the same side and is maintained. But a cerebellar and labyrinthine lesion may coexist. In cerebellar abscess the patient may lie on one side in flexion with the eyes deviating towards the sound side, or the head may be tilted with the chin pointing to the sound side and the occiput turned towards the side of the lesion.

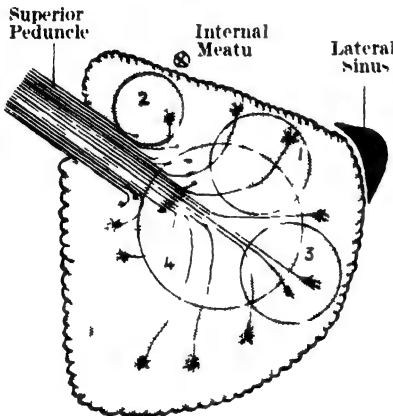


FIG. 189

Sites of cerebellar abscess.

Those in circles 1 and 3 will give no localising signs; those within 2 press upon the peduncle; and those within 4 involve the dentate nucleus, thereby producing symptoms.

**Treatment.**—There should be no hurry to operate as long as an abscess is only suspected, since non-suppurative encephalitis may subside, but immediately the presence of an abscess has been established it must be drained without delay, the patient being in danger of sudden death from respiratory failure.

Whenever possible an abscess should be drained along its stalk. The first step is a radical mastoid operation, removing the tegmen tympani et antri freely to expose the middle fossa and the bone internal to the lateral sinus to give access to the posterior fossa. In this way a track leading into either the temporal lobe or the cerebellum will often be found. If the abscess cannot be reached in this way the brain may be exposed through a separate incision. This is required more often in cerebellar than in cerebral abscess (see p. 851).

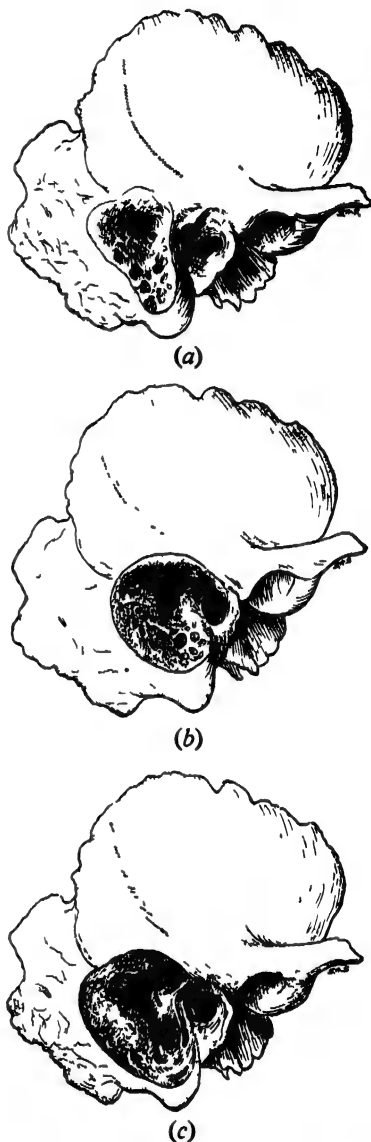
## OPERATIONS ON THE EAR

The operation for acute suppuration in the mastoid process following otitis media, often called *Schwartze's operation* (Fig. 190 (a)), aims to preserve the middle ear intact. A curved incision in the skin is made half an inch or less behind the auricle and carried down to the bone. It should extend upwards as far as possible without injuring the temporal fascia and far enough downwards to expose the tip of the mastoid process. It is often advisable, and even essential, in order to obtain a better exposure to make another incision, 1 in. in length, directly backwards from the junction of the middle and lower thirds of the first incision. The mastoid process is exposed with

a rugine, but the cartilage of the meatus is not detached from the bone and the posterior wall of the bony meatus is preserved. The cells and antrum are then opened with a gouge, beginning at the tip of the mastoid process. The various groups of cells already mentioned (p. 380) must each be sought and opened up. If a probe is passed into the aditus from the antrum, care must be taken not to dislocate the incus by touching the short process. The resulting cavity is often very large. If the lateral sinus projects much forwards into it the wound may be stitched up and drained at the lower angle, but it is usually safer to leave the wound wide open and allow it to granulate. Although the healing takes longer, there is much less risk of residual abscess forming later, a common occurrence after primary or even secondary suture of such wounds.

The operation for chronic suppuration of the middle ear, called the *radical mastoid operation*, aims to throw the middle ear, attic, aditus and antrum into a single cavity. It is the preliminary stage in operations for the relief of the intracranial complications of chronic middle ear suppuration (Fig. 190 (b) and (c)).

A curved incision is made behind the auricle which is turned forwards, and the back of the cartilaginous meatus is detached from the bone. With mallet and gouge the bone lying behind the upper part of the posterior meatal wall is cut away until the antrum is opened. There is thus left between the antrum and the attic a bridge of bone forming the outer wall of the aditus. This bridge is cut away and also the outer wall of the attic. The bridge and posterior meatal wall may be cut away until the horizontal semi-circular canal is exposed. It can be recognised as a prominence of dense white bone in the floor of the aditus, and must not be injured. The malleus and incus, if present, are removed and the whole cavity rendered smooth. In curetting the tympanum and opening the Eustachian tube, the facial nerve must be avoided. It runs across the inner wall of the tympanum in the aqueduct of Fallopius and then turns down between the anterior extremity of the horizontal canal and the oval window towards the stylomastoid foramen. Consequently, curetting from behind forwards is much less likely to cause damage than if the curette is used in the opposite direction. It is also necessary to avoid



(Butterworth.)

FIG. 190

(a) Schwartze operation: the middle ear remains intact. (b) Radical operation: the posterior meatal wall and ossicles removed. (c) Shows the relation of the semicircular canals and facial nerve to the cavity produced by the radical operation.

dislocation of the stapes, an accident which would open the path for infection of the labyrinth. The operation is completed by enlarging the meatus and forming a flap which helps to line the bone cavity. A narrow knife is passed down the meatus along a director and made to cut outwards along the floor of the meatus to its junction with the pinna ; it is then swept upwards and backwards just outside the concha to the roof of the meatus. The cartilage and fibrous tissue are dissected off the flap thus formed and the skin left is stitched back to the deep surface of the pinna. The whole cavity is packed lightly, the pinna replaced, and the post aural wound sutured.

It is advisable in most cases to reopen the wound a week or ten days later, cover the cavity of bone with a Thiersch graft from the thigh and restitch the wound.

If this is not done the cavity must be packed either with a strip of gauze soaked in eusol or with boracic powder changed daily until the epithelium has spread from the edges of the skin flap all over the cavity. This takes from six to twelve weeks.

LIONEL COLLEDGE.



## CHAPTER XXI

### AFFECTIONS OF THE NOSE AND ACCESSORY SINUSES

**S**URGICAL ANATOMY.—The nasal cavity, situated between the base of the skull and the roof of the mouth, is divided by a median septum into two more or less symmetrical halves called the nasal fossæ.

These communicate with the pharynx by the posterior nares or choanæ. The nasal fossæ are further subdivided into meatuses (Figs. 191 and 192) into which open the various ostia of the accessory sinuses. The nasal fossæ are roughly triangular in shape, the apex of the triangle being the narrow roof formed by the cribriform plate of the ethmoid, while the floor is made up of the palatal processes of the maxillæ and the horizontal processes of the palate bones. The septum is made up of the quadrilateral cartilage in front, which articulates with the perpendicular plate of the ethmoid and the vomer behind. The posterior edge of the vomer divides the choanæ.

The lateral wall of each nasal fossa is a complicated structure. There are three overhanging scroll-like laminæ of bone called turbinates or conchæ running anteroposteriorly, one above the other, which divide the outer wall into a corresponding number of grooves or meatuses. The turbinates do not quite reach the front of the lateral wall, so that there is a smooth area in front of them, the upper part of which is called the *agger nasi*.

The inferior turbinate, or maxillo-turbinate, is an independent bone covered by a thick mucosa with ciliated epithelium and large venous spaces in the submucous tissue. It overhangs the inferior meatus into the anterior end of which the lachrymal duct opens.

The middle turbinate is a folded-over portion of the ethmoid and covers the middle meatus. The mucosa is rather tightly bound down to the underlying bone which is sometimes expanded by the presence of an ethmoidal cell in its anterior end.

The middle meatus is a most important and complex region and into it open the ostia of the anterior group of sinuses.

The fronto-nasal duct from the frontal sinus opens into the upper part of a semilunar groove, the prominent lips of which are formed by the bulging of underlying ethmoidal cells. High up on the posterior lip of the semilunar groove is a rounded projection called the *bulla ethmoidalis*, on the surface of which some of the ethmoidal cells open. Below this is a sharp crescentic lamella, called the *uncinate process*. At the posterior edge of the semilunar groove is the opening of the maxillary antrum. This sometimes has an accessory opening still farther back.

The superior turbinate, also a projection from the lateral mass of the ethmoid, is much smaller than the middle or inferior turbinates. Behind and above its posterior end is a space called the *spheno-ethmoidal recess* into which open the posterior ethmoidal cells and the sphenoidal sinuses. The recess lies in the angle between the ethmoid and the anterior surface of the body of the sphenoid. It is rather narrow, since the most posterior

ethmoidal cell is, as it were, plastered on to the outer part of the anterior face of the sphenoidal sinus.

The nasal mucous membrane is usually divided into two portions, the *pars respiratoria* and the *pars olfactoria*, according to the different function which each fulfils. The olfactory portion, which lines the upper third of the outer wall of the nose and nasal septum, has a thick greyish-yellow felted



FIG. 191

A drawing showing the outer wall of the left nasal fossa.

A, the inferior turbina; 1, the uncinate process; 2, the bulla ethmoidalis, with the opening of the antrum immediately below and behind (a rod protrudes from it); 3, the middle turbinal; 4, the superior turbinal; 5, the sphenoidal recess; 6, the frontal sinus; 7, the sphenoidal sinus; 8, the middle meatus.

appearance and contains bipolar perceptive olfactory cells, the peripheral processes of which pass to the surface of the neuro-epithelium and end in olfactory hairs. The central processes become grouped into about twenty bundles called the olfactory nerves, and these pass through the cribriform plate, pierce the meninges and so enter the olfactory bulb of the brain.

The respiratory portion, which lines the lower two-thirds of the nasal

cavity and is continued through the ostia into the paranasal sinuses, is covered by a columnar ciliated epithelium. The mucosa varies in thickness in different parts, and has a rich blood supply in the submucous tissue which shows in certain places definite cavernous plexuses. These are most marked over the inferior turbinate.

The blood supply of the nose is mainly derived from the sphenopalatine artery (a branch of the internal maxillary) and the anterior and posterior ethmoidal arteries from the ophthalmic. The descending palatine and the pterygopalatine arteries (branches of the internal maxillary) also contribute a little.

The venous system drains backwards into the sphenopalatine vein, forwards into the anterior facial vein and upwards into the ethmoidal

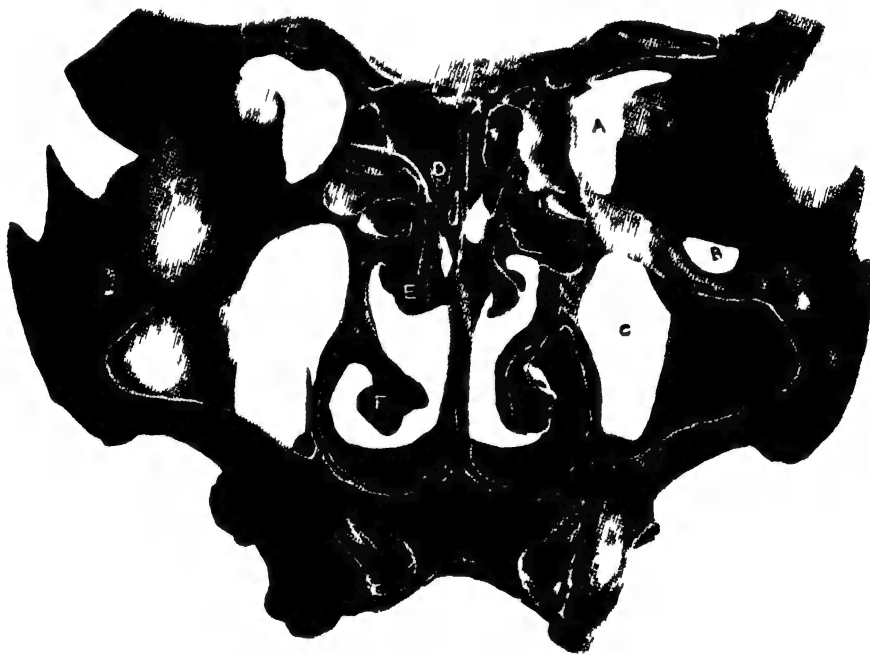


FIG. 192

A section through the bones of the face looking backwards. The orbits, antra and nasal fossæ are shown.

D, E and F are the superior, middle and inferior turbinals respectively.

veins. These latter communicate with the ophthalmic vein, the veins of the dura and the sagittal sinus.

The nerve supply is chiefly derived from the ophthalmic and maxillary divisions of the trigeminal. The back part of the cavity is supplied by the posterior superior and posterior inferior nasal nerves, the nasopalatine nerve, and the anterior palatine nerves which come from the sphenopalatine nerve and ganglion. The anterior part of the cavity is supplied by the anterior ethmoidal nerve from the ophthalmic division.

The lymphatics from the anterior part of the nasal cavity drain into the facial and submaxillary group of lymph nodes. There is a free anastomosis between the intranasal anterior lymphatics and those of the skin over the external nose. The lymphatics from the posterior part of the cavity drain

backwards to the lateral wall of the nasopharynx and then to the deep cervical glands. Some lymphatics drain into the nodes of the retropharyngeal space, and these appear also to be the destination of the main lymphatics from the paranasal sinuses.

## THE NOSE

### NASAL OBSTRUCTION

This is such an important factor in the predisposition to and the production of inflammatory conditions of the nose and accessory sinuses that it may be well to consider the chief causes of nasal obstruction that are met with before discussing the inflammatory conditions themselves.

Cavities lined with mucous membrane are predisposed to inflammation when their drainage and ventilation is obstructed. When any obstruction which may have been present is removed the inflammation, in a large number of cases, undergoes resolution. Obstructions may occur either on the medial wall or on the lateral wall of the nasal cavity, and may be anterior or posterior. They may also be behind the nasal cavity itself in the nasopharynx. The situation of the obstruction is of considerable clinical significance.

Obstruction in the inferior meatus is mainly due to horizontal crests or spurs low down on the septum. These may diminish the air-way through the inferior meatus and, by causing turgescence, lead to hypertrophy of the inferior turbinate, which then adds further to the obstruction. It is usually the anterior septal obstruction which, by creating a partial vacuum behind it, leads to turgescence of the turbinate. Apart from septal obstructions, hypertrophy of the inferior turbinate from other causes, such as sinus suppuration or adenoids, may give rise to an almost complete blockage of the inferior meatus. The drainage from the accessory sinuses is not interfered with by this type of obstruction.

Obstruction in the middle meatus is a more serious affair. High deflections of the septum, for instance, may press on the middle turbinate and so lead to retention of secretions in and diminish the ventilation of the anterior group of sinuses.

In addition to high deflection of the septum there are several conditions involving the outer wall which may cause obstruction in the middle meatus. Amongst these may be mentioned accessory cells in the middle turbinate, overgrowth of the middle turbinate, an accessory cell in the uncinate process, or an enlarged bulla ethmoidalis (Fig. 193). When the olfactory sulcus is obstructed by septal deviation (or anomalies of the outer nasal wall), the drainage of secretions from and the ventilation of the posterior ethmoidal cells and sphenoidal sinus are impaired, as these drain into that part of the superior meatus called the spheno-ethmoidal recess. The hyperplastic changes in the mucosa which may be brought about predispose these sinuses to infection and inflammation. The importance of obstructive lesions

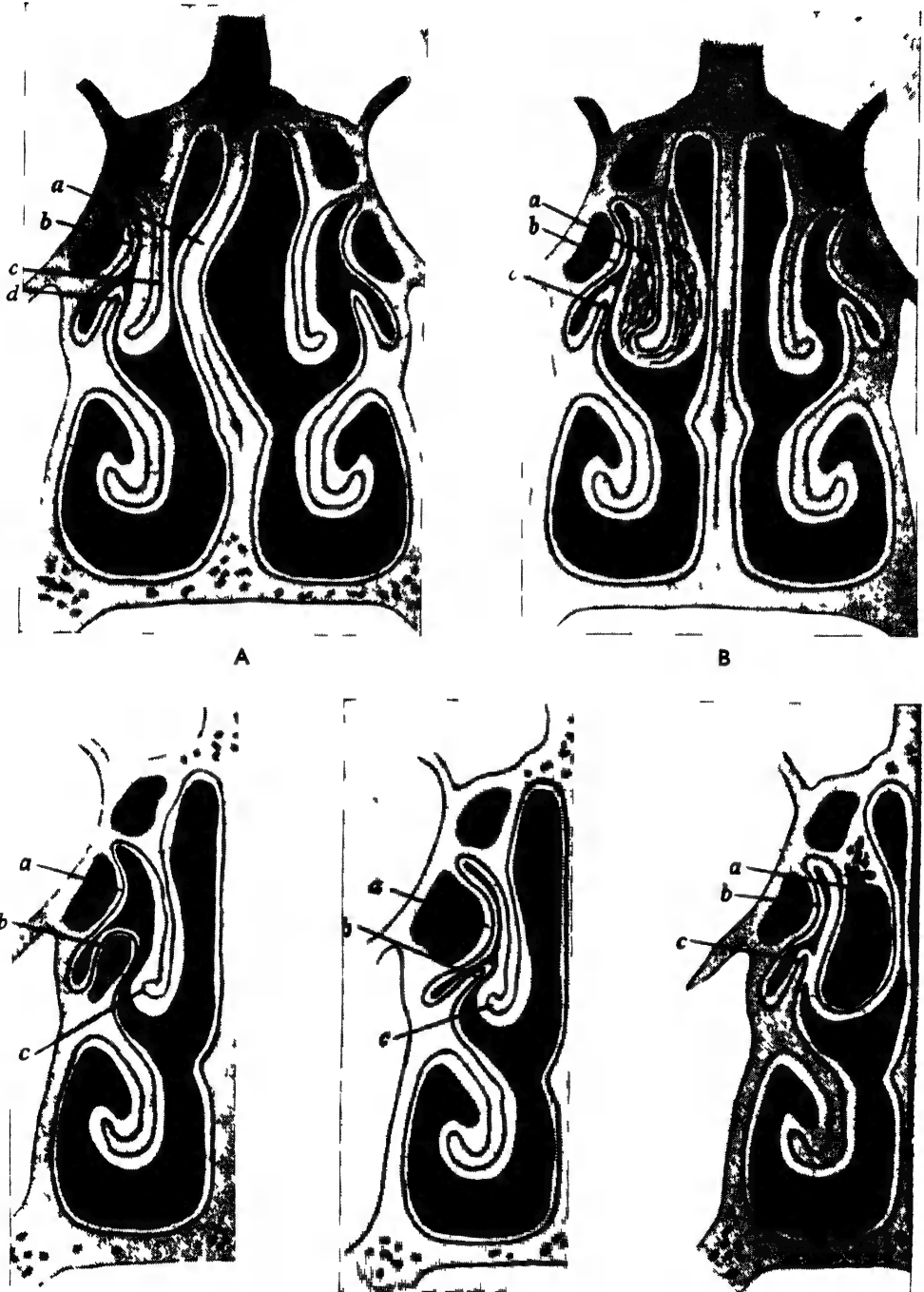


FIG. 193

Various types of nasal obstruction

A, (a) high deviation of the septum, (b) bulla ethmoidalis, (c) middle turbinal, (d) uncinate process B, (a) overgrowth of the middle turbinal, (b) bulla ethmoidalis, (c) uncinate process C, accessory cell in the uncinate process, and D, enlarged bulla ethmoidalis; (a) the bulla, (b) uncinate process; (c) middle turbinal. E (a) large accessory cell in middle turbinal, (b) the bulla, (c) uncinate process.

in the nose as a factor in the production of disease should not be underestimated.

### DEVIATIONS OF THE SEPTUM

Malformations and deviations of the septum may be developmental or traumatic in origin. When traumatic, the deflection is usually confined to the cartilaginous part but, when developmental, all the parts of the septum may be involved. The type and situation of the deviation is not of so much significance as the degree of obstruction which is caused, and this is not always easy to estimate by rhinoscopy. A simple C-shaped convexity may show a concavity on the opposite side, but there may also be a compensatory convexity farther back. Crests, spurs and high deviations in the region of the perpendicular plate of the ethmoid cannot always be accurately defined, and it is very common to find at an operation a spur far back on the vomer which is touching the outer wall. Very few people have a perfectly straight septum, and it is only in cases in which the obstruction is severe enough to give rise to symptoms that treatment is called for. The chief indications are interference with the function of the inferior turbinates and the prevention of ventilation of and drainage from the accessory sinuses.

*Treatment.*—Every degree of septal deflection, however limited, or however complicated, can be dealt with by the operation of sub-mucous resection. This may be carried out under either local or general anæsthesia. The anterior end of the septal cartilage is dislocated into one nostril and an incision is made over this through which the mucosa and mucoperichondrium are raised from the cartilage. This is the most difficult step of the operation, and subsequent success depends on its proper performance. Great patience is often required, and the surgeon should not be content or anxious to proceed until he is satisfied that the elevator can be gradually pushed back along the cartilaginous and bony septum, following its twists and turns and raising the mucoperiosteum from it. This will not be a matter of great difficulty if the elevator is in the right plane and if the side and not the tip of the instrument is used to aid the stripping process. The elevation should be carried upwards towards the cribriform plate and down to the floor of the nose, whilst it should go backwards almost to the posterior part of the vomer. The mucoperichondrial elevation of the other side should then be started by working round the exposed anterior edge of the septal cartilage, and great care should be taken not to make any perforation in the mucosa of the opposite side. When the elevation has been satisfactorily accomplished for both sides of the septum, a long-bladed speculum is inserted astride the cartilage and the mucoperichondrial flaps are thus held away from it. All the deflected portions of the septum, cartilaginous and bony, can be removed with appropriate punch forceps. When this has been achieved the mucoperichondrial flaps will come together and hang straight in the middle line, giving a free and equal air-way on either side. It is advisable to use a light pack in each nostril to keep the flaps in position

and to prevent a hæmatoma from forming between the flaps. The packing may be removed after twenty-four hours.

### HÆMATOMA OF THE SEPTUM

This rarely follows operation and is usually the result of injury. The extravasation of blood causes a smooth, rounded swelling on both sides of the septum and so gives rise to obstruction in each nostril. Inspection will show a soft, red swelling blocking the entrance to each nostril, and its relation to the septum is revealed by the probe.

*Treatment* consists in the first instance in refraining from interference as in many of the cases the clot is gradually absorbed. If this does not take place an incision should be made far forward and the clot turned out. A small gauze drain should be inserted to keep the incision open and a light pack on either side to prevent the hæmatoma re-forming.

### ABSCESS OF THE SEPTUM

This usually occurs when a hæmatoma has become infected, and may take place under an apparently intact mucosa. Symptoms of pain, fever and tenderness usually differentiate it from simple hæmatoma. The abscess should be incised and drained efficiently.

### PERFORATION OF THE SEPTUM

This may occur as the result of a simple perforating ulcer or from syphilis or lupus. It is often met with in chromic-acid workers. Occasionally a traumatic perforation may follow operation on the septum. Perforating ulcer affects the anterior part of the septum near the anterior nares and is usually the result of irritation from dust settling on some slight projection in this region. A small crust is formed which is rubbed or picked off by the patient, but another soon forms. Gradually a small sharply defined ulcer forms under the scab and, as this becomes deeper, it gradually erodes the cartilage and then the mucosa on the other side, until a clean-cut perforation is produced. The patient may be quite unconscious of this or may experience a slight whistling noise on inspiration.

Perforations due to lupus also involve the anterior cartilaginous part of the septum. They are not usually clean-cut and show characteristic lupus nodules at the edges and in neighbouring parts of the mucosa.

Perforations due to syphilis usually involve the bony part of the septum as well as extending forward into the cartilage. They are often irregular in shape, and if they extend high up may be associated with external deformity due to a sinking in of the nasal bridge.

Small perforations may be closed by one of the usual surgical plastic devices but are often best left alone.

## EPISTAXIS

Bleeding from the nose is a symptom of a large number of different conditions. In children it is a common sign of adenoids. It may occur in acute specific fevers such as measles, scarlet fever, diphtheria or typhoid, whilst it is also found in many blood diseases such as hæmophilia, arteriosclerosis, anæmias, etc. It may be due to injury or to the presence of a simple or a malignant growth. Nearly all spontaneous hæmorrhage from the nose arises from vessels on the anterior part of the septum just inside the vestibule. This area is known as Little's or Kieselbach's area. When any excoriation of the mucosa over the plexus of vessels in this region occurs, bleeding is apt to ensue. This can usually be controlled by a small gauze plug. In some cases the bleeding is very troublesome, and intramuscular injections of hæmoplastin or the local application of snake venom may be helpful additions to the plugging. The most satisfactory method of stopping bleeding from this region is by cauterising the bleeding area with the galvanocautery. This seals up the vessels effectively. When the bleeding comes from other parts of the mucosa chief reliance must be placed upon packing and, in some cases, a post-nasal plug may be required. This is, however, seldom necessary and should never be left in for more than twelve hours at a time, owing to possible ill-effects on the ears and sinuses. I have known cases of hæmorrhage that persisted in spite of all treatment stop most dramatically when the patient was transfused.

Profuse bleeding may occur from a hæmangioma of the septum, and is likely to persist until the tumour is removed and its point of attachment cauterised. Severe unilateral bleeding in elderly people should arouse suspicion of a malignant tumour if a possible septal origin can be excluded.

## INJURIES TO THE NOSE

Injuries to the nose are of common occurrence, and may be caused by a fall or by a direct blow. Fracture or dislocation may occur, and this is often associated with damage to and deviation of the septum. Bleeding is often profuse and the internal and external swelling may mask the extent of the actual injury for some time. In very recent cases it may be possible to restore the contour, but in cases of several weeks' duration it will be advisable to refracture the bones so as to mobilise them before they can be moulded into the correct position. An external splint of stent tissue held in position by strapping will keep the bones in proper position. It will often be necessary to put the septum straight by means of a submucous resection.

## FOREIGN BODIES IN THE NOSE

In the large majority of cases these are voluntarily introduced by the patient, usually a child. Boot buttons, beads, beans and peas are among those that are commonly met with. If the history is a definite one there is usually no difficulty in extraction, but a general anæsthetic



should always be employed in children. A bent hook passed beyond the foreign body is the best instrument to employ. The foreign body usually lodges in the middle meatus, but occasionally quite large foreign bodies are found impacted in the inferior meatus. Often there is no definite history, but a unilateral purulent discharge in a child should at once arouse suspicion. Those cases in which the object has been *in situ* for a long time are not by any means easy to deal with owing to the swelling of the mucosa, but after cocainisation and general anæsthesia the foreign body is usually located by the use of a probe.

### THE NASAL MANIFESTATIONS OF ALLERGY

In former years the disorders that come under this heading were classed as nasal neuroses. They comprise so-called vasomotor rhinitis, hay fever and asthma.

#### VASOMOTOR RHINITIS

Paroxysmal rhinorrhœa or allergic coryza is a comparatively common affection and is most often seen in young women and in those whose work lies in dusty surroundings. Tobacco workers, millers, vulcanite polishers, etc., are often affected, but the moulds in ordinary house dust and the fumes of petrol and other things which are met with in daily life are marked etiological factors. In some of those people who have an exaggerated susceptibility to foreign substances and physical agents which are harmless to normal individuals (a condition called allergy), emanations from animals, face powders containing orris root, and various foodstuffs such as eggs and tomatoes are predisposing factors. The *symptoms* consist in sudden attacks of sneezing with a profuse watery discharge from the nostrils, accompanied by marked nasal obstruction. The rhinorrhœa may last a few minutes or may persist for several hours, the patient becoming exhausted by the incessant sneezing. Examination of the nose reveals a pale, boggy, mucous membrane with great swelling of the inferior turbinates. The mucoid discharge contains many eosinophile cells.

*Treatment* should consist in an endeavour to find the determining factor, and if a specific irritant is found from the history or from reaction to skin tests with various allergens, desensitisation may give satisfactory results. The local treatment of the nose is not very satisfactory, although empirical cauterisation of the tubercle of the septum or of the inferior turbinates is successful in some hands. Zinc ionisation is very disappointing, whilst submucous injection of a suitable sclerosing fluid into the inferior turbinates is rather variable in its effects. In cases in which a calcium deficiency is suspected tablets of calcium and parathyroid have proved effective.

#### HAY FEVER

Hay fever is closely allied to vasomotor rhinitis, but the exciting irritant is the pollen of certain grasses or flowers. The grasses which are most commonly responsible are timothy and cock's-foot, and the

hay-fever season lasts only while these grasses are in flower. The symptoms are very similar to those of vasomotor rhinitis with, in addition, intense itching of the nose and conjunctiva, lachrymation and photophobia.

If the patient can be shown to be sensitive to any specific pollen an extract of this will usually effect a cure, whilst prophylactic inoculation before the season begins may confer some degree of immunity. The pollen extract of timothy grass probably gives protection against other grasses. Innumerable solutions have been advised as nasal sprays or douches, of which ephedrine is a usual constituent. In some cases a saturated solution of quinine sulphate is of use, whilst in others oily solutions of 0.5 per cent. menthol or 0.1 per cent. formaldehyde may be effective.

### ASTHMA

About 45 per cent. of all asthmatic patients show allergic reactions. The exciting substances are of great variety and include animal emanations, house dust and cosmetic powders, whilst milk and eggs are among the more common foods which cause it. The most usual cause of asthma of nasal origin is hyperplastic ethmoiditis accompanied by nasal polypi, and sinus infections may be secondary to this. It may be necessary to deal surgically with gross abnormalities of the nose or with infected sinuses, but the result as regards the asthma is often disappointing.

### HEADACHE OF NASAL ORIGIN

It is probably true that sinus disease and intranasal pressure are the cause of more headaches than any other factor, and must always be considered when any attempt is made to diagnose obscure headache. Investigation of nasal headache will involve not only the consideration of abnormal conditions in the nasal cavities and paranasal sinuses, but also the affections of the adjacent nerve structures.

In sinusitis there may be no pain whatever, but its presence in and about the nose may be due to acute inflammation, to chronic inflammation, to pressure contacts in the nasal cavities due to abnormal anatomical conditions, or to nasal new growths. The character and degree of headache depends largely on whether the disease is capable of producing some form of pressure. This is commonly present when the mucosa in a sinus is so swollen that abnormal contacts are produced, or if, owing to a blocked ostium, the products of inflammation are pent up in the sinus. The negative pressure or so-called vacuum headache, if it exists at all, is difficult to prove, but many believe with Sluder that it can cause intense pain in the frontal sinus region.

In acute sinusitis there is usually a constant pain that is more or less localised in the region of the sinus, and a neuralgic type which is periodic and occurs in the distribution of that branch of the trigeminal nerve that serves the affected sinus. In chronic sinusitis the headache is more indefinite, and not so well localised.

Fig. 194 shows the surface areas supplied by the three divisions of the trigeminal, and also that supplied by the occipital nerves, whilst Fig. 195 shows the areas to which pain is usually referred when the various sinuses are affected.

Intranasal pressure is often the cause of headache, and is commonly due to high deviation of the septum and abnormal contact between the mucosa of the septum and that of the middle turbinate, or to such degree of pressure on the turbinate that the ostia of the anterior group

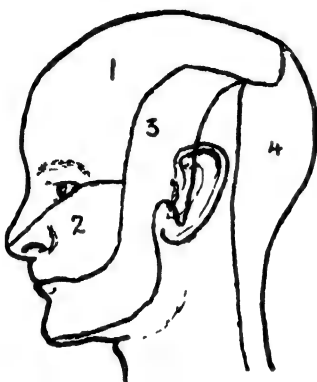


FIG. 194

Nerve supply of face and scalp.  
(After Skillern.)

1, 2 and 3 are the divisions of fifth nerve, and 4 represents the occipital nerve.

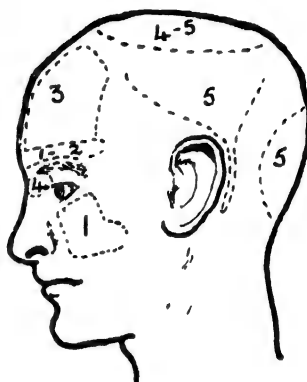


FIG. 195

Areas of referred pain in nasal sinusitis. (After Skillern.)

1, acute maxillary; 2, acute frontal; 3, chronic frontal; 4, chronic ethmoidal; and 5, chronic sphenoidal sinusitis.

of sinuses may be partially occluded and their drainage and ventilation thereby impeded. Nearly all headaches from intranasal pressure depend upon irritation of the sensory nerve endings in the mucosa by some pressure, and the effect that such irritation produces on the sphenopalatine ganglion or the anterior ethmoidal nerves.

## INFLAMMATORY DISEASES OF THE NOSE

### ACUTE RHINITIS

A cold in the head is a very common condition, but its etiology is still uncertain and a satisfactory method of treatment has yet to be found. No specific organism is responsible, but the typical symptoms are as definite and distinct as in many of the acute infectious diseases in which the specific organism has been isolated.

A fit of sneezing accompanied by a feeling of fullness or irritation in the nose is soon followed by a serous discharge, which increases rapidly. The nose then becomes blocked, with loss of smell and headache, due to extension of the inflammation to the mucosa of the accessory sinuses, and with deafness due to extension to the mucosa of the Eustachian tube, whilst general symptoms of malaise and chilliness, sometimes accompanied by a rise in temperature, are

often present. After a few days the discharge rapidly subsides, but the inflammatory process may spread to the pharynx, larynx, or Eustachian tube.

If *treatment* is thought to be necessary it should begin by giving a very hot bath at bedtime followed by 10 gr. of Dover's powder or  $\frac{1}{2}$  gr. of omnopon. Local applications of menthol or camphor in liquid paraffin or insufflations of bismuth subnitrate powder will relieve the congestion and obstruction, whilst tablets containing quinine, belladonna and morphine may help to diminish the unpleasant symptoms. In the majority of cases the affection will run its course uninfluenced by treatment.

### CHRONIC HYPERTROPHIC RHINITIS

This is caused by those conditions which give rise to a chronic hyperæmia of the mucosa and a passive engorgement of the submucous venous spaces. It may result from repeated attacks of acute rhinitis, and is frequently met with in patients suffering from sinus suppuration or in those who are constantly exposed to a dusty atmosphere or work amidst irritating fumes. In cases of marked deviation of the septum the inferior turbinate on the concave side often undergoes hypertrophy, but this is compensatory and is not of necessity pathological. The most common symptom is nasal obstruction. This varies with the state of the weather, being worse in damp weather and in hot atmospheres. It is often worse at night, the side of the nose that is resting on the pillow being the more obstructed. This alternates if the patient turns over on to the other side. This intermittent stenosis is often a characteristic feature of the condition.

On account of the nasal obstruction, mouth breathing is developed, with consequent secondary inflammation in the pharynx and nasopharynx, and the accumulation of sticky secretions that are difficult to dislodge. This often extends to the Eustachian tubes, producing a feeling of fullness in the ears and deafness.

Examination of the nose shows the mucosa over the inferior turbinate to be engorged and hypertrophied; often the anterior end only is affected, or there may be a fringe-like overgrowth along the lower border. In some cases examination with the postnasal mirror reveals a mulberry-like swelling of the posterior end.

The degree of hypertrophy cannot be estimated until the mucosa has been shrunk by the application of a pledget of wool wrung out in a 10 per cent. solution of cocaine. This removes the swelling due to vascular engorgement and leaves the thickening due to true hypertrophy unaffected. Treatment is determined by the amount of hypertrophy, the severity of the symptoms and the underlying pathological conditions which may be found. Mild cases may be relieved by the use of 10 per cent. argyrol drops, but if the degree of hypertrophy is at all severe reduction in the size of the turbinate is essential. This may be carried out with the galvano-cautery, the red-hot wire being plunged through the mucosa and submucous tissue until the bone is reached. If this is done in several places the resulting scar tissue anchors

the mucosa to the bone without undue sacrifice of the ciliated epithelium.

Submucous injections of sclerosing fluid, such as a 5 per cent. quinine-urea solution or a 30 per cent. solution of carbolic acid in paraffin, are also used to effect a similar result by producing scarring in the vascular tissue of the submucosa and the adherence of the mucosa to the underlying bone. If these measures fail, removal of the redundant tissue should be carried out with scissors and forceps or snare. Too much tissue should not be removed, and under no circumstances should the whole turbinate be sacrificed.

### ATROPHIC RHINITIS

Atrophic rhinitis is an atrophy of the nasal mucosa and underlying soft tissues with eventual absorption of the bony inferior turbinate. The etiology of this disease is unknown, although many theories have been put forward to account for it. Many investigators have tried to isolate a specific bacillus, and at one time the *Bacillus foetidus* of Perez was thought to be responsible, but this has not been proved. The most likely explanation is that put forward by Grünwald and others, that it is due to undiagnosed sinus suppuration or purulent rhinitis in children from untreated adenoids. In support of this latter view it may be mentioned that twenty-five years ago cases of atrophic rhinitis were very commonly seen at hospitals, but since children's tonsils and adenoids have been more efficiently treated atrophic rhinitis is a comparative rarity. Some regard it as a deficiency disease and claim cures by treatment with fat-soluble vitamins.

The pathological changes consist in a progressive atrophy of the mucosa and of the underlying turbinate bone, with a destruction of the ciliated epithelium and its replacement by squamous or cubical epithelium. There is no ulceration, but the thick viscid secretion which is exuded dries to form crusts which decompose and give off the peculiar sickly stench that is so characteristic of the disease.

When the nose is examined the nasal cavities are seen to be unusually roomy and filled with greenish crusts. These may also be seen in the nasopharynx. When the crusts have been removed the atrophy of the inferior turbinate and sometimes of the middle turbinate also, allows structures on the outer wall, such as the bulla ethmoidalis and uncinate process, to be seen. The posterior pharyngeal wall and, often, the Eustachian cushion come into view. The only conditions that are likely to cause any difficulty in diagnosis are tertiary syphilis and suppuration in the accessory sinuses. The absence of ulceration should exclude the former and the presence of atrophy the latter. The prognosis as regards the cure of atrophic rhinitis is bad, but it is always possible by suitable treatment to prevent the crusting and thus keep the main symptoms of fœtor and nasal obstruction in abeyance.

The *treatment* mainly consists in cleanliness and in preventing the secretion from drying into crusts. After the crusts have been removed by forceps and by syringing, the nostrils should be packed with long

strips of gauze soaked in a solution of 5 per cent. ichthyol in glycerin. This should be changed each day, and the patients can be taught to do it themselves. Once a week the mucosa should be painted by the surgeon with a solution of 1:1000 formalin. If the treatment is carried out conscientiously the tendency to crust formation will begin to disappear in a few weeks and the mucosa will assume a more healthy appearance, though it is doubtful whether the ciliated epithelium is ever regenerated. At this stage the question of diminishing the abnormal patency of the nasal cavities by surgical means should be considered. There are several methods of effecting this; those most usually adopted are the dislocation of the naso-antral wall inwards, as suggested by Lautenschläger, and the submucous insertion of cartilage grafts on the floor of the nose and along the septum.

### NASAL POLYPI

Nasal polypi are usually pedunculated tumours of hyperplastic tissue, which commonly spring from the middle turbinate, the uncinate process, or the ethmoidal cells, though they may arise in the maxillary, frontal or sphenoidal sinuses. They are no longer regarded as new growths and are purely inflammatory formations due to long-standing rhinitis or catarrhal sinusitis. Microscopical examination shows them to consist of a sort of myxomatous tissue covered with ciliated columnar epithelium. They are often associated with a chronic osteitis or caries of the underlying bone. There is another possible origin for the polypi, and many observers consider that those which occur in vasomotor rhinitis, hay fever, bronchial asthma and some cases of non-suppurative hyperplastic sinusitis are primarily of allergic origin.

Polypi almost invariably spring from some part of the lateral nasal wall, and may be either pedunculated or sessile. Sometimes the pressure of the cedematous masses distends the nasal cavity and causes an obvious broadening of the external nose.

*Symptoms.*—The symptoms are often complex on account of the nasal obstruction and the associated inflammation of the nose and sinuses which usually coexist. The leading symptom is generally nasal obstruction, but its onset may be so gradual as to pass unnoticed by the patient. Headache, watery discharge from the nose and loss of smell are often complained of. Sometimes there is a sensation as of a foreign body and, occasionally, the patient notices the polypus projecting in the nostril. Examination reveals the characteristic smooth, glossy, bluish grey swelling which is freely movable, and which the use of a probe will show to be attached to the region of the middle turbinate.

*Treatment.*—This should consist in the thorough removal of the polypi, along with the bone to which they are attached. It will usually be advisable to deal surgically with any accompanying infection of the accessory sinuses, more particularly the ethmoidal labyrinth. Large single polypi, which are often seen in elderly people, may be removed under local anæsthesia with the cold wire snare, whilst polypi of allergic origin often respond to small doses of radium.

## THE ACCESSORY SINUSES

These are a group of air-containing cavities which communicate with the nose by small openings or ostia. There is reason to suppose that they are residual olfactory organs which have, during evolution, been largely shut off from the nasal cavities. They play a considerable part in the production of serious disease of the face and head owing to their tendency to infection, whilst, from their proximity to the brain and its coverings, they are not infrequently the primary source of intracranial disease. Similarly, their close relation to the orbit may result in cellulitis or orbital abscess, whilst involvement of the optic nerve may give rise to impairment of vision. Mental disease may be due to inflammatory disease in the posterior group of sinuses.

Purulent secretion from infected accessory sinuses may be responsible for inflammatory conditions in other parts of the respiratory tract, while the swallowing of septic material from the sinuses may set up various digestive disorders and gastric disturbances.

### ACUTE SINUSITIS

Acute inflammation of the mucosa lining the sinuses is due in the majority of cases to the extension of infection from the nasal cavities. The inflammatory processes may be simply catarrhal or may pass through the mucopurulent stage into acute suppuration in one or more of the sinuses. This, however, is more often the result of frequent attacks of acute inflammation than of a single attack. The resolution of inflammation depends on many factors, any one of which may affect adversely the return of the tissues to normal. When this inflammation takes place in closed cavities, the drainage from which may be interfered with in many ways, additional factors have to be taken into account. The only general one which need be considered is the nature and virulence of the infecting organism. Influenza is by far the most frequent cause of acute inflammation, but any of the acute infectious disorders, particularly scarlet fever, may be responsible. In the great majority of instances the infection is streptococcal, but the pneumococcus or micrococcus catarrhalis is occasionally found in pure culture. When the infection is a mild one and drainage is not interfered with, the local symptoms of sinus involvement may be merged into those of the acute rhinitis which caused it, and so escape detection, but if the infecting organism is of a virulent type the symptoms may be extremely severe, the pain intense, and intracranial complications be set up.

Of the special factors which must be taken into account, those that are of chief importance are conditions which prevent proper aeration of and drainage from the sinuses. Any form of nasal obstruction, whether it be a deflected septum, hypertrophied turbinates or nasal polypi may induce an undue congestion and oedema of the mucosa in some parts of the middle meatus, whereby the nasal opening of the frontal sinus is markedly obstructed or the drainage from the ethmoidal cells or maxillary antrum interfered with. Similar conditions



affecting the superior meatus may prejudice recovery in inflammation of the posterior ethmoidal cells or sphenoidal sinus.

**Catarrhal Inflammation** of the mucous membrane of the sinuses is common in any illness that is accompanied by acute rhinitis, but retention of the discharge, giving the clinical signs and symptoms of sinusitis, is relatively uncommon. If acute inflammation with retention occurs the temperature is raised and general febrile symptoms with malaise are present. When the **frontal sinus** and fronto-ethmoidal cells are involved there is tenderness on pressure in the supraorbital region and the inner canthus, more particularly at the inner angle of the orbit against the floor of the frontal sinus. The pain has often a very definite periodic character, beginning in the morning, reaching its zenith at midday and gradually passing off entirely during the afternoon, only to reappear the next morning.

When the **antrum** is affected the pain and tenderness are more localised to the nasal bone and malar region, but are very often frontal. If the **posterior ethmoidal cells** and **sphenoidal air sinus** are involved the pain is referred to the occipital region.

These symptoms increase in severity if left untreated, until the pressure in the sinus forces the contents through the natural opening and the pain is relieved by a gush of mucus, mucopus or blood-stained pus from the nose. If relief is not obtained in this way an abscess forms which bursts through the bony wall of the sinus with the formation of an orbital or extradural abscess, whilst extension through neighbouring lymph or venous channels may set up a meningitis or cavernous sinus thrombosis.

In all acute inflammatory conditions in which extension occurs from the nasal cavity, all the sinuses of the anterior group are usually infected, and the maxillary antrum is probably always involved no matter what other sinus is affected. The inflammation in the antrum may settle down quickly, leaving the other sinuses to clear up later; or the other sinuses may clear up and leave the antrum still affected.

The maxillary antrum may be infected from another source, namely, from the roots of the teeth, usually the second premolar and the first and second molars, which project into the antral cavity and are separated from it only by a very thin plate of bone. In cases in which the antrum is infected from an apical abscess or from faulty extraction this cavity alone may be involved.

Examination of the nose by anterior rhinoscopy will show intense congestion of the mucosa, and in some cases swelling of the inferior turbinate, so that the upper part of the cavity cannot be seen until the swollen tissues have been shrunk by means of a 5 per cent. solution of cocaine. It is important to see the middle meatus of the nose to ascertain whether there is any discharge beneath it that may be coming from the anterior group of sinuses. This may be absent, particularly in frontal sinus cases, but the middle turbinate itself will often be seen to be swollen. The olfactory sulcus should be examined for the presence of discharge and, in all cases, posterior rhinoscopy should, if possible, be undertaken.



*Treatment.*—The main object of treatment is to diminish the congestion of the nasal mucous membrane, more particularly in the region of the ostia, so as to facilitate the discharge of inflammatory products from the sinuses. To achieve this result there are many things which can be done both by the surgeon and by the patient. The most valuable procedure on the part of the surgeon is the placing of pledgets of cotton-wool wrung out in a 10 per cent. solution of cocaine and 1 : 1000 adrenalin in the middle meatus and high up towards the spheno-ethmoidal recess. After these have been in position for half an hour considerable contraction and ischæmia of the mucosa will be found to have occurred in the region of the hiatus semilunaris and the under-surface of the middle turbinate, and also in the posterosuperior region of the nose. This relief of pressure by the middle turbinate on the hiatus semilunaris will tend to promote a flow of mucopus from any of the anterior group of sinuses which may be affected. The introduction of cotton-tipped probes dipped in cocaine solution will produce further ischæmia, and rapid improvement may be expected to occur after these applications.

The employment of an electric light head-bath at a temperature rising to 170° and falling to 100° for half an hour is often useful. Short-wave diathermy for twenty minutes each day is also helpful.

Additional measures for promoting discharge can be used by the patient. Amongst these are hot saline douches and mentholised steam inhalations at frequent intervals. These tend to stimulate the ciliary action of the epithelium and promote the flow of inflammatory products from the sinuses.

Although the large majority of cases respond satisfactorily to these measures, there remain a few in whom discharge does not come away freely, more particularly when the maxillary antrum is involved. In these a timely puncture of the antrum through the naso-antral wall and gentle lavage may be sufficient to turn the scale and obviate the necessity for surgical interference. It is, however, rarely necessary.

If, however, expectant treatment should fail or the symptoms become aggravated, some surgical treatment may be inevitable, but this should be as limited in extent as is compatible with the evacuation of the retained discharge. As regards the antrum, a small intranasal opening may be made under the inferior turbinate, and this is usually all that is necessary, whilst for the frontal sinus and ethmoidal cells the dislocation of the middle turbinate or resection of its anterior end with the opening of the bulla ethmoidalis and the passage of a sound into the frontal sinus will usually suffice. If an abscess should present externally in the region of the inner canthus, simple incision and the insertion of a tube will be found to be sufficient, any further operation on the affected cells or sinuses being delayed until the acute symptoms have subsided.

### CHRONIC SINUSITIS

Chronic inflammation of the accessory sinuses may arise as a result of an acute inflammation which has failed to undergo resolution, or from repeated attacks of acute inflammation. In the antrum a

chronic condition may be established owing to infection from a septic tooth without any previous acute attacks.

In a good many cases the presence of chronic sinus suppuration is not suspected, and is often discovered only in the search for a possible source of local or other sepsis. Such local symptoms as bad breath, bad smell, intermittent bad taste associated with nasal or postnasal discharge, nasal obstruction, deafness, pain and headache will call for a careful examination of the accessory sinuses.

#### CHRONIC SUPPURATION IN THE ANTERIOR GROUP.

It is often difficult to determine which sinus or sinuses of this group is affected, as all of them have their openings in the middle meatus of the nose, and any discharge from them will appear in some part of this cleft. It is often only by a process of exclusion that the diagnosis can be established. As the maxillary antrum is very often affected, and as it is easily accessible, it is usual to try to exclude this cavity first.

Examination of the nose after shrinking the swollen mucosa with cocaine may reveal pus in the middle meatus rather far back. If this is wiped or sucked away and the patient is asked to put his head down between his knees with the affected side uppermost, so as to place the normal ostium in a good position for drainage, further nasal examination may show that pus has reappeared in the cleft. This sign (Fraenkel's) is a useful indication of the probability of pus in the antrum. Posterior rhinoscopy may show a streak of pus coming from the middle meatus over the posterior end of the inferior turbinate. We may next proceed to the transillumination test. A small lamp placed in the mouth of a normal patient in a dark room should illuminate the cheek bones, give a bright crescentic infraorbital tache and a red reflex through the pupils. If the antrum is affected a dark shadow may replace the infraorbital tache, and the luminous glow of the pupil be absent. Fluid in the cavity does not always affect the test, but a thickened mucosa diminishes the translucency.

X-ray examination in a suitable postero-anterior position, which should be standardised, may show a blurred outline of the antrum or a definite fluid-level on one side, but the only really accurate test is puncture of the antrum and aspiration or washing out of pus from the cavity (Fig. 196).

The puncture is best effected under the inferior turbinate through the naso-antral wall. After previous cocainisation, a straight or slightly curved trochar and cannula may be used. After aspirating the pus with the syringe some normal saline solution should be injected into the antrum, and if the fluid from the nose is received into a basin it will be seen to contain flocculent pus.

If the diagnosis of antral suppuration is established by these means, attention should then be directed to the possibility of the frontal sinus and anterior ethmoidal cells being also involved.

Examination of the nose may show a streak of pus far forward in the middle meatus just under the anterior end of the turbinate.

If this is mopped away it may reappear in five to ten minutes without the position of the head being altered.

Transillumination is of little help owing to the varying thickness of the anterior wall, but X-ray examination will give most useful information both with regard to the possibility of thickening of the mucosa or of the existence of a fluid-level (Fig. 196). In some cases it may be possible to pass a suitably curved cannula up the frontonasal

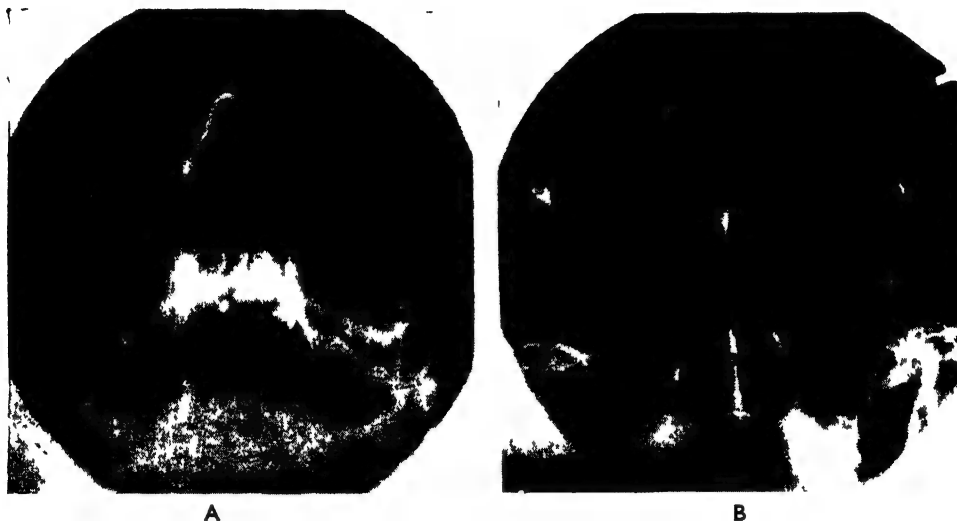


FIG. 196

Two X-ray prints showing A, the right antrum filled with fluid, and B, the frontal sinus, in which a fluid level is well defined.

duct and into the sinus, but often anatomical peculiarities make this impossible. If it can be achieved, gentle suction may reveal the presence of pus. The anterior ethmoidal cells are often involved in suppuration of the frontal sinuses or maxillary antrum, but they may be affected alone. There are two main types, the hyperplastic type with polypus formation and a profuse watery discharge, and the purulent type with a granular appearance of the mucosa and excessive crust-formation owing to the drying of the purulent discharge. After careful removal of secretions, the insertion of a long-bladed Killian speculum under the middle turbinate and the forcing of the turbinate away from the uncinate process may enable the bulla to be seen and the site of the infection revealed.

#### CHRONIC INFLAMMATION IN THE POSTERIOR GROUP OF SINUSES

This will show itself on anterior rhinoscopy by the presence of discharge in the olfactory sulcus, or, on posterior rhinoscopy, by discharge seen on the back of the septum, on the lateral nasal wall near the Eustachian tube, on the upper surface of the posterior end of the middle turbinate, or adhering to the roof of the nasopharynx. X-ray examination in the vertico-menta position may show blurring of the

posterior cells and sphenoid. Puncture of the sphenoid and posterior cells may be carried out after the middle turbinate has been forcibly pressed aside by the blades of a long speculum.

The *treatment* of chronic inflammation in the accessory sinuses depends upon (1) the establishment of free drainage and aeration, (2) the removal of the cause if possible, and (3) increasing the patient's resistance. Chronic sinusitis with pronounced obstructive lesions necessitates the removal of the obstructive lesions whether they be of septal, turbinal or other origin, so that the treatment will be essentially operative. Vaccine therapy is not justified if there is any obstruction to drainage, though it may be useful once this has been established. The same applies to short-wave diathermy.

**Maxillary Antrum.**—The establishment of drainage is best achieved by making a large opening through the naso-antral wall under the inferior turbinate. This may be done either under local or general anæsthesia. Subsequent irrigation through the opening may be necessary. It will generally bring about a cure of the condition. In some cases in which the antrum has been infected from a tooth socket or from a piece of root pushed up into it, the approach through the canine fossa under the upper lip (Caldwell-Luc operation) is advisable. This enables the cavity to be inspected, any foreign body removed, or polypoid mucous membrane to be dealt with. A counter-opening through the naso-antral wall into the nose provides permanent drainage.

**Frontal Sinus.**—When nasal obstruction is a marked feature on the affected side, relief of this, together with the removal of the anterior end of the middle turbinate, will relieve pressure on the infundibulum, and the passage of a sound will overcome obstruction at the fronto-nasal opening. It is not always easy or even possible to pass a sound into the sinus, and in such cases some abnormality of anatomical configuration is in all probability the reason. In these cases it will be necessary to remove the obstructing ethmoidal cells with a curette or biting forceps so as to allow a sound to pass into the sinus (Fig. 197). A large number of cases will be cured by this procedure, but it should be remembered that in many the anterior ethmoidal cells are simultaneously involved, and these must be removed if ascending infection of the frontal sinus is to be avoided. The majority of cases are cured by these intranasal methods, but in some the headache, tenderness, discharge and eye symptoms persist. Under these circumstances an external operation is advisable. The one that gives the most satisfactory results is Howarth's operation.

**Ethmoid Labyrinth.**—A large variety of operations has been devised for dealing with chronic inflammation in this region. In all cases some form of intranasal operation should be tried before resorting to the external approach. Partial or complete excision of the middle turbinate, with removal of polypi and exenteration of the anterior ethmoidal cells, is usually required, but occasionally the opening of the bulla ethmoidalis and the anterior cells under the middle turbinate will suffice. Complete exenteration of the whole lateral mass of the ethmoid by some such operation as Mosher's gives good results in advanced cases. An external approach may be necessary when there is an

external fistula or an orbital abscess, or when intranasal operation has failed.

**The Sphenoidal Sinus** may be approached intranasally after removal of the middle turbinate, or it may be dealt with by making a sub-mucous resection of the septum and following the septum backwards and upwards until the anterior face of the sphenoid comes into view. This transseptal operation gives excellent results. The transantral approach is also useful in some cases. If an external operation on the

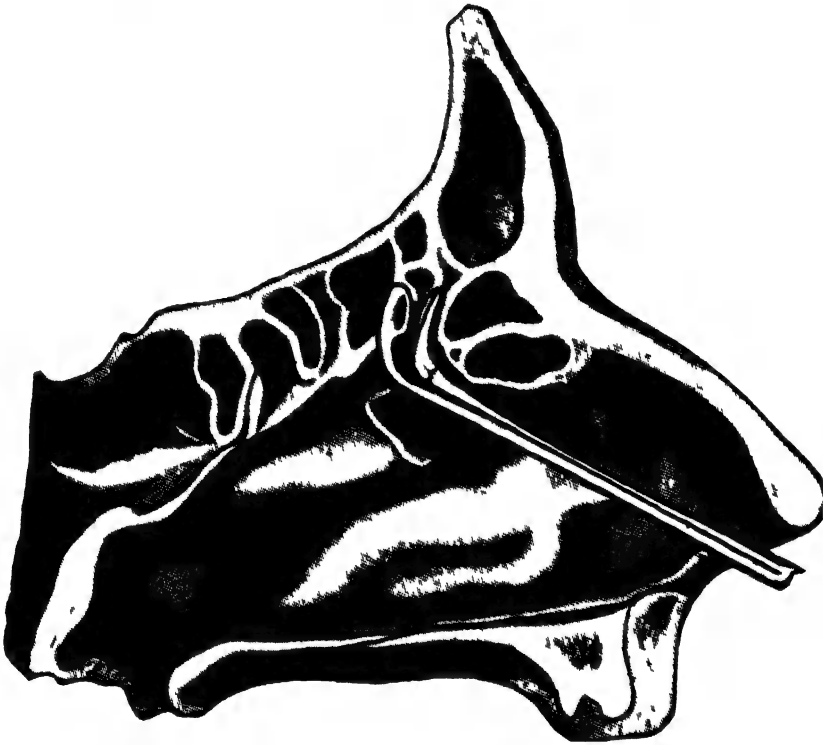


FIG. 197

A drawing illustrating the disposition of cells round the fronto-nasal duct with the fronto-ethmoidal cells mounting up into the floor of the frontal sinus. Rongeur forceps are shown nibbling these cells away

ethmoid labyrinth is being performed it is easy to extend the removal backwards to include the posterior ethmoidal cells and the anterior wall of the sphenoid.

**MUCOCELE** is rather an unusual phenomenon that is occasionally found in relation to the sinuses. It is characterised by a slowly increasing swelling that usually presents externally and gives the impression of a new growth rather than of an inflammatory swelling. It is most frequently seen in the region of the inner canthus and arises from the ethmoid or frontal sinus. When of large size the swelling may displace the eye outwards and downwards. The contents of the mucocele are usually a thick glairy, tenacious fluid, often containing cholesterin crystals. The origin of mucocele is doubtful, but it is usually due to a low-grade inflammatory condition which has resulted in

partial closure of the ostium or, occasionally, to an injury in the region of the ostium causing partial blockage. As the cyst-like swelling increases in size considerable erosion and absorption of neighbouring bony structures may take place, so that in advanced cases the ethmoid labyrinth may be converted into one large cavity continuous with the frontal sinus and a large area of the dura exposed.

*Treatment* consists in surgical approach by the external route and the establishment of a large track between the mucocele and the nasal cavity.

### COMPLICATIONS OF SINUS DISEASE

These may be orbital, ocular, intracranial or general. Orbital cellulitis and abscess are not uncommonly seen in children as a result of ethmoidal disease, the infection passing through the os planum into the orbit. It is seen more rarely in adults, and demands an external incision. Optic neuritis and retrobulbar neuritis may be due to disease of the ethmoid and sphenoid, whilst occasionally loss of vision may be due to septic absorption from infected sinuses. Extradural and cerebral abscess, meningitis and cavernous sinus thrombosis may be caused by extension of suppuration from the frontal sinus or ethmoidal labyrinth, whilst osteomyelitis of the frontal bones from infection of the diploe or of the superior maxilla is a dreaded sequela.

### SINUS INVOLVEMENT IN CHILDREN

Sinus infection in children is often overlooked, but it is undoubtedly more common than was formerly allowed. Routine examination of children who have infected tonsils and adenoids reveals the fact that a great many have infected maxillary antra. The large majority clear up satisfactorily when the tonsils and adenoids are removed; but in some, puncture of the antrum or the establishment of a small intranasal opening may be necessary.

### NEW GROWTHS

Growths of the nose may be either simple or malignant. Of the **simple** ones, papilloma arising from the mucous membrane is very rare, but warty growths are not uncommonly seen on the skin lining the vestibule. Hæmangioma arises usually from the septum, and is often responsible for violent bleeding. It may also grow from the ethmoid labyrinth. In this situation it tends to recur locally if not thoroughly removed. Chondroma is a rare manifestation, but may originate in the septum, whilst osteoma is sometimes seen on the lateral wall, and in some cases in the sinuses themselves.

**Malignant** neoplasms are, unfortunately, fairly common. They usually start in some portion of the ethmoid labyrinth and spread downwards into the antrum, upwards into the frontal sinus, or inwards into the nasal cavity. In many cases they may involve a considerable area before their presence is suspected, but unilateral bleeding in elderly people is often significant of their presence.

*Treatment* is best carried out by incision under the upper lip, the mask of the face being lifted off the underlying bone structures. The tumour may be removed by diathermy, and it is remarkable what extensive growths can be charred away between two electrodes and removed piecemeal. Often one has to go up to the dura and back to the nasopharynx. In dealing with antral tumours the extension is often backwards to the pterygomaxillary fossa, and if this portion cannot be entirely removed by electrosurgery it is advisable to pack a dozen radium needles of 1 mg. each into the cavity for four days.

The operation of lateral rhinotomy (Moure) which used to be employed as a method of approach for these growths has been largely given up in favour of the procedure outlined above.

Malignant growths of the sinuses do not appear to be of a severe degree of virulence, so that a high percentage of cures may be looked for.

## ADENOIDS

Hyperplasia of the lymphoid tissue, which is normally situated on the roof of the nasopharynx (the pharyngeal or Luschka's tonsil), is commonly called adenoids. This hyperplasia frequently follows one of the acute specific fevers, but undoubtedly occurs apart from infection, and may perhaps be regarded as an exaggerated response of the defensive mechanism of the body in the process of acquiring immunity. The underlying cause is, however, very obscure. Some observers think it is due to improper feeding (excess of sugar and starchy foods), whilst others regard it as due to an essential vitamin deficiency or predisposed to by a general lack of nutrition.

Adenoid vegetations are met with in early life, being most commonly seen between the ages of four and fourteen, but they occur in infants, and may also be found in adults.

Adenoids are usually situated on the vault of the nasopharynx and may project downwards over the back of the septum, thus partially blocking the choanæ, or they may extend down the posterior pharyngeal wall and be seen without the aid of a post-nasal mirror below the soft palate. In some cases there are extensions round the mouth of the Eustachian tube.

Adenoids are composed of masses of lymphoid tissue, usually disposed in vertical ridges. They are not encapsuled in any way, which accounts for their varying shape and situation.

The *symptoms* vary with the degree of hyperplasia. In advanced and long-standing cases the picture of chronic nasal obstruction is produced. There is mouth-breathing by day and snoring at night. The nares become pinched, the palate high and arched, the jaw underhung and the mouth always open. Mentality is dulled, the expression is vacant and the so-called adenoid facies is produced.

Many cases are seen long before such marked changes occur, and come under observation on account of ear troubles, nasal symptoms or general reflex disturbances.

Those with ear symptoms are brought complaining of deafness,



which is usually due to Eustachian-tube obstruction or aural discharge. The deafness often varies with the weather and is usually worse with a cold. The tympanic membranes are retracted, and occasionally there is a perforation through which a sticky mucopus exudes.

The nasal symptoms that are complained of are snoring at night, mouth-breathing during the day, recurrent attacks of cold in the head, a tendency to choking or snuffling whilst eating and a constant mucoid nasal discharge.

The general symptoms that are most usually noticed are listlessness, inability to concentrate, and general apathy and dullness. These are often associated with restless sleep, night terrors, and nocturnal enuresis, whilst the physical development is poor and the chest flat and retracted owing to imperfect expansion of the lungs.

*Diagnosis.*—The diagnosis is seldom in doubt, and is often made from the symptoms, but examination with the post-nasal mirror will reveal the adenoid mass and show its extent. Children tolerate examination with the mirror very well indeed, and it should seldom be necessary to make a digital palpation of the nasopharynx.

*Treatment.*—The fact that adenoids are present does not of necessity mean that they should be removed, and when they are not causing any symptoms they should be left alone, as otherwise the defensive mechanism of the patient may be upset. Proper breathing exercises and the instillation of 2 per cent. argyrol drops will often suffice for mild cases, but when the symptoms are marked no time should be lost in removing the growths.

When operation is decided on, it is best done under general anæsthesia with the child lying on its back and the head extended. The mouth is opened with a Doyen's or Sydenham's gag, and some form of guarded curette is usually employed. This is introduced behind the soft palate and pushed firmly backwards against the vault of the nasopharynx and then drawn downwards with a sweeping movement along the posterior pharyngeal wall, a certain amount of pressure being maintained at the same time. The main mass of adenoids that lie centrally and are caught in the hooks attached to the curette come away in one piece. Small outlying masses may be removed with a ring knife or Löwenberg's forceps.

Some surgeons prefer to use a La Force adenotome for the operation, but this instrument is not suited to cases in which there is any depression in the nasopharyngeal wall due to an abnormally projecting vertebral body.

After the operation the child should be turned on its face and the bleeding stops in a few moments. Rest in bed is essential for a few days if aural or other complications are to be avoided.

WALTER HOWARTH.



## CHAPTER XXII

### THE PHARYNX AND ŒSOPHAGUS

**S**URGICAL *Anatomy of the Pharynx and Œsophagus.*—The pharynx has a muscular, a fibrous and a mucous coat. The muscular coat is composed of the inferior, middle and superior constrictors with slips from the stylopharyngeus and palatopharyngeus. These flat muscles are inserted posteriorly into a median raphe attached above to the basilar process of the occipital bone. The inferior constrictor overlaps the middle and the middle overlaps the superior. The lower fibres of the inferior constrictor, arising from the cricoid are almost horizontal and are continuous with the muscular coat of the Œsophagus, while the upper fibres arising from the thyroid cartilage ascend obliquely over the lower part of the middle constrictor towards the median raphe. The lower portion is sometimes described as the cricopharyngeus, and it is in the interval between the lower horizontal and upper oblique portions that a pharyngeal pouch herniates through the wall of the pharynx.

The pharyngeal aponeurosis or fibrous coat is dense where the muscular coat is absent, *i.e.*, in the intervals between the origins of the constrictor muscles.

The mucous coat, containing mucous glands and lymphoid follicles, is continuous with that of the upper air and food passages.

The isthmus of the fauces, by which the mouth opens into the pharynx, is bounded above by the soft palate and laterally by the anterior and posterior pillars formed by the palatoglossus and palatopharyngeus. Between these pillars which represent parts of the second and third branchial arches lie the tonsils. The supratonsillar fossa at the upper part of the tonsil represents the remnant of the recess in the wall of which the tonsil is developed. The outlet is narrowed by a fold of mucous membrane, the plica semilunaris stretching across the angle at the junction of the pillars with the soft palate. The supratonsillar fossa or crypta magna is thus part of the tonsil. Another fold, the plica triangularis, stretches backwards from the anterior pillar and blends with the surface of the tonsils, but has a free crescentic margin directed inwards and backwards, and so forms a kind of sling for the tonsil.

On its deep surface the tonsil is not in direct relation to the superior constrictor of the pharynx, but is separated from it by the palatopharyngeus. This muscle spreads out behind and sends a slip, the tonsillopharyngeus, to be directly attached to the tonsil. In a tonsil removed by a clean dissection the remnant of this attachment may be seen dividing the tonsil into upper and lower segments.

The lymphoid tissue on the back of the tongue forms the lingual tonsil. On either side are the palatine or faucial tonsils and above lies the adenoid tissue of the pharyngeal tonsil in the nasopharynx, with outlying extensions at the mouth of the Eustachian tube. There is a further connecting band of lymphoid tissue, often enlarged behind the posterior pillar of the fauces, and other outlying follicles in the pharynx. This forms Waldeyer's ring,

which drains into a wider lymphatic area than do the tonsils alone, but all this lymphoid tissue is confined to the neck.

At the lower border of the cricoid cartilage opposite the 6th cervical vertebra the pharynx becomes continuous with the œsophagus, which extends for 9 or 10 in. down to the 10th or 11th dorsal vertebra. The abdominal portion is about 1 in. in length. The distance from the teeth to the cardiac orifice is about 16 in. (40 cm.), but often rather more. The œsophagus is narrow (1) at its upper opening, (2) where it is crossed by the aorta and the left bronchus, and (3) at its passage through the diaphragm. It is at these situations that foreign bodies are apt to lodge and malignant disease to develop.

*Examination of the Pharynx and Œsophagus.*—Only a reflecting mirror or headlight and a tongue depressor are necessary to examine the oropharynx. The condition of the palate, teeth, gums, tongue and floor of the mouth should be noticed at the same time, and also the movements of the soft palate. To expose the tonsils and detect pus concealed in the crypts, a second tongue depressor of small size held in the other hand is useful. The nasopharynx should be surveyed with a small mirror. To examine the laryngopharynx the use of the laryngoscope is essential (see p. 445).

The hypopharynx, the lowest part of which lies behind the larynx and ends at the lower border of the cricoid, can only be seen by endoscopy. Hypopharyngoscopy effected by pulling the larynx forward with a curved sound is of little use.

Examination of the œsophagus is now confined to radiography and œsophagoscopy. Auscultation gives no reliable information and there is no longer any reason for the use of bougies in diagnosis. They should only be used for dilatation when introduced under vision through the œsophagoscope. For radiography the patient should swallow a paste consisting of 2 oz. of subcarbonate of bismuth or barium sulphate mixed with milk and breadcrumbs. This will show on the screen or in a radiogram as a dark shadow the outline of a dilatation, stricture or pouch.

The cardinal symptom of œsophageal disorders is dysphagia, usually of gradual onset and, unless the cause is revealed by examination of the mouth and pharynx, which may show some obvious ulceration, the œsophagoscope should always be employed after preliminary radiography. The presence of an aneurysm compressing the œsophagus should always be excluded by physical examination and radiography before passing the œsophagoscope. The tubes are similar to those used for bronchoscopy, but are larger, longer and require no lateral fenestrations (see p. 447).

### FOREIGN BODIES IN THE PHARYNX

Large foreign bodies in the laryngopharynx, such as a tooth plate, a bone or a coin, may lodge behind the larynx in the region of the cricoid and cause only pain and dysphagia until removed with laryngeal forceps, but a large piece of meat impacted over the entrance to the larynx will cause sudden asphyxia. Unless the foreign body is hooked out with the finger or laryngotomy is performed at once the accident is immediately fatal. Consequently such specimens are common in museums. There is in the museum of the Middlesex Hospital a specimen which displays a billiard ball impacted at the entrance to the larynx.

Small foreign bodies, especially pins and fish bones, may stick in the fauces, the back of the tongue, the pyriform fossa or the tonsils. Pain aggravated by swallowing is the chief symptom, but is not a

reliable guide, as the patient may refer unpleasant sensations to a point at some distance from the actual situation of the foreign body.

The whole pharynx, including the nasopharynx and the larynx, should therefore be searched thoroughly after spraying with a weak solution of cocaine, before concluding that no foreign body is present. This is often the case as the disagreeable sensation caused by a slight abrasion persists after the foreign body has passed onwards. Great care, however, is required not to overlook a fish bone, which is difficult to see and may be imbedded with only a small part projecting. Slight hæmorrhage or a small hæmatoma sometimes gives a clue. It is important to remove such foreign bodies as soon as possible, as an acute inflammation of the pharynx may be caused. If the wall of the pharynx is perforated surgical emphysema may result, and still worse, cellulitis of the neck may cause hæmorrhage from the great vessels, mediastinitis or even spinal meningitis. When located the foreign body must be removed, according to its situation, with straight forceps or Mackenzie's angular laryngeal forceps guided by a mirror. Endoscopic removal is sometimes easier for foreign bodies situated low down.

### FOREIGN BODIES IN THE ŒSOPHAGUS

A variety of foreign bodies impacted in the Œsophagus is recorded, but the common ones are, in adults, tooth plates or bones (Fig. 198), and in children, coins (especially halfpennies) or safety-pins. Sometimes pieces of meat or fruit stones, which normally would pass easily, become impacted above a stricture, so that the onset of dysphagia in cancer of the Œsophagus is sometimes sudden.

The majority of foreign bodies stick in the upper third of the Œsophagus, and a coin is nearly always seen by X-rays opposite the top of the sternum lying in the coronal plane (Fig. 199). If the coin is lying with its diameter anteroposteriorly it is probably in the trachea. A foreign body occasionally remains for long periods in the Œsophagus without producing dysphagia or doing much harm, but it is likely at some time to produce ulceration followed by mediastinitis, perforation of the trachea or of a large vessel. Foreign bodies should therefore be removed without unnecessary delay, but not as a rule without localisation by X-rays, and no attempt should be made without suitable instruments. It is better to leave a coin in position a little



FIG. 198

A specimen showing a "merry thought" impacted in the Œsophagus.

longer than to remove it with a coin catcher, which, though it succeeds, may cause a serious accident. A bougie should never be used.

The diagnosis is usually obvious from the history and X-ray examination, but a non-opaque foreign body can sometimes be revealed by making the patient swallow a gelatine capsule containing bismuth, which may be arrested at the site of obstruction.

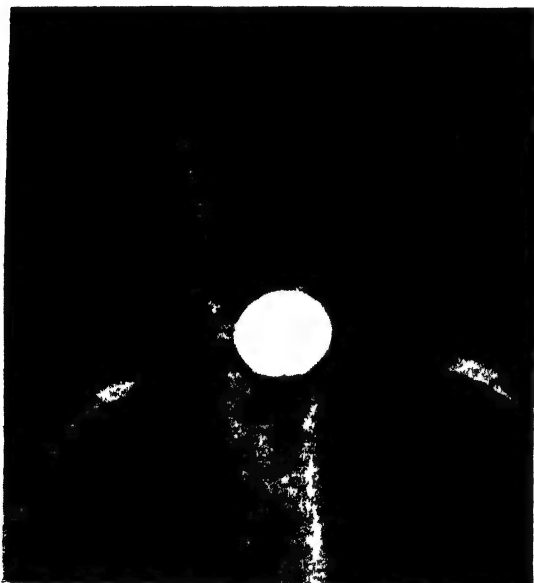


FIG. 199

An X-ray photograph of a halfpenny in the common situation in the œsophagus, i.e., opposite the top of the sternum.

In removing the foreign body by the œsophagoscope, care is to be observed that the beak of the instrument does not shoot past an object lying high up in the hypopharynx or gullet. A small tube also may easily pass an object concealed in the folds of mucous membrane, which would be opened out by a larger tube. Metallic objects may be so changed in appearance that they are not easily recognised, or may be embedded in an inflammatory mass.

A foreign body, such as a rabbit bone, lying across the lumen should be seized by the tip and not near the middle, so that it swings

into the long axis of the gullet. The same applies to a tooth plate, but if this is large it is sometimes necessary to divide it with endoscopic shears, as it is an object very likely to lacerate the wall of the œsophagus. Special instruments have been designed for closing safety-pins lying open with the point upwards, but the best method is to protect the point with the end of the œsophagoscope and grasp the pin by the clasp. External œsophagotomy is very rarely necessary.

## DISEASES OF THE PHARYNX

### ACUTE PHARYNGITIS

Simple catarrhal pharyngitis is part of an ordinary coryza, or of one of the infectious fevers, or may accompany an attack of acute tonsillitis. The mucous membrane shows only slight inflammatory swelling and congestion of a transient character. Streptococcal infection of the pharynx, sometimes called acute septic pharyngitis, presents far more acute and serious symptoms. The mucous membrane of the pharynx becomes swollen, purple and glazed with great enlargement of the uvula, and in severe cases sloughing of the tonsils and

gangrene of the uvula follow. The œdema may spread down to the aryepiglottic folds and cause dyspnoea, or, if the infection has its origin in dental sepsis, it may extend to the neck and cause a diffuse brawny swelling (**Ludwig's Angina**). The patient is always gravely ill and may be delirious, with a swinging temperature, showing remissions accompanied by sweating. The breath is foetid and dysphagia is severe. The pulse is bounding and rapid but soon becomes feeble, and the patient may succumb rapidly from exhaustion, or later from mediastinitis or pleurisy. The urine should always be examined for albumin and sugar, and care is to be taken in making the diagnosis that the condition of the pharynx is not masking an attack of scarlet fever or diphtheria.

*Treatment.*—Antiscarlatinal serum is the most useful general remedy, though occasionally other organisms than the streptococcus hæmolyticus are found. If septicæmia ensues, prontosil or rubiazol is invaluable. Hot applications to the neck and spraying the pharynx with eusol can be recommended.

In Ludwig's angina incisions in the neck may be required, but they seldom reach a collection of pus. It is stated by Clermont that the infection begins in a remnant of thyroglossal tissue, and that a deep midline incision from the chin to the body of the hyoid bone will always reach pus in the base of the tongue. Tracheotomy is occasionally required for the relief of dyspnoea.

Streptococcal infection may produce a membranous pharyngitis indistinguishable from diphtheria, and occasionally the appearance of Vincent's angina is membranous rather than ulcerative.

In agranulocytic angina severe tonsillitis and pharyngitis going on to gangrene and sloughing are usually rapidly fatal. Blood examination shows a leucopenia with a relative lymphocytosis. The application of X-rays to the long bones and injections of pentnucleotide to produce granulocytopoiesis are recommended. Blood transfusion is only temporarily effective.

In leukæmia also a similar condition in the pharynx may be encountered. Therefore bacteriological investigation should never be omitted, and a blood count is often essential to establish a correct diagnosis.

**Vincent's Angina** is common amongst soldiers and relatively uncommon in civilian life. Ulceration with membranous exudation attacks the tonsil, but this is often, if not invariably, secondary to a gingivitis around the teeth. The infection is caused by a symbiosis between the bacillus of Vincent and a spirillum. The bacillus is fusiform, 6 to 8  $\mu$  in length and 1  $\mu$  in breadth. It may contain vacuoles, be arranged in pairs or radiating bundles and be mobile. The organism does not stain by the method of Gram but by the ordinary basic stains, and can be cultivated in broth to which ascitic fluid has been added. The spirillum is long, sinuous and very mobile, and does not stain so readily as the bacillus. It cannot be cultivated.

The ulcer on the tonsil is irregular with sharply cut edges, the floor being covered with a yellow membranous slough. The ulceration may extend widely over the pharynx, and in very severe cases cellulitis

may spread down the neck to the mediastinum and pericardium. With treatment the patient usually recovers in ten days, but the peridontal disease needs attention. Mandl's paint (pigmentum iodi) should be applied to the ulcer. A good application which must not be swallowed is :

Vin. Ipecac. . . . .	℥ <sub>xxx</sub> .
Glycerin . . . . .	ʒi.
Liq. arsenicalis . . . . .	ad. ʒi.

A saturated solution of antimony and potassium tartrate may be substituted for the liq. arsenicalis. Another treatment is to paint the lesions with an alkaline solution of salvarsan, and occasionally in resistant cases this should be injected intravenously.

### TONSILLITIS

Acute tonsillitis is classified as catarrhal, lacunar or parenchymatous according to its degree of severity. In the common lacunar form, often called follicular, the crypts become filled with desquamated epithelium, fibrin, pus cells and organisms, forming white or grey dots at the mouths. In the parenchymatous form the whole tonsil and the adjacent pharynx become swollen and infiltrated, and an abscess may form in the substance of the tonsil.

The disease usually occurs in young adults, and sometimes in children. In children it may precede an attack of acute rheumatism, so that removal of the tonsils slightly diminishes the frequency of primary attacks, but the operation has no influence in preventing recurrent attacks nor does it influence the incidence of endocarditis.

Acute inflammation is particularly liable to occur in the remnants of tonsils which have been partially removed, and in tonsils which are the site of chronic inflammation or suppuration. The specific fevers, especially scarlet fever, often begin with an acute attack of parenchymatous tonsillitis. The commonest causal organism by far is the streptococcus pyogenes, but varieties of pneumococci and staphylococci are also found either pure or in mixed infections. In the epidemic streptococcal form the contagion may be carried by milk. The initial symptoms are malaise and anorexia, sometimes with a chill or even a definite rigor. The temperature rises to 102° or 104° F. with corresponding increase in the rate of the pulse, which is full and bounding. There is pain in the throat radiating to the ears and increasing difficulty in swallowing, with much salivation.

The condition must be distinguished from diphtheria and Vincent's angina by bacteriological examination. Parenchymatous tonsillitis may indicate the onset of scarlet fever before the appearance of the rash. Mild cases must be distinguished from secondary syphilis. The illness usually lasts from four to eight days.

The faucial type of glandular fever resembles severe tonsillitis. The cervical glands enlarge, and there is both a relative and absolute lymphocytosis. The spleen may be enlarged. The illness may last several weeks, but the prognosis is good.

*Treatment.*—At the onset a mercurial purge should be administered

followed by senna or cascara. Aspirin is useful to relieve the pain, or a mixture containing sodium salicylate may be prescribed.

Locally a paint containing 2 per cent. each of  $\beta$ -eucaine and formalin in glycerin is useful, or in very severe infections with much exudation the throat may be sprayed with eusol. Gargles are useless and increase the pain, but a compress to the neck, either hot or cold, often gives relief. Junket, ice-cream, custard and thick soups are foods easily swallowed. The dysphagia may be relieved by an attendant standing behind the patient with the palms applied behind and below the angle of the jaw on either side. Firm pressure is made just as the patient swallows. If the disease is recurrent, after eliminating such causes as dental sepsis or external sources of infection, the tonsils should be removed, but not until after an interval of six weeks.

### QUINSY

The formation of a peritonsillar abscess, or quinsy (Fig. 200), which is usually unilateral, is shown by increasing œdema of the uvula and the appearance of a tense swelling on one side of the soft palate, which bulges downwards and forwards. The abscess should not be allowed to burst through the soft palate or into the tonsillar fossa but should be opened with sinus forceps at its most prominent point, after painting with 10 per cent. cocaine. A yellow spot often indicates the point at which the pus may be most easily reached, but if this has not appeared, the centre of a line between the base of the uvula and the upper wisdom tooth may be chosen. A peritonsillar abscess may occasionally be opened through the supratonsillar fossa.

Much less commonly a quinsy presents behind the lower half of the tonsil. In this case there is some danger that the pus may track along the carotid sheath, if the opening for drainage is not placed suitably, and it may not be possible to reach the pus without removing the tonsil. This provides very free drainage and healing is rapid and simple.

A rare but grave complication of quinsy is hæmorrhage from erosion of one of the large vessels in the immediate neighbourhood.

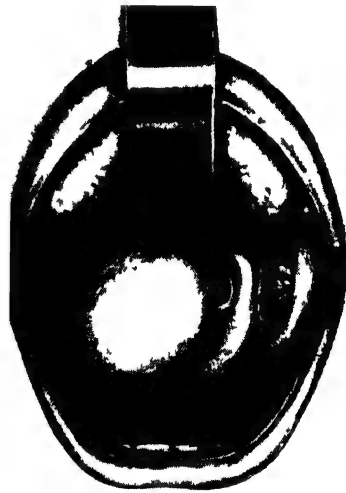


FIG. 200

A peritonsillar abscess.

### RETROPHARYNGEAL ABSCESS

This is caused by suppuration in the glands of Henle situated between the posterior pharyngeal wall and the prevertebral aponeurosis. It



usually occurs in children, often as a complication of one of the infectious fevers. On rare occasions the pus tracks down from the mastoid process. The child suffers from dysphagia and dyspnoea due to the tongue falling back against the swelling, which is not usually in the middle line but lies behind one tonsil. Palpation as well as inspection is important in diagnosis. If unrelieved the abscess may burst and flood the larynx and trachea with pus or track down into the posterior mediastinum. The child should be held on its side with the head down and the face turned rather downwards. The abscess may then be opened without an anæsthetic with a guarded knife or sinus forceps.

A chronic form of retropharyngeal abscess arises in children from tuberculous caries of the cervical spine, producing the same symptoms, of which the earliest may be nasal obstruction. To avoid septic infection such an abscess should be opened through an incision behind the sternomastoid. The posterior edge of the muscle is exposed, the carotid sheath with its contents is drawn forward and the position of the transverse process defined before the abscess is opened. Large acute abscesses are better opened by this route than by the internal.

### CHRONIC PHARYNGITIS

This shows itself by a congestion of the mucous membrane over the soft palate, fauces and uvula, which may be elongated. In the so-called granular pharyngitis hypertrophied patches of lymphoid tissue occur on the posterior pharyngeal wall. In pharyngitis sicca the mucosa becomes dry and atrophic. The symptoms are dryness, tickling and cough. Local causes are chronic tonsillitis, nasal obstruction, or the discharge from an accessory sinus of the nose. Excessive smoking, the abuse of alcohol and faulty voice production in professional speakers are important factors. General conditions such as gout, diabetes, arteriosclerosis and cardiac disease are more important than local causes.

Rheumatic pharyngitis is particularly painful. Pathological conditions in the nose should be corrected, and a simple gargle or the astringent, *Trochisci Krameriaë*, may be given. Disorders of digestion require attention, and patients may be sent for spa treatment to Ems, Luchon or Aix-les-Bains.

In the **Plummer-Vinson** syndrome, which occurs only in middle-aged women, dysphagia, accompanied by achlorhydric anæmia, is the cardinal feature. The skin is dry and there are cracks at the corner of the mouth. The dysphagia is due to chronic glossitis and atrophy of the mucous membrane of the pharynx and œsophagus, where chronic inflammatory and degenerative changes in the plexus of Auerbach have been found. Blood examination shows microcytic anæmia with low colour index, and a few normoblasts but no megaloblasts. The administration of iron in large doses is indicated.

### CHRONIC TONSILLITIS

Enlargement or hypertrophy of the tonsils is sometimes regarded as synonymous with chronic inflammation, but, in fact, size is no



criterion of this, and in many small children some hypertrophy of the tonsils appears to be a physiological necessity rather than a pathological reaction.

The tonsils may be large, but often, especially in adults, are small, flat and concealed by the pillars of the fauces. There is a chronic infection of a crypt, especially of the crypta magna or supratonsillar fossa from which pus may be expressed. A chronic abscess may form in the substance of the tonsil, usually in a deep situation near the capsule.

Collections of epithelial scales forming cheesy masses in the crypts are often troublesome, but are not necessarily an indication of chronic infection. The principal criterion is a history of recurring attacks of acute tonsillitis or of quinsy, and the lymphatic glands in the neck just below and behind the angle of the jaw are generally enlarged. A deep crimson band of congestion may be seen along the anterior faucial pillar. Such tonsils are of importance as sites of focal sepsis causing arthritis, muscular rheumatism or nephritis.

Indigestion and retching are frequent *symptoms*. The breath may be foul and the patient notice a bad taste in the mouth. There is often a reflex cough with general symptoms of fatigue and anæmia from toxic absorption.

*Treatment.*—Paints containing iodine or resorcin may be tried in adults and blocked crypts slit up with a small knife or the electric cautery. Unless there is some general contraindication it is better to remove the tonsils, especially if there is a history of recurrent acute tonsillitis or quinsy or any pronounced cervical adenitis. In small children simple hypertrophy or a single attack of tonsillitis is not sufficient reason for removal of the tonsils, as the need for lymphoid tissue at that age to develop immunity and defensive reactions leads to recurrence and hypertrophy elsewhere, especially of the adenoid tissue in the nasopharynx.

*Removal of the Tonsils.*—In children up to the age of 15 years it is feasible to remove tonsils entire by the guillotine; this is possible also in many adults, but as a rule, in order to ensure complete removal, dissection is preferable. The choice of operation in individual cases is determined by the practice of the operator, but when there is a history of quinsy the line of cleavage around the capsule is difficult to define and the guillotine operation may be impossible. Whichever method is employed the whole tonsil should be removed, especially the lower pole from which the tonsil may reproduce itself. In exceptional cases, even when tonsillectomy has been complete, the tonsil may be reproduced from the lingual portion and provide a target for the critical.

In children a general anæsthetic is required, but ethyl chloride is sufficient for the guillotine operation. In adults a longer general anæsthetic is necessary. Intratracheal gas and oxygen is often used, but is not suitable for this purpose as it increases the bleeding. Local anæsthesia is sometimes employed. A light application of 5 per cent. solution of cocaine is made to the fauces, but is not essential. Novocain with adrenalin is then injected into the fauces around each tonsil in five spots, one above and two each in front and behind the tonsil. The risk of post-operative hæmorrhage and of blood escaping through the larynx down the trachea seems to be greater than with general anæsthesia.

When the guillotine is used, the patient lies supine with the shoulders a little raised and the head extended. The mouth is opened with a Doyen gag placed between the front teeth, but not wide enough to stretch the anterior faucial pillars. The ring of the guillotine held in the right hand is passed under the lower pole of the right tonsil with the handle pointing to the left of the patient. The handle is then depressed so that the tonsil is levered on to the alveolar eminence at the posterior end of the mylohyoid ridge, which forces it through the ring (Sluder). To complete this movement the forefinger of the left hand is applied to the anterior faucial pillar (Whillis), and the whole tonsil can be felt to slip through the ring. The blade is then pushed home by the thumb of the right hand. Care is to be taken that the blade passes between the anterior pillar and the tonsil so that the edge is not nipped. The tonsil is then removed by pronating the hand. The left tonsil is removed by changing the guillotine to the left hand or by standing behind the head of the patient. By this method the tonsil with its supra-tonsillar fossa is removed complete in the capsule, which is often everted in the process. Two sizes of guillotine should be available.

In removal by dissection the patient may lie supine with the head either dropping far back or tilted over to the right side. If local anæsthesia is used the patient sits in a chair and depresses the tongue himself.

Under general anæsthesia the mouth is opened with a Doyen or Sydenham gag, though many operators prefer to use the Davis gag, which gives a good view of the tonsils by forcing down the base of the tongue with the patient lying on the back, but the mouth cannot be fully opened and excessive pressure is often made on the front of the neck. The tongue is drawn forwards with a tongue clip or silkworm gut passed through the tip. The reflection of mucous membrane between the anterior pillar and the tonsil is incised or torn through with scissors or long-bladed dissecting forceps. Suitable forceps must be used to hold the tonsil and draw it out of its bed. The dissection is best done by teasing out the tonsil with the long-bladed dissecting forceps, so that the vessels are torn across and retract. There is then very little bleeding and ligatures are rarely required. The tonsil is thus shelled out of its bed, but it often remains attached by the lower pole to the lingual tonsil at the base of the tongue from which it has to be divided with scissors.

Careful sponging or the use of an electric suction pump is necessary to keep the field of operation clear from blood. The only artery likely to bleed persistently is the tonsillar branch of the facial, which enters the tonsil about the middle of its bed on the posterior pillar. If it does not retract it should be picked up with long forceps and underrun with a silk ligature on a Reverdin needle. Bleeding may continue after the guillotine operation from a button-holed vein which cannot retract. If there is a persistent oozing of blood or post-operative hæmorrhage, deep silk ligatures on a Reverdin needle should be passed through both pillars and tied. The stitches should be removed the following day and not allowed to cut out.

If the mucous membrane covering the edge of the anterior pillar is kept intact there is usually not much post-operative pain, but this varies and can be controlled by Euphagin or aspirin applied locally in a gargle. Adults should receive an injection of morphia on the night following the operation.

### KERATOSIS

Small, white, adherent excrescences are sometimes seen scattered over the tonsils. These were formerly thought to be caused by the *leptothrix buccalis*, which is sometimes present, and the condition

was called pharyngomycosis. True mycosis does occur but is extremely rare. These sickle-shaped projections are formed by an abnormal proliferation of highly keratinised epithelial cells. They occur not only on the faucial tonsils but also on the lingual tonsil and sometimes on the soft palate. Unless this distribution is observed, the appearance can easily be mistaken for lacunar tonsillitis, but there are no symptoms beyond slight discomfort or roughness in the throat. The patient is usually a young female.

No *treatment* is of any avail, but the condition disappears spontaneously.

### TUBERCULOSIS OF THE PHARYNX

Apart from tuberculous deposits in the tonsils tuberculosis of the pharynx is nearly always, but not invariably, secondary to pulmonary tuberculosis. It occurs much less frequently than laryngeal tuberculosis and usually heralds a rapidly fatal termination. Miliary tubercles become deposited in the mucous membrane and produce patches of ulceration. Single discrete tuberculous ulcers are much less common. This form of ulceration is very painful and causes much dysphagia. The best application for the relief of pain is orthoform powder if available. Applications of cocaine may be necessary to allow the patient to eat.

**Lupus** causes a more chronic form of ulceration, which may heal in one place, while it spreads in another with the formation of characteristic nodules. The contraction of the thin scar tissue causes much deformity of the soft palate and fauces. Sometimes the posterior wall of the pharynx only is affected. General *treatment* with arsenic and cod-liver oil is important, and local treatment with light from a Kromayer lamp may be effective.

### SYPHILIS OF THE PHARYNX

The tonsil is said to be the commonest site of **extragenital chancre**. It appears as an indolent ulcer with enlargement of the glands at the angle of the jaw, but the general inflammation of the tonsil obscures the classical characters of the chancre, which may be concealed. The unilateral situation distinguishes it from tonsillitis, but if the possibility of the correct diagnosis is forgotten it may be mistaken for carcinoma, Vincent's angina or a gumma. The examination of a scraping for the *treponema pallidum* and before long the appearance of a secondary rash will settle the diagnosis. Hot mouth washes and painting the ulcer with a 20 per cent. solution of nitrate of silver are the only local measures indicated.

**Secondary Syphilis** shows itself by symmetrical crescents of erythema on the anterior pillars and velum palati. The tonsils are often enlarged at the same time. Mucous patches appear in any part of the pharynx. They are round or oval, slightly raised and surrounded by a narrow inflammatory areola. The surface is covered by a thin opalescent membrane. They produce a sore throat and dysphagia.

**Tertiary Syphilis** produces severe effects in the pharynx. A diffuse

or circumscribed gumma may break down and produce a superficial serpiginous ulcer, which is characteristic, or deep ulceration with sharply cut edges and a yellow slough covering the base. This latter causes great destruction and healing is followed by gross scarring and deformity, especially on the posterior pharyngeal wall and soft palate, which may be perforated or become a mass of cicatricial tissue. Adhesion of the palate to the posterior pharyngeal wall may narrow or entirely obliterate the opening between the oropharynx and nasopharynx.

Dysphagia is the chief *symptom*, but perforation of the palate produces also regurgitation through the nose and a nasal voice, while adhesions cause partial or complete nasal obstruction, sometimes with deafness. General *treatment* is of great importance, but plastic operations for the restoration of a passage between the oropharynx and nasopharynx or attempts at dilatation give only discouraging results.

### NERVOUS AFFECTIONS OF THE PHARYNX

In **globus hystericus** there is no visible change. The sensation of a lump rising in the throat is probably due to spasmodic contraction of the constrictor muscles.

**Nystagmus** of the pharynx may be due to disease of the central nervous system such as tabes dorsalis, or to some local cause of reflex irritation. The soft palate moves up and down as often as sixty times a minute, sometimes with a clicking sound. The vocal cords also are sometimes implicated. The lesion is said to be in the olive.

**Paralysis** of the pharynx may be caused by central nervous disease in tabes dorsalis or syringomyelia. It may also be peripheral in origin and result from the toxins of diphtheria and influenza or is sometimes an early symptom of myasthenia gravis.

If the paralysis is bilateral the soft palate hangs downwards and forwards, and reflex movements cannot be elicited. When the paralysis is unilateral, as it may be in syringomyelia, the uvula is drawn towards the sound side, while the paralysed half of the palate is lower and less arched. The voice is nasal, and there is nasal regurgitation of fluids on swallowing. If the constrictors are affected there is increasing difficulty in swallowing, especially fluids. It should always be noticed at the same time whether there is paralysis of the vocal cords and tongue. The tongue deviates to the paralysed side on protrusion and fibrillation may be observed in the paralysed half. In progressive bulbar paralysis the hypoglossal nucleus with its morphological continuation backwards is affected, so that the tongue and muscles attached to the hyoid bone are paralysed, but the pharyngeal muscles and intrinsic muscles of the larynx escape. The dysphagia and dysphonia are due to inability to fix the larynx, which by its weight stretches the palatopharyngeal muscles. The atrophy of the mylohyoids and digastrics renders the patient unable to open the mouth widely, and as the larynx cannot be fixed laryngoscopic examination is difficult or impossible (see p. 445).

### PHARYNGEAL POUCH

A pressure pouch (Fig. 201), sometimes called a Zenker's diverticulum, arises as a small pouch in the posterior median line of the hypopharynx. It emerges between the oblique and transverse portions of the inferior constrictor muscle, and as it enlarges and sags downwards it becomes the direct continuation of the pharynx, the Œsophageal opening lying concealed in front. As more and more food enters it the pressure on the back of the Œsophagus increases and with it the dysphagia. The pouch may eventually sag down into the posterior mediastinum, where radiography will display a characteristic retort-shaped shadow (Fig. 202).

The majority of the patients are elderly men, whose symptoms are long-standing dysphagia and regurgitation of undigested food.

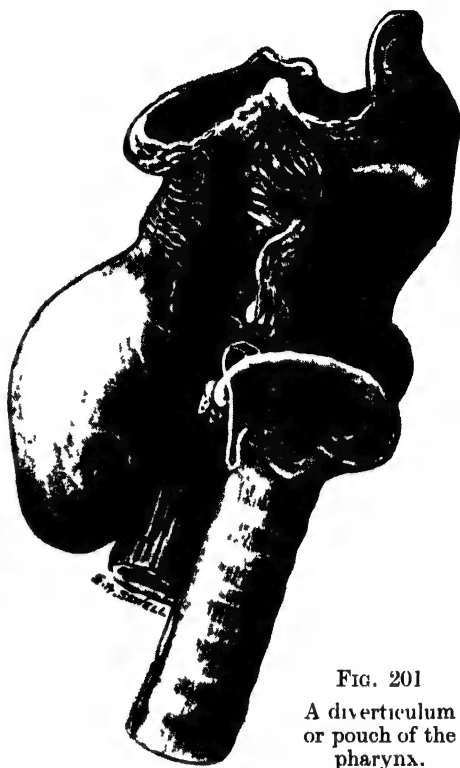


FIG. 201  
A diverticulum  
or pouch of the  
pharynx.



FIG. 202

A semilateral view of a pharyngeal pouch  
shown by radiography.

Sometimes a swelling, which can be emptied by pressure, appears in the neck after eating. Cough is caused by the overflow of liquids from the pouch, and the patients become slowly emaciated. Occasionally malignant disease supervenes at the orifice. Relief may be obtained by washing out the decomposing food with a funnel and tube, but removal of the pouch gives good results. To avoid mediastinitis or wound infection of the neck the operation should be performed in two stages. By an oblique incision along the anterior border of the sternomastoid under local anaesthesia if necessary, the carotid sheath is exposed and retracted. The pouch, which consists of thickened mucous membrane and connective tissue, is dislocated out of its bed and left

out of the wound (Fig. 203). Its neck, which may be twisted slightly, is fixed to the edges of the wound at its upper part by catgut stitches. The relief of symptoms is immediate. After ten days the projecting pouch is cut away, its edges invaginated and the wound sutured.

The œsophagoscope is not necessary either for diagnosis or during the operation, but if used, caution is necessary to avoid perforating the thin wall.

### TUMOURS OF THE PHARYNX

**Benign Tumours** of the oropharynx, with the exception of papilloma, are rare, but adenoma, fibroma, lipoma, angioma and both simple and dermoid cysts have been recorded. Papilloma occurs on the tonsils, faucial pillars, soft palate and uvula.

Such tumours often give no symptoms, but if causing cough from irritation of the pharynx, if interfering with speech or deglutition, or if increasing in size they should be excised. A tumour such as an angioma, likely to cause hæmorrhage, should be removed by diathermy.

**Malignant Tumours** of the oropharynx include sarcoma, endothelioma and carcinoma. Any form of sarcoma or a mixed tumour may occur. The usual site is the tonsil, about puberty. An enlargement of one tonsil should always be regarded as suspicious of malignant disease. A smooth prominent swelling appears, which may easily be mistaken for a quinsy, and is associated with some enlargement of the lymphatic glands at the angle of the jaw. Ulceration, fungation and hæmorrhage appear relatively later than in carcinoma, though the course of the disease is rapid.

In order to establish an exact diagnosis so that the treatment may be appropriate, a piece of the tumour is removed for microscopic examination. The use of the diathermy knife is recommended for this in order to avoid hæmorrhage and the risk of dissemination.

**CARCINOMA** is usually squamous-celled and attacks the fauces, tonsil and soft palate. The lower part of the anterior pillar is said to be the usual site of origin. From there it soon spreads to the tonsil and the side of the tongue. The glands at the angle of the jaw become enlarged at an early stage, but are not invariably affected. There is discomfort and salivation associated later with pain, radiating to the ear. Later still, hæmorrhage, dysphagia and general wasting follow.

It may easily be mistaken for an ulcerating gumma, especially if the Wassermann test happens to be positive, but the edge of a carcinoma is hard and raised and not so sharply cut as that of a gumma.

**LYMPHO-EPITHELIOMA**, described by Schminke and Regaud, arises in lympho-epithelial tissue. Such tumours were formerly classified as alveolar sarcoma. The tumours consist of epithelial cells and lymphocytes intimately connected. The epithelial cells grow in sheets and show no signs of differentiation or of epidermoid evolution. The lymphocytes fill up the syncytial network formed by the epithelium. This form of epithelioma is rare, but is of importance because it is highly sensitive to treatment by radiation, and no attempt should be



FIG. 203

Pharyngeal pouch hanging outside the primary incision a few days before its removal at the second stage of the operation.

made to treat it by excision. If a squamous carcinoma is detected at an early stage, the tumour can be excised through the mouth. The cheek is retracted, the palatoglossus divided near the tongue, and the tumour excised with a wide margin by means of the diathermy knife.

The approach to these tumours, however, is dominated by the mandible, and if the tumour is no longer in quite an early stage, or has reached the side of the tongue, the lower jaw must be either divided or partially excised in order to expose it. This is the only possible method of treatment if the tumour has become attached to the inner aspect of the angle of the jaw.

The best method is to ligature the external carotid and perform laryngotomy. A long incision is made through the lower lip and carried backwards below the horizontal ramus. The mandible is then divided a little towards the same side, and the whole of that half of the mandible is removed along with the tumour attached to it, by disarticulating it at the temporomandibular joint. The pharynx must be carefully reconstructed by an inner and outer row of stitches, catgut being used for the inner layer in the mucous membrane. The patient must be fed with a tube, and the lymphatic glands excised later. The operation has the disadvantage of inflicting a mutilation, but the patient can eat in comfort after it and talk reasonably well, and a number



FIG. 204

A drawing illustrating cardiospasm. The increase in length and the sigmoid bend are well seen.

have survived the operation many years without recurrence.

If the tumour is not fixed to the jaw and has not a syphilitic basis, treatment by radium offers a promising alternative, provided the mouth is not grossly septic. Large needles attached to silk threads and containing from 1 to 1.5 mg. of radium should be thrust into the tissues near the periphery of the growth. The silk threads should be tied in pairs and brought out of the mouth. Large needles are essential, as small ones will not remain in or near the base of the tongue. Eight or ten such needles should be inserted and kept in position if possible for ten days. It is generally better, however, to employ either deep X-ray therapy or teleradium in the form of the radium beam. Sarcoma and lympho-epithelioma give better results than squamous carcinoma, but good results can be obtained with the last when the tumour is still confined to its site without local infiltration and the



lymphatic glands show no enlargement, or if palpable, are still movable and would be removable by operation. Treatment by surgery and radiation should not be combined.

Tumours of the laryngopharynx are described for convenience with tumours of the larynx.

## DISEASES OF THE ŒSOPHAGUS

### ŒSOPHAGECTASIA

This condition of the œsophagus is described also as achalasia or



FIG. 205

The X-ray appearance of an early stage of cardiospasm.

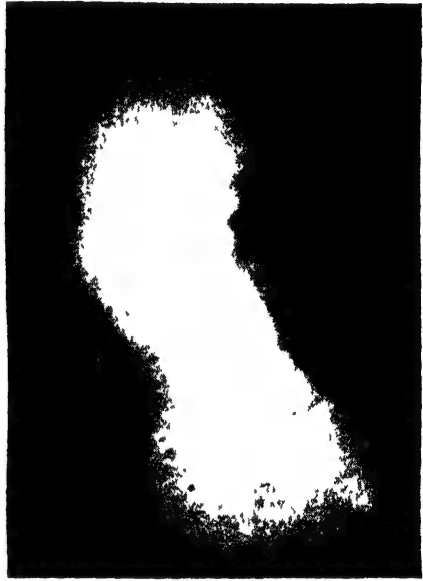


FIG. 206

The X-ray appearance of an advanced stage of cardiospasm.

cardiospasm. The œsophagus is dilated and hypertrophied and is obstructed at the lower end (Fig. 204). The patients are usually, but not invariably, women, and the disease progresses slowly for many years. The obstruction at the lower end is largely functional. There is increasing dysphagia with emaciation, and the dilated gullet contains undigested but decomposing food and liquids. There is occasional regurgitation. The œsophagus becomes lengthened and the dilatation affects chiefly the lower end, so that instead of being spindle-shaped it usually becomes sigmoid. The mucous lining is inflamed and ulcerated with patches of leukoplakia and polypoid hyperplasia. Eventually carcinoma may supervene. The circular muscle becomes hypertrophied and the plexus of Auerbach has been found to be degenerated, and inco-ordination from want of relaxation (achalasia) has been suggested as a cause.

In addition, phrenospasm, on the view that the obstruction is at the diaphragm and not at the cardia, and congenital malformation have been suggested as etiological factors.

Radiographic examination, after the administration of a barium meal, shows enormous dilatation of the œsophagus, terminating in a funnel-shaped extremity passing to the left towards the cardia (Figs. 205 and 206). The weight of the fluid may cause the lower part of the dilated gullet to sink below the level of the diaphragmatic opening.

On examination with the œsophagoscope a large quantity of turbid fluid is encountered and should be removed by suction. The œsophagoscope can be passed into the cardia without difficulty. Relief can be given by washing out the œsophagus regularly with a funnel and tube. The most effective treatment is repeated dilatation of the cardia by means of a bag containing mercury. Sympathectomy has been given a trial, but has not proved so successful clinically as the experimental results promised. This is apparently due to the difficulty of dividing all or a sufficient number of the sympathetic fibres, which run along the arteries supplying the stomach. Spinal anæsthesia has had some successes but probably as many failures.

### **DIVERTICULUM OF THE ŒSOPHAGUS**

Traction diverticulum is usually situated near the bifurcation of the trachea, where the left bronchus crosses the œsophagus and is caused by enlarged tracheobronchial glands becoming adherent to the wall of the œsophagus. The traction is caused by movements of respiration and deglutition.

Pulsion diverticulum is still more rare. Epiphrenic diverticulum occurs in the lower part of the œsophagus on the left side and is formed by a hernia of the mucous membrane through the longitudinal muscle fibres.

These conditions do not usually call for any treatment, but excision of the diverticulum has been successful in a few cases.

### **RUPTURE OF THE ŒSOPHAGUS**

The œsophagus is very intolerant to the passage of instruments by which the wall can be damaged easily unless great care is exercised. Spontaneous rupture is rare, and only occurs during vomiting or violent retching, but probably never in a healthy œsophagus. It is always seen in the lower third in the long axis and is followed by severe pain in the epigastrium and shock, so that the symptoms resemble those caused by perforation of a gastric ulcer. The resulting mediastinitis is invariably fatal, usually in about twenty-four hours.

### **CICATRICAL STENOSIS OF THE ŒSOPHAGUS**

This is rarely the result of disease, but it may follow ulceration caused by syphilis or scarlet fever and sometimes other fevers. It more commonly is due to swallowing corrosive fluids, whether by accident or with suicidal intent. Such strictures caused accidentally are comparatively common in America and Eastern Europe. The stricture is

apt to form where the fluid comes into closest contact with the lining of the œsophagus, *i.e.*, at the narrowing caused by the crossing of the left bronchus. The healing of the ulceration may, however, produce multiple strictures. If the stricture is impermeable, gastrostomy is urgently necessary. It may be possible later to construct a gullet from the skin of the chest-wall by a plastic operation. If the stricture is permeable it may be very gradually dilated by bougies passed through the œsophagoscope, never blindly. Another method is to perform gastrostomy and give the patient a silk thread to swallow. The end of the thread is found in the stomach with the aid of a cystoscope and



FIG. 207

Carcinoma of œsophagus at level of the aortic arch, as revealed by a barium swallow.

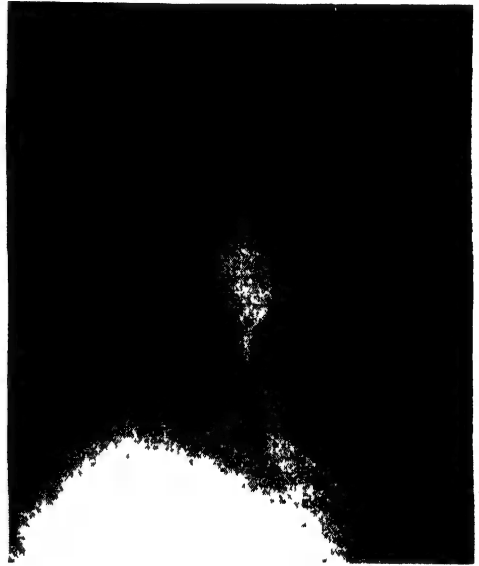


FIG. 208

Carcinoma of the œsophagus at the cardiac end, as revealed by a barium swallow.

brought out through the gastrostomy. Graduated metal olives are threaded on the silk in groups of three on an endless chain which passes through the mouth, down the gullet, through the stricture, out of the gastrostomy and, after cleansing, back into the mouth again. This treatment is very prolonged but is capable of yielding satisfactory results.

### CONGENITAL SHORTENING OF THE ŒSOPHAGUS

Some cases of dysphagia, especially in children but also in older patients, are explained by congenital shortening of the œsophagus, which ends at the level of the VIIth dorsal vertebra. The stomach is thus partly thoracic and there may be an associated diaphragmatic hernia (p. 580). There may be a stricture from chronic inflammatory changes at the junction of the short œsophagus and the thoracic stomach. The normal function of the cardia being in abeyance, regurgitation of gastric juice into the œsophagus produces peptic ulceration, a cause of

severe pain. The thoracic stomach may be dilated and cause vomiting, often a prominent symptom in infants. The stricture should be treated by dilatation under endoscopic vision. Postural treatment is also of help in preventing regurgitation of gastric juice.



FIG. 209

Esophagus from a case of cardio-spasm. A squamous-celled carcinoma has arisen in the dilated esophagus.

### MALIGNANT DISEASES OF THE OESOPHAGUS

The most common cause of oesophageal obstruction is malignant disease. An ulcerating squamous-celled carcinoma is the usual form (Fig. 209), but occasionally the lower end of the gullet is invaded by a carcinoma of the stomach, or a spheroidal-celled carcinoma may develop in a nest of gastric mucosa in the lower oesophagus. Sarcoma is quite rare. Metastases seldom occur, but local extension to glands or pleura and perforation of the trachea or left bronchus are common, so that recurrent laryngeal paralysis, broncho-pneumonia or pulmonary gangrene may be late effects. Perforation of the aorta is rare. The majority of patients are males, and the middle and lower thirds of the gullet are the usual sites. In the upper third it is less common, but in that situation it usually occurs in females, and, in the absence of pronounced dysphagia, may be mistaken for primary malignant disease of the thyroid gland, which it has invaded from behind.

Painless dysphagia accompanied by rapid wasting is the only early symptom. The disease occasionally runs its course without causing dysphagia, ulceration of the growth maintaining the gullet patent. A mass of glands at the root of the neck may be the first indication, or there may be dyspepsia or cough, or symptoms arising from the invasion of surrounding structures.

Examination by X-ray before a screen may show slowing of the opaque fluid at the level of the aorta, or obstruction at the cardia with filling of the oesophagus alone, or the passage through a stricture. An X-ray photograph will usually demonstrate the situation and degree of the stenosis. The length may be estimated by inverting the patient, so that the fluid indicates the lower extremity of the stricture. There is little or no dilatation above a malignant stricture (Figs. 207 and 208).

The *diagnosis* should be confirmed by œsophagoscopy, and if doubt still exists, a portion of growth may be removed for microscopic examination. This should be avoided if possible though it is commonly recommended, because it may lead to rapid extension of the growth and deprive the patient of the one slender chance of successful treatment by radiation. Fig. 209 shows a malignant growth arising in a case of cardiospasm.

*Treatment* is either by applying radium in containers fixed round an intubation tube, or more conveniently by inserting radon seeds into the growth with a long trocar and cannula through an œsophagoscope. This treatment usually relieves the dysphagia for several months and occasionally produces apparent cure.

Thoracotomy, after producing preliminary collapse of the right lung, has also been employed to provide access for the insertion of radon seeds from outside, but it only exposes one side of the œsophagus. A few good results have been obtained by using deep X-rays, but this method is liable to cause damage to the lungs. If radiation gives no relief the stricture may be intubated with a flexible metal Souttar tube, if necessary after preliminary dilatation with a bougie. If the stricture is high up or low down and the patient is showing signs of exhaustion from lack of fluid, gastrostomy should be performed without delay. In a few dramatic cases the œsophagus has been resected with success, but such attempts are usually attended by a fatal result.

LIONEL COLLEDGE.

## CHAPTER XXIII

### THE LARYNX

**S**URGICAL *Anatomy of the Larynx.*—The principal cartilages of the larynx are the thyroid and cricoid, which can be palpated on external examination of the neck. The notch in the upper border of the thyroid cartilage is easily felt, even in a fat short neck and is often visible in males, so forming an unfailing landmark. The body of the hyoid bone lies above it but the great cornu on either side is more easily felt than the body. Palpation of the thyroid cartilage with the forefinger in the notch and the thumb and second finger on each wing gives a fair idea of the size of the larynx, which is smaller and softer in women than in men. It is also higher in women and partially concealed by the chin. Lateral movement on the œsophagus and front of the spine normally produces a distinct click.

An interval occupied by the cricothyroid membrane separates the lower border of the thyroid from the ring of the cricoid cartilage, which in adults is opposite the 6th cervical vertebra, and rather higher in children. A lymphatic gland called after Poirier lies on the front of the cricothyroid membrane. Below the cricoid the cricotracheal membrane connects it to the trachea, of which there are about seven or eight rings in the neck, but the length of trachea in the neck depends on extension of the head, whereby the trachea can be pulled up out of the thorax for an inch or more. The thyroid isthmus lies in front of the 3rd and 4th tracheal rings, but it may cover a larger area. The whole length of the trachea, which reaches to the 4th dorsal vertebra, is about  $4\frac{1}{2}$  in. in the adult.

In ordinary quiet breathing the larynx scarcely moves, but in laryngeal obstruction it is drawn forcibly down towards the thorax at each inspiration, which is accompanied by stridor. In tracheal stenosis the movement of the larynx is much less, while stridor caused by the dyspnoea accompanies both inspiration and expiration.

Inspection with the laryngoscope should include many structures besides the vocal cords which bound the glottis. On the base of the tongue are the circumvallate papillæ and the foramen cæcum, the lymphoid follicles forming the lingual tonsils and the central and two lateral glosso-epiglottic folds. These connect the epiglottis with the base of the tongue and enclose the right and left vallecula on either side. The *epiglottis*, slightly yellow from the underlying yellow elastic fibrocartilage, projects up behind the base of the tongue, but it may curve backwards and overhang the entrance to the larynx. On either side the free border merges with the aryepiglottic folds, which pass obliquely downwards and backwards to the tips of the arytenoids and enclose the cartilages of Wrisberg. The arytenoid cartilages articulate below with the cricoid cartilage, on which they rotate so that the glottis opens and shuts. Their apices project upwards and backwards and support the cartilages of Santorini, which are sometimes very prominent. The ventricular bands, or false vocal cords, are folds of mucous membrane lying above the true cords, but farther apart, so that normally the glottis is visible. Below and outside the ventricular bands are the ventricles of the larynx, the

openings of which lie between the true and false cords. The subglottic region is narrow immediately below the cords but widens so that the upper rings of the trachea and occasionally its bifurcation are often visible in the mirror.

The *hypopharynx* or laryngopharynx lies behind the larynx and extends down to the lower border of the cricoid cartilage, where it joins the oesophagus. Only the upper part is visible as far down as the arytenoid cartilages, but on either side a narrow opening bounded internally by the aryepiglottic fold and externally by the thyroid ala lead down to the fossa pyriformis. This opening is widened on phonation, and it should be observed whether the pyriform sinus is being properly drained or whether the entrance is occupied by a pool of mucus or mucopus.

The *vocal cords*, the most conspicuous structures in the mirror on account of their apparent whiteness, as well as their position, are the ligaments of the thyro-arytenoid muscles formed by the upper extension of the crico-thyroid membrane. In front they are attached to the vocal processes of the arytenoid cartilages and their anterior surfaces. The mucous membrane covering the vocal cords is thin and closely adherent. Examination of the cords in the mirror includes observation of their movements and tension as well as of their colour and surface.

*Methods of Examination.*—Examination with the laryngeal mirror shows an image similar to that in any ordinary mirror, in that it is reversed in the anteroposterior direction but the right remains on the right and the left on the left, as the figure illustrates (Fig. 210).

The larger the mirror that can be used conveniently, the better the view. The reflecting surface is warmed over a spirit lamp to prevent a cloud of moisture condensing on the glass, and the back of the mirror tested on the cheek or the dorsum of the hand to avoid the risk of burning the palate of the patient. This is of particular importance if cocaine has been applied to abolish the pharyngeal reflex. The patient must raise his head, open his mouth wide and protrude his tongue fully without holding his breath. The tongue is held with a tongue cloth between the thumb and second finger, and the first finger is used to raise the upper lip. The laryngoscope is usually held in the right hand, but it is wise to practise holding it in the left hand so that forceps can be used with the right hand while the patient holds his own tongue. The mirror is made to follow the curve of the tongue and then applied firmly to the soft palate and uvula. Using the soft palate as the fulcrum the various parts are brought into view by tipping the mirror. Steady pressure without touching other structures does not excite the pharyngeal reflex, but if the pharynx is very irritable a little 5 per cent. cocaine may be applied to the palate. This is rarely necessary if the examination is not made too soon after a meal.

If only the posterior part of the larynx is brought into view the patient must say "eh," which raises the epiglottis and improves the view; if the anterior part of the larynx is still not visible, a high note, "ee," must be sounded whereby the epiglottis is raised to the utmost and the anterior commissure is brought into view. This phonation will show whether the cords adduct normally to the middle line. To test abduction the cords should be made to adduct; then on drawing a breath the cords abduct to the full extent and if either or both are fixed the position is noted. The most



FIG. 210

The larynx in normal quiet breathing as seen by laryngoscopy.

careful manipulation is unpleasant to the patient, and it is better to make several short examinations with intervals for rest than to keep the mirror in position too long at one time (Fig. 211).

Direct examination of the larynx with a tube spatula is essential for laryngoscopy in small children, and is now generally used for intralaryngeal operations, though these can often be performed by the indirect method with a mirror. The best model is that of Chevalier Jackson, which is illuminated by a small lamp at the distal end, or the modification of Negus,

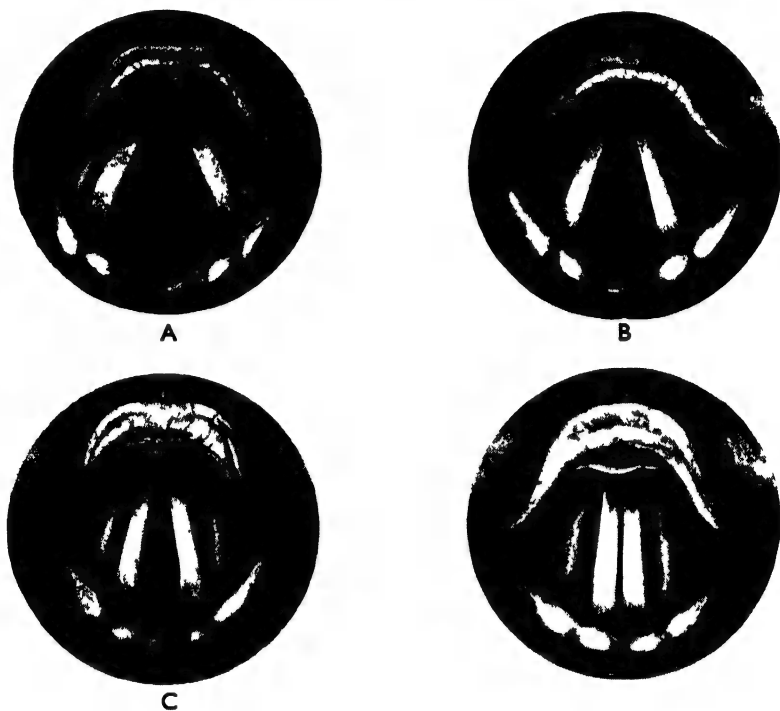


FIG. 211

Laryngoscopic views of the normal larynx in (A) deep inspiration, (B) quiet breathing, (C) death and (D) phonation.

(Butterworth.)

who has introduced a cystoscopic lamp with an optical system which projects the light forward down the larynx and trachea. The older instruments with proximal illumination are efficient but more difficult to use owing to the position of the lamp and its reflector. The examination can be made sitting, but it is better to have the patient lying on his back with the shoulders raised and the head supported by an assistant. A general anæsthetic may be used, but is not necessary unless the patient is very intolerant, and an injection of scopolamine or hyoscine one hour beforehand is usually sufficient. In either case 10 per cent. cocaine must be applied to the back of the tongue, the epiglottis, the lower pharynx and larynx to abolish spasm and reflexes. An effective method of cocaineisation is to thrust a swab on a long carrier into each pyriform fossa and hold it firmly there for thirty seconds against the superior laryngeal nerve as it perforates the thyrohyoid membrane. The area of distribution is soon rendered anæsthetic. Cocaine should not be used in children, but a general anæsthetic is permissible.

The head and neck of the patient should project beyond the end of the table and be supported by the left arm of an assistant, who sits opposite



the head on the right-hand side of the table. With his left foot on a stool in order to support his arm on his knee, the assistant can extend or flex the head as required. A special movable head-rest can replace the assistant, but proper support of the head and neck is essential to all endoscopic examinations. The usual fault lies in allowing over-extension of the head and neck. The laryngoscope or tube spatula held in the left hand is introduced until the epiglottis comes into view. The beak must not be allowed to go too far into the deep pharynx, otherwise all landmarks are lost, but it is passed just over the epiglottis and then tilted so as to pull the epiglottis forwards and expose the larynx. The right hand is thus left free to use forceps for removal of a small tumour or a piece of tissue for microscopic examination.

For examination of the trachea or bronchus a longer tube passed through the glottis is necessary.

In order to protect the air passages from infection by the pharynx and to facilitate the introduction of the bronchoscope through the glottis, Chevalier Jackson first introduces an open laryngeal spatula with the left hand, hooks the larynx well forward, then passes the bronchoscope through with the right hand and withdraws the spatula. In this way the bronchoscope is introduced without touching the tongue or pharynx, but it is possible to insert the bronchoscope in the same way as the laryngoscope. The beak is passed between the cords when they separate during inspiration, and it may be necessary to wait a few moments for the glottis to open until spasm has passed.

Occasionally, in the case of real dyspnoea the instrument may have to be pushed carefully through the glottis. When advancing the bronchoscope beyond the larynx, the direction of the trachea backwards as well as downwards along the dorsal spine must be kept in mind, and the head of the patient correspondingly elevated by the assistant. Secretion should be cleared by a tube attached to a suction apparatus, and the bronchoscope advanced under direct vision only, the assistant always holding the head so that the trachea beyond is in line with the bronchoscope. Before attempting to pass the instrument into either main bronchus the carina must be identified, again keeping in mind that the right bronchus is the continuation of the trachea, and that the left is endoscopically a lateral branch. To enter either main bronchus the head of the patient must be moved a little towards the opposite side.

To avoid subglottic oedema from pressure on the larynx it is necessary in swinging the bronchoscope to one side to keep the fulcrum at the thoracic aperture and not at the level of the larynx, and in children to avoid using too large a bronchoscope. Advancing the instrument down the main bronchus, the openings of the upper and of the lower lobe bronchus can be identified and on the right the orifice of the middle lobe bronchus can be seen anteriorly below the upper lobe bronchus.

### FOREIGN BODIES IN THE LARYNX

A large foreign body impacted in the larynx is likely to cause sudden death from asphyxia, but if the opportunity is offered an immediate laryngotomy or tracheotomy would be indicated (Fig. 212).

Small foreign bodies, such as a pin or a nail cause sudden pain, cough and loss of voice. Even in this case spasm of the vocal cords may have an immediately fatal effect, but it usually soon passes if the foreign body becomes impacted. If it moves fresh attacks of spasm

are produced, and sometimes hæmoptysis. If the foreign body is not soon removed inflammatory œdema, perichondritis and abscess may follow, though a long period, even years, may pass without the development of serious symptoms.

Examination by indirect or direct laryngoscopy will usually establish the diagnosis, but the assistance of radiography may be required in a suspected case.

*Treatment.*—Tracheotomy may be necessary to relieve dyspnœa before removal is undertaken by direct laryngoscopy with straight forceps. The need may arise during an attempt at removal, so that the necessary instruments should be ready. The foreign body must be firmly grasped in the forceps before any attempt is made to withdraw it, otherwise it may slip and fall down into the trachea or a bronchus. If this method fails laryngofissure is indicated.



FIG. 212

Bolus of food impacted in the larynx causing sudden death.

**ACUTE LARYNGITIS.**—Acute inflammation of the larynx commonly takes the form of acute catarrh secondary to the acute rhinitis of coryza or the specific fevers, and is often associated with tracheitis and bronchitis. The vocal cords become swollen and injected, in severe cases their movements may be impaired, and there is transient hoarseness or even complete loss of voice. Occasionally submucous hæmorrhages occur, which may either absorb or later organise into small fibrous tumours. The best treatment is rest of the voice and an inhalation of steam containing

tinct. benzoini co. In children it may cause severe attacks of dyspnœa at night (croup or laryngitis stridulosa), and has then to be distinguished from diphtheritic or membranous laryngitis and from the spasm of laryngismus stridulus which occurs in rickets. In laryngismus stridulus the voice and breathing are normal between the attacks of dyspnœa, which may be accompanied by tetany (carpo-pedal spasm).

A more severe form of acute laryngitis is caused by infection with the streptococcus pyogenes from the teeth, mouth or pharynx. The onset is often sudden, with symptoms of fever, and the mucous membranes become much swollen. The epiglottis and loose tissue of the aryepiglottic folds swell rapidly, and dyspnœa is more liable to be caused at this level than by swelling of the cords, so that the term

*œdema of the glottis* is misleading. Prontosil or serum should be given; any collection of pus, such as a quinsy, must be opened, and if the breathing is seriously embarrassed tracheotomy performed before the patient becomes exhausted by dyspnoea and general intoxication, either of which may be fatal.

This type of septic laryngitis, occurring in typhoid, smallpox, diphtheria, erysipelas or pneumonia may lead to perichondritis. The cricoid is the cartilage most liable to be affected, but the thyroid and arytenoids do not always escape. The inflammation usually ends in suppuration, causing necrosis and exfoliation of the cartilages and the formation of sinuses, with much deformity and stenosis of the larynx. Pus should be evacuated by incision as soon as it becomes localised and fragments of cartilage removed, but the vocal cords are liable to become fixed by ankylosis of the crico-arytenoid joints, and tracheotomy is almost inevitable.

Chronic perichondritis with similar results is secondary to syphilis, tuberculosis or malignant disease, especially the last; and permanent tracheotomy is required.

Membranous laryngitis, which occurs in children, is usually diphtheritic, occasionally streptococcal and rarely due to caustics. The membrane is usually present also in the pharynx, but the diagnosis of diphtheria may be difficult if it exists only in the larynx, from which it spreads down the trachea. Examination by the direct method is required in this case to establish the diagnosis. If diphtheria is even suspected treatment by antitoxin should be given.

Increasing dyspnoea is an indication for tracheotomy, which should not be delayed until the condition of the patient, usually a child, is grave.

### CHRONIC LARYNGITIS

This results from repeated attacks of acute inflammation, but important predisposing causes are mouth-breathing from nasal obstruction, chronic catarrh from infection of the accessory sinuses of the nose, abuse of the voice combined with faulty production, direct irritation, sometimes occupational, but commonly from excessive smoking and some general disorders amongst which gout is important.

Alteration in the voice may be slight, but it is usually hoarse, and the singing voice may be lost. There is excessive secretion from the larynx and cough. In severe cases with pronounced local changes the diagnosis may have to be made from tuberculosis, syphilis and malignant disease. Hyperæmia and hyperplasia are rarely unilateral, as in the early stages of these diseases. In a localised form the changes may occur at the junction of the anterior and middle thirds of the cords as "singer's nodules." These are seen chiefly in female singers and school teachers, and are best treated by lessons in voice production, so that abnormal strains on the cords from forcing the voice are eliminated.

Atrophic laryngitis, in which the larynx is lined with foul green crusts, is almost invariably secondary to atrophic rhinitis (ozæna), but it may occur primarily and even spread down the trachea.

Pachydermia of the larynx is localised to the vocal processes where the papillæ increase and outgrowths of highly keratinised epithelium project. The outgrowth on one side commonly fits into a corresponding depression on the summit of the opposite one, so that the cords can almost meet in spite of these excrescences. Gout is often associated with the pachydermia, which may be benefited by treatment at a spa such as Luchon or Ems.

### TUBERCULOSIS OF THE LARYNX

This is so rarely primary that it should always be regarded as a complication of phthisis. The first manifestation may be laryngeal, but unless the disease is arrested the pulmonary lesion will soon be revealed by physical signs or by the presence of tubercle bacilli in the sputum. When in doubt radiography may reveal the primary lesion in the lung before physical signs appear.

In most cases the larynx becomes infected from the sputum, either through slight abrasions or possibly the unbroken mucous membrane, but a persistent laryngeal catarrh is frequently the forerunner of a definite deposit of tubercle.

The bacilli thus reach the lymphatics, where the disease starts, and whence it spreads in the larynx. There is therefore a predilection for the disease to attack the neighbourhood of the posterior commissure, the part most richly supplied with lymphatics, and in some cases the path of infection from the lung to the larynx may be by the lymphatics.

In the last twenty years the disease has diminished in frequency both absolutely and in relation to pulmonary tuberculosis by about one half. This is shown both from clinical records of early cases and by post-mortem observation on cases of phthisis. It occurs now in about 5 per cent. of early and about 30 per cent. of advanced cases. The age incidence corresponds to that of phthisis, but senile tuberculosis of the larynx is not uncommon and may be mistaken for malignant disease. Sex has no influence, and the old view that it is commoner in males is no longer correct.

Localised patches of anæmia or hyperæmia in the larynx and chronic laryngitis, which are not tuberculous, are frequent in sufferers from phthisis and may open the path to true tuberculous infection.

In early cases the interarytenoid fold is most commonly attacked in the form of a deposit, which may be raised into a peak by the compression of the arytenoids during phonation, or of an irregular indolent ulcer. The posterior laryngeal wall immediately above and behind the vocal process of the arytenoid is also a frequent site for the deposit of early tubercle. Such a lesion may be unilateral or more advanced on one side. The central and posterior thirds of the vocal cords are the next most frequent sites. The cord becomes pink, loses its lustre and tension, and an indolent shallow ulcer appears. A more extensive stage of this ulceration produces a "mouse-nibbled" appearance of the vocal cords. These are the three areas commonly affected early. Much more rarely the arytenoids, the epiglottis, the aryepiglottic folds and the ventricular bands may be the primary site in the larynx, the incidence being about equal among these areas. The cases thus tend

to fall into an intrinsic and an extrinsic group. At this stage, which is favourable for treatment, there may be no local symptoms or only a husky voice. In the more advanced stage of the disease the extrinsic parts of the larynx show a pale translucent swelling of the mucous membrane. The surface over the arytenoids and the aryepiglottic folds become pyriform on one or both sides. The swelling of the epiglottis is described as turban-shaped, and it may hide the interior of the larynx from view. Finally, deep ulceration may cause destruction of the epiglottis and perichondritis of the arytenoid cartilages, or rarely of the thyroid or cricoid.

This perichondritis is occasionally localised to the neighbourhood of a crico-arytenoid joint, which becomes ankylosed with fixation of the corresponding vocal cord.

In the later stages pain and difficulty in swallowing are the most serious symptoms, accompanied by cough and expectoration. The voice becomes weak and husky, but functional loss of voice is rather a symptom of phthisis and occurs without local changes in the larynx. Dyspnoea is rare, but subglottic oedema, extensive supraglottic swelling or perichondritis occasionally call for tracheotomy.

The *diagnosis* has to be made from syphilis, intrinsic cancer and occasionally from pachydermia. Rarely aspergillosis causes an ulceration around the glottis indistinguishable in appearance from tuberculosis.

In early cases the *prognosis* is now less unfavourable than in the last century, when the disease was almost invariably fatal. Recovery is now possible in over 30 per cent. of cases, but the outlook depends largely on the condition of the lungs. Advanced cases are still almost hopeless.

*Treatment.*—Rest to the larynx by maintaining absolute silence is the sheet anchor in treatment and can be followed only in a sanatorium. In selected cases localised lesions on the cords may be treated by applications of the electric cautery to produce fibrosis. Occasionally the epiglottis may be amputated with punch forceps. In advanced cases a linctus of morphia or heroin is necessary for the cough, lozenges or insufflations of orthoform or anæsthesin to relieve painful ulceration, and applications of cocaine or alcohol injections of the superior laryngeal nerve to alleviate dysphagia, but tube feeding may be necessary.

### SYPHILIS OF THE LARYNX

Primary sores are reported to have been observed on the edge of the epiglottis and even on the left false cord, but in such unusual situations the disease is not likely to be recognised before the appearance of secondary manifestations.

Secondary syphilis commonly shows itself in the larynx as an erythema. The vocal cords have a mottled appearance. Mucous patches on the epiglottis, aryepiglottic folds or vocal cords are less common, appear later and are evanescent. They may be followed by superficial ulceration which is a rare and a late manifestation of the secondary stage. The only local treatment required is an inhalation of benzoin vapour.

In the *tertiary stage* laryngeal syphilis may have very serious effects.

(1) A diffuse gummatous infiltration may affect the epiglottis, the arytenoid eminences or the false cords. The colour is deep red or purple with sometimes a yellow spot which indicates an area of softening. A circumscribed gumma appearing as a definite tumour in the same situations is rare and multiple nodular gummata rarer still. (2) The commonest manifestation of tertiary syphilis is deep ulceration supervening on a gumma. The ulcer takes the form of a crater with sharp punched out edges and a congested areola. The base is grey and sloughy. (3) Gummatous perichondritis attacks the thyroid cartilage chiefly, causing much swelling inside the larynx and narrowing of the glottis with dyspnoea. Any of the other cartilages may be attacked with the formation and exfoliation of a sequestrum. (4) The larynx may be much distorted by scars and adhesions after healing. The epiglottis may be destroyed and the vocal cords united by cicatricial webs. There may be subglottic stenosis or stenosis of the trachea, and the crico-arytenoid joints may become ankylosed.

The *symptoms* correspond to the various pathological conditions. There may be only hoarseness, but the voice is strong and raucous. Ulceration of the extrinsic regions causes pain and dysphagia. Stenosis causes dyspnoea and stridor, worse at night. The insidious progress of the stenosis allows the patient to become gradually accustomed to it so that no distress is caused.

The *diagnosis* may have to be made from tuberculosis and malignant disease. Tertiary syphilis affecting a vocal cord may be indistinguishable in appearance from a carcinoma.

Examination of the chest and sputum, the Wassermann test and sometimes removal of a piece for microscopic examination may be employed, but the Wassermann test is often negative in this form of tertiary syphilis.

*General treatment* is essential. Arsenical preparations should be used with caution as they are liable to produce sharp reactions in the larynx. Injections of bismuth are preferable, and a few cases respond best to mercurial inunctions. Potassium iodide likewise must be given with caution if there is any laryngeal stenosis, but it is of great value provided that there is a free airway. Tracheotomy may be necessary to relieve dyspnoea from increasing stenosis of the glottis, but the need may sometimes be averted by prompt antisyphilitic treatment if the patient is kept in bed. The stenosis does not respond well to dilatation, and the tracheotomy cannula may have to be worn permanently if antisyphilitic treatment does not prove effective.

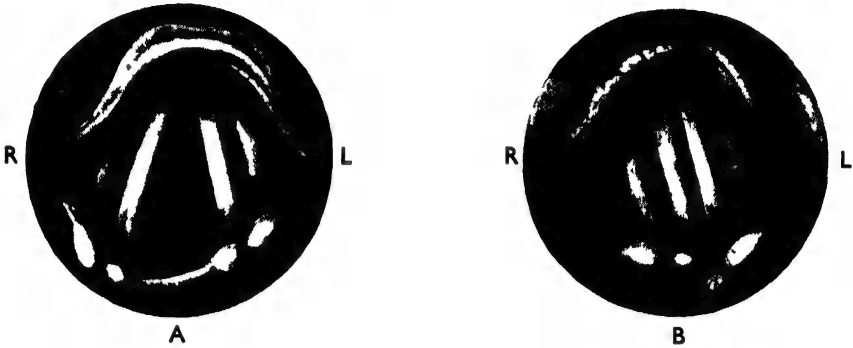
### PARALYSIS OF THE LARYNX

The laryngeal muscles act as (*a*) adductors, (*b*) abductors and (*c*) tensors of the vocal cords. In addition, the thyro-epiglottidean and aryteno-epiglottidean muscles act as sphincters of the larynx.

The vocal cords can assume four cardinal positions. (1) Extreme abduction produced by deep inspiration. (2) Moderate abduction in quiet respiration. (3) The cadaveric position, which is seen after

death or in complete paralysis of the recurrent laryngeal nerve, between moderate abduction and the median position. (4) The median position which is seen during phonation (Fig. 211).

The nerve supply of the laryngeal muscles is the recurrent laryngeal, except the cricothyroid, a tensor of the cords supplied by a branch of the superior laryngeal, which is also the sensory nerve of the larynx.



Paralysis of the left recurrent nerve. Position of the vocal cords on (A) inspiration and (B) phonation.

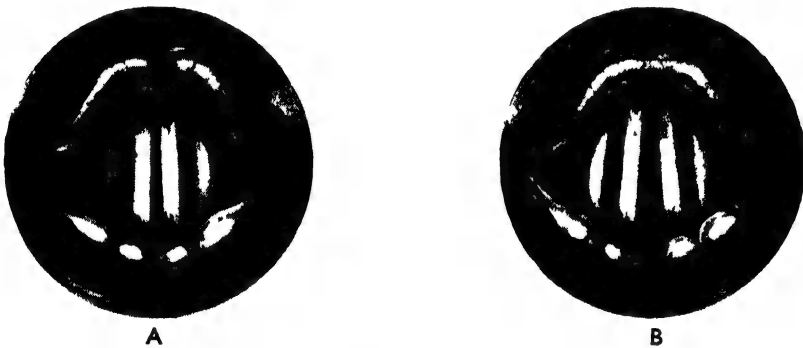


FIG. 214

Double abductor paralysis. Position of the vocal cords on (A) inspiration and (B) forced expiration.

Although the recurrent laryngeal nerves supply both the abductors and adductors, pressure or progressive lesions of the trunk or bulbar centre of the nerve produce firstly abductor paralysis. The internal tensors and the arytenoideus are affected next and the adductors last (Semon's law). The cortical centres act bilaterally so that laryngeal paralysis, unless functional, is not produced by cortical lesions. A lesion in the bulb may produce a unilateral or bilateral abductor or total paralysis (Figs. 213 and 214).

*Adductor paralysis* or functional aphonia is usually seen in young women. The crico-arytenoidei laterales and the arytenoideus are the muscles concerned, and there is usually also a paralysis of the tensors—the thyro-arytenoidei. The condition sometimes accompanies or follows an attack of acute laryngitis. It is important to remember that this functional paralysis is sometimes the earliest symptom of pulmonary tuberculosis.



Although the patient is unable to phonate, she can cough naturally. On examination the cords may make no attempt to approximate, they may move towards the middle line but fail to meet, or they may meet but immediately fly apart without the production of any sound. The edges of the cords are usually concave from paralysis of the tensors.

General treatment is more important than local. Anæmia must receive treatment. Strychnine is useful. Electricity applied locally usually has only a temporary effect. Lessons in voice production and proper breathing are necessary to obtain permanent cure.

*Paralysis of the cricothyroid muscles*, the external tensors of the larynx, is rare and usually results from diphtheria. If the whole superior laryngeal nerve is affected it may be accompanied by anæsthesia of the larynx. In addition to hoarseness and weakness of the voice, therefore, there may be coughing and choking from food entering the larynx.

The cords are slack and have an irregular outline, or the affected cord may be at a higher level than the other in unilateral paralysis. The treatment must be directed to the relief of any lesion of the superior laryngeal and, if necessary, to preventing food entering the larynx by employing tube feeding.

*Bilateral paralysis of the abductor muscles*, the crico-arytenoidei postici, may be central from tabes dorsalis, bulbar paralysis or syringobulbia. Bulbar paralysis, however, is more likely to produce paralysis of the tongue and hyoid muscles, and abductor paralysis of the cords is rare. In the course of the vagus or recurrent nerves pressure may be produced by aneurysms, cancer of the œsophagus, enlarged lymphatic glands, malignant or even simple goitre, and cut-throat or goitre operations may also produce abductor paralysis. It may follow a thyroid operation after an interval of several weeks. Peripheral or toxic causes are typhoid fever, influenza, diphtheria, rheumatic fever, pneumonia, scarlet fever and lead poisoning. Myopathic paralysis is generally due to direct extension of local disease, such as carcinoma of the lower pharynx. It may be simulated by ankylosis of the crico-arytenoid joints following a rheumatic arthritis or periarticular cicatrisation.

In progressive lesions the diminished power of abduction is first shown by the inability of the cords to separate beyond the cadaveric position on deep inspiration. When all power of abduction is lost, the cords remain in the middle line in the position of phonation owing to contracture of the unopposed adductors. The cords may be sucked towards the middle line on inspiration and blown slightly apart during expiration. The voice is usually unaffected and the chief symptom is increasing dyspnoea, worse on exertion and at night. In tabetics laryngeal crises takes the form of adductor spasm apart from abductor paralysis.

Antisyphilitic treatment occasionally succeeds in appropriate cases if not of too long duration. If the attacks of dyspnoea cause distress or threaten suffocation tracheotomy is necessary. It should be remembered that the lesion causing the paralysis may also cause dyspnoea from direct pressure on the trachea. A possible alternative



is to divide the recurrent laryngeal on one side and anastomose it to the phrenic nerve, which is capable of restoring movement to the cord. Occasionally one or both cords become completely paralysed and move out into the cadaveric position. In this case the dyspnoea is relieved but the voice is lost.

*In unilateral abductor paralysis* the causes are similar. In tabes, for example, the paralysis may be bilateral or unilateral, but as the left recurrent nerve turns round the aorta it is more likely to be stretched by an aortic aneurysm. Paralysis of the right cord is most commonly caused by a tuberculous lesion at the apex of the right lung, rarely from an innominate or subclavian aneurysm. At first the outward movement of the cord is defective, but later it becomes fixed in the middle line. The voice is not affected and treatment must be directed to the cause. Tracheotomy is not required as there is no dyspnoea except occasionally on exertion.

*In complete recurrent laryngeal paralysis* one or both sides of the larynx may be affected. The bilateral condition is rare, but complete paralysis of one cord with abductor paralysis of the other is often seen. Complete paralysis is a further stage of abductor paralysis following Semon's law. The affected cord is motionless in the cadaveric position with its edge concave from paralysis of the tensors.

The arytenoid cartilage is tilted forwards, which makes it very prominent, and it may be mistaken for an inflammatory swelling or tumour. On phonation the healthy cord which is taut and active crosses the middle line to approximate with the paralysed cord. The whole larynx in consequence looks asymmetrical and is sometimes thought to be tilted to the sound side. In the rare bilateral cases both cords are concave and lie motionless in the cadaveric position. In unilateral cases there may be hoarseness until the sound cord has adapted itself to cross the middle line and come into apposition with the paralysed cord. In bilateral cases phonation is lost entirely, and if in addition the thyro-epiglottidean and aryepiglottidean sphincters of the larynx are paralysed, there may be coughing and choking from food and drink entering the larynx and trachea.

A number of associated paralyses are described in which the last four cranial nerves are concerned. A variety of syndromes can thus occur of which the best known is that of Hughlings Jackson. In this there is associated paralysis of the larynx, palate, tongue and the sternomastoid with the trapezius. It is more important to classify these syndromes according to the level of the lesion causing the paralyses than by names. The most important situation is at the level of the jugular foramen, where the ninth, tenth, eleventh, twelfth and sympathetic may all be involved. The last is shown by narrowing of the pupil and enophthalmos (see p. 492).

### TUMOURS OF THE LARYNX

**Innocent Tumours** of the larynx are comparatively rare. The most common are papilloma and fibroma, the latter originating

sometimes from the organising clot of a submucous hæmorrhage. Lipoma, angioma, chondroma, adenoma and myxoma are very rare.

**PAPILLOMA** (Fig. 215) is usually single but in children may be multiple. The growths are then situated on the cords and ventricular bands, and sometimes extend below the cords and even to the trachea, but not upwards to the epiglottis. The growth is warty and varies in colour from white to pink and red, and is usually but not invariably pedunculated. A single growth may attain large size in an adult. Hoarseness is the only symptom unless the tumour is large or multiple, when dyspnoea and stridor may result. **FIBROMA** is always single and is generally attached to the upper surface of the middle or anterior third of the vocal cord.



FIG. 215

Papilloma of the larynx in a child.

**ANGIOMA** occurs on the cords and also on any part of the mucosa in the neighbourhood. It is an occasional cause of hæmoptysis.

Innocent growths should be removed with laryngeal forceps either by the indirect method with the aid of a mirror or by the direct method using an endoscopic tube. In children dyspnoea may call for tracheotomy, after which papillomatous tumours sometimes disappear spontaneously. Chondroma or lipoma may also call for this before removal by laryngofissure. Angioma may be treated by the electric cautery to check hæmorrhage but will require for its removal laryngofissure, or very rarely lateral pharyngotomy, depending on the situation. Radium is sometimes used for benign growths, but there is great danger of cartilaginous radionecrosis, especially in children, so that simple excision is much safer.

**Malignant Tumours** of the larynx are relatively uncommon, as they only account for about 2 per cent. of the total incidence of cancer. They are ten times more common in men than in women, but the etiology is unknown, though smoking is considered by some to be a predisposing cause. They occur usually at the same period of life as elsewhere in the body, but are occasionally seen in quite young people.

Squamous-celled carcinoma is far more common than any other variety, but basal-celled carcinoma occurs in 2 per cent. of cases, while papillary carcinoma and adenocarcinoma are still more rare. Sarcoma and endothelioma are also rarely encountered. Metastatic deposits are extremely rare, but hypernephroma has been observed in the larynx. Laryngeal growths only rarely cause metastatic tumours in other organs.

Malignant tumours of the larynx were classified by Krishaber into intrinsic and extrinsic.

(a) Intrinsic tumours arise from the vocal cords, the ventricles and the ventricular bands.

(b) Subglottic tumours, though intrinsic in situation, present special clinical features.

(c) Extrinsic tumours which arise on the epiglottis, aryepiglottic folds and in the pyriform sinus, are from a pathological and surgical standpoint pharyngeal tumours.

(d) Mixed tumours may be either extrinsic or intrinsic in origin, but in an advanced stage occupy both situations.

INTRINSIC CARCINOMA, especially the common form which arises on the anterior portion of a vocal cord and spreads slowly along the cord, takes long to invade the cervical lymphatic glands, the vocal cords possessing few lymphatic vessels (Figs. 216 and 217).

EXTRINSIC CARCINOMA runs a more rapid course, invades the lymphatic glands early and is far more difficult to cure by excision (Fig. 218).

A. INTRINSIC CARCINOMA.—The only *symptom* in its early stage is hoarseness. There is no pain, cough or dyspnoea until the later stages, when the growth spreads across to the opposite cord at or often below the anterior commissure. In a still later stage the voice is reduced to a harsh whisper, the growth becomes extrinsic, and may produce perichondritis with dysphagia, salivation and general signs of cachexia.

The *diagnosis* has to be made from simple tumours, chronic laryngitis, and especially from tuberculosis and syphilis. Fixation of the



FIG. 216

Laryngoscopic appearance of an intrinsic carcinoma of the larynx.



FIG. 217

Specimen showing an intrinsic carcinoma of the larynx.

cord is an important sign in favour of malignant disease, but it is important to remember that the cord remains mobile in the early stages and that fixation indicates that infiltration has begun. The patient should be examined for signs of pulmonary tuberculosis by auscultation, radiography and testing the sputum and for syphilis by the anamnesis and by blood tests. A chronic fibrotic form of tuberculosis often simulates carcinoma, so that it is frequently necessary to remove a portion of growth with forceps for microscopic examination to make certain of the diagnosis.

In the early stages the growth may be excised by laryngofissure, or occasionally, if it is confined to the anterior part of both cords, by removing the front of the thyroid cartilage with the underlying growth. In more advanced cases, while the growth is still confined to the cavity of the larynx, total laryngectomy is indicated.

*Treatment* by radium can give good results in early cases suitable for laryngofissure, but rarely succeeds in the more advanced cases.

*B.* In SUBGLOTTIC CANCER hoarseness is not necessarily the earliest symptom, but the patient complains more of symptoms of laryngeal catarrh, and the disease may progress insidiously in this way until stridor develops. It is particularly apt to spread through the crico-thyroid membrane and to invade the gland of Poirier. It occasionally produces paresis of one vocal cord of obscure origin. Such cases are rarely suitable for laryngofissure, and total excision of the larynx is usually necessary.



FIG. 218

Laryngoscopic appearance of an extrinsic carcinoma of the larynx.

*C.* EXTRINSIC CANCER, usually on the epiglottis, aryepiglottic fold or on the lateral wall of the pharynx with extension to the aryepiglottic fold, at first produces only local discomfort which increases to pain radiating to the jaw and ear and aggravated by swallowing. Pain in the ear may for months remain the only symptom, or the first sign be the appearance of a hard gland in the neck. The voice acquires a characteristic muffled quality without actual hoarseness, especially if the growth is pedunculated and projects into the pharynx.

The progress is rapid with a steady increase of pain, salivation and dysphagia, and the development of a fixed mass of malignant glands in the neck.

The use of the laryngoscope is essential for diagnosis; and if the nature of the disease is suspected the diagnosis is simple.

In women a particular type of carcinoma appears in the pharynx, in the mucosa on the back of the cricoid plate, long causing the one symptom of dysphagia. The upper edge may be visible in the mirror, or sometimes only an area of congestion is seen on the back of the larynx above the growth. In men a pool of mucus at the entrance may be the only physical sign of a growth starting in the pyriform sinus below.

For this group of tumours, if seen early, the treatment is excision by lateral pharyngotomy combined with dissection of the cervical glands. Deep X-ray treatment can cause the tumour to disappear, but usually the improvement is temporary, and this treatment apparently may produce a general dissemination which otherwise is rare.

*D.* In the mixed form the disease is usually inoperable, but a pharyngolaryngectomy is occasionally successful. Tracheotomy may be performed for the relief of dyspnoea in cases otherwise inoperable, but the results are disappointing and the growth is apt to invade the tracheal aperture and to reach the skin surface.

## OPERATIONS ON THE LARYNX AND PHARYNX

**LARYNGOTOMY.**—A temporary opening for respiration is made through the cricothyroid membrane in cases of sudden and urgent laryngeal obstruction, or as a preliminary measure to some operations on the mouth and upper air passages, instead of a tracheotomy, which is usually preferable but takes longer and is more difficult. Laryngotomy is only suitable for adults, owing to the small size of the larynx in children. It has been replaced to some extent by intratracheal insufflation.

Local anaesthesia, by infiltration of the skin over the cricothyroid membrane with novocain, should be employed if there is dyspnoea and time allows. The head is extended and well fixed, and the landmarks of the laryngeal cartilages defined by palpation. A transverse incision in the skin, 1 in. long, is made over the cricothyroid membrane. The sternohyoid and sternothyroid muscles are retracted to either side to expose the membrane, which is perforated transversely by a narrow knife or sharp-pointed scissors. By keeping close to the upper edge of the cricoid cartilage, injury to the cricothyroid branch of the superior thyroid artery on either side is avoided.

**TRACHEOTOMY.**—Laryngeal obstruction calls for relief of the dyspnoea by tracheotomy. Such indications as injuries, tumours, inflammatory stenoses from syphilis and diphtheria have already been outlined. It is also indicated as a preliminary to the operation of lateral pharyngotomy to facilitate administration of the anaesthetic, maintain the air-way during the manipulation of the larynx and exclude blood from the lower air passages by packing the larynx and lower pharynx.

An opening into the trachea above the isthmus of the thyroid gland which covers the 3rd and 4th rings is called a high tracheotomy, and one below is a low tracheotomy. In practice, however, this distinction is not now maintained, because the thyroid isthmus should always be divided if possible and a median tracheotomy performed. Otherwise, if the tracheotomy tube slips out, the isthmus may cover the opening and prevent the tube from being re-inserted before the patient is asphyxiated. For a child with diphtheria, however, a high tracheotomy is indicated, while a tracheotomy for malignant disease should be placed as low as possible. In adults local anaesthesia by infiltrating with 2 per cent. novocain and a few drops of adrenalin should always be employed, especially if there is stridor. In children chloroform slowly administered is safe, but the anaesthesia must be light, so that there is no sudden increase of dyspnoea or cyanosis.

The patient lies on the back with a support under the shoulders so that the neck is extended, but if stridor is present it is to be remembered that the neck cannot be fully extended without increasing the dyspnoea. The extension draws the trachea up from the thorax and projects it forwards. The point of the chin and the suprasternal notch are to be kept carefully in the line. The skin, platysma and superficial fascia are divided by a vertical median incision from the lower border of the cricoid cartilage downwards. The anterior jugular veins, running either side of the midline, should be identified and drawn aside with retractors. The two layers of the superficial division of the deep cervical fascia are then divided, and the pre-tracheal muscles identified and retracted. The trachea is exposed by blunt dissection, and the isthmus of the thyroid gland drawn down or divided between clamps. The trachea is then steadied with a hook, if the patient is a child, and incised in the middle line. The edges are held apart with a dilator, and the warmed tube introduced. If the patient is an adult, it is better to cut an opening in the front of the trachea to fit the tube, for in this way pressure necrosis of the tracheal cartilages is

avoided. The insertion of the tube produces a bout of coughing, which may be prevented by instilling a few drops of 2 per cent. cocaine with a hypodermic syringe between two rings before the trachea is opened, if there is no urgency and no risk of blood entering the trachea. In the low operation the inferior thyroid plexus of veins must be avoided.

Points of special importance are to keep strictly in the middle line and to open the trachea in the midline. If the trachea be drawn to one side it may be missed and a wound inflicted in the cesophagus, the common carotid artery or the vertebral column.

The pre-tracheal fascia must be well cleared so that the tube is not inserted between it and the trachea. The opening must be in the front and not to one side of the trachea, or the tube will not lie comfortably in it. The cricoid cartilage must not be injured in children, or sub-glottic laryngeal stenosis will result.

For most purposes, Durham's tracheotomy tubes with a lobster-tailed inner tube and pilot serve best (Fig. 219). The outer tube should always be inserted with a pilot, which should be kept at hand in case the former slips and needs to be re-inserted. To insert the tube the pilot should be held at right angles to the neck, and as the tube is inserted it is rotated towards the middle line and raised.

In diphtheria the tube should be removed as soon as possible after the emergency has passed, but if a permanent tracheotomy is designed the outer tube should be left in

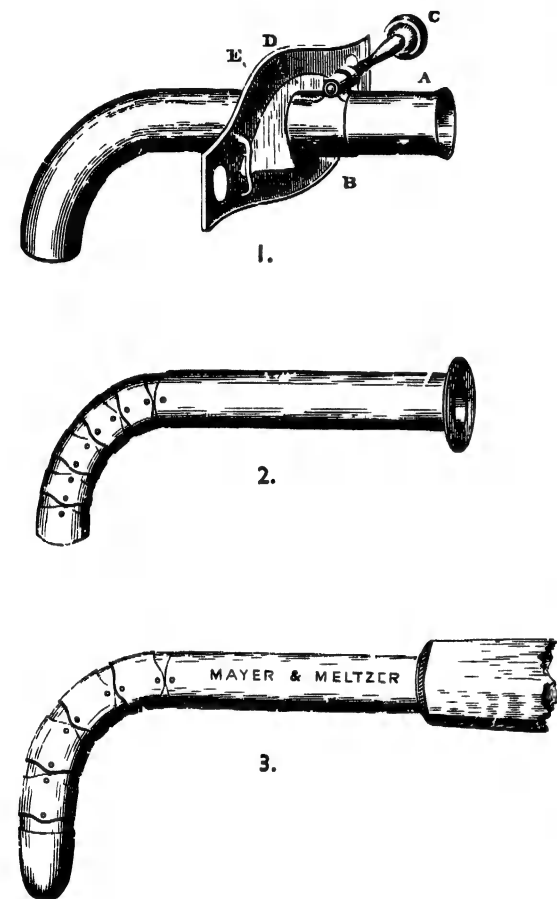


FIG. 219

Durham's tracheotomy tube, showing its component parts. (*Mayer & Phelps.*)

1. Outer tube. 2. Inner tube. 3. Pilot tube.

position for at least ten days to make a track before being removed. The inner tube may be taken out for cleaning as often as desired.

When a patient sits up and brings the head forward the trachea slips down and away from the surface, so that a greater length of tube is required, and the shield may need adjustment to fit comfortably against the skin. If the tube has to be re-inserted, the head of the patient must be extended to bring the trachea forward and out of the thorax, otherwise it may be impossible to find the opening. When a tube is to be removed and not replaced, it is advisable to do this in the morning rather than in the evening, because dyspnoea is always greater at night.

**LARYNGOFISSURE AND PARTIAL LARYNGECTOMY.**—The removal of a

tumour, usually malignant, is almost the only indication for laryngofissure, but it is occasionally performed for localised tuberculosis, the removal of foreign bodies and the relief of rare forms of laryngeal stenosis. Under local infiltration or chloroform anæsthesia, a preliminary tracheotomy is performed. The thyroid cartilage is divided in the middle line and the cricothyroid membrane opened. The inner perichondrium of the cartilage is raised by blunt dissection and the whole length of the vocal cord removed.

When the neoplasm occupies the anterior commissure, the operation may be modified by dividing the thyroid cartilage on either side of the middle line and removing the anterior portions of both cords with the overlying piece of cartilage. The larynx heals without any stenosis sufficient to cause symptoms, and the patient preserves a useful voice.

**TOTAL LARYNGECTOMY.**—In cases of intrinsic carcinomata too advanced for removal by the above operations total excision of the larynx is required. A large flap of skin, the base of which is at the level of the hyoid bone, is turned upwards. The larynx is freed from its external connections, and the pharynx is opened laterally. The pharyngeal mucous membrane is divided round the entrance to the larynx and dissected out of each pyriform fossa. The epiglottis should always be removed with the larynx. A feeding tube is inserted and the pharynx closed with catgut sutures and stitched to the base of the tongue. The skin flap is sutured to the edge of the trachea, and the rest of the wound closed with ample lateral drainage. Pharyngolaryngectomy is a modification of this operation, in which a part of the pharynx is removed with the larynx. The gap in the pharynx is closed at a later plastic operation.

**LATERAL PHARYNGOTOMY.**—The excision of tumours on the epiglottis, the aryepiglottic folds and the post-cricoid region requires two distinct steps. First, an anatomical dissection to expose the tumour, and second, its removal with a margin of at least half an inch of surrounding tissue. Access to the pharynx is provided by removing the ala of the thyroid cartilage and the great cornu of the hyoid bone. The tumour is excised and the cut margins of the pharynx are sutured to the skin. Perfect drainage is thus obtained, and the risk of bronchopneumonia is small. The pharyngostome must be closed by a plastic operation later.

The principal risks in these operations arise from streptococcal infection from the pharynx and from sloughing. They are minimised by drastic dental extraction at least ten days previously and by covering all raw surfaces with skin flaps. If serious infection does occur sulphanilamide is of great value.

LIONEL COLLEDGE.



## CHAPTER XXIV

### THE CHEST

#### INJURIES TO THE THORAX

**I**NJURIES to the chest-wall may be divided into two main groups : (1) non-penetrating injuries or contusions and (2) penetrating wounds.

#### NON-PENETRATING INJURIES

The severity or otherwise of these "crush injuries" to the intra-thoracic viscera depends upon several factors : (a) the force of the injury ; (b) its type, *e.g.*, whether sudden or gradual—in the former case the injury is likely to be more severe owing to inability of the thoracic viscera to adjust to sudden strains ; (c) the age of the patient—in young children the resilience of the chest-wall may result in little damage to it, with serious damage to the intrathoracic structures ; (d) the condition of the lung at the time of impact, whether distended or relaxed—in the former state pulmonary rupture is more frequent and extensive than when the lung is relaxed.

Severe intrathoracic injuries are usually associated with fractures of ribs, sternum, occasionally of the scapula and rarely of vertebræ, and result from crush injuries, falls from a height, run-over and other motor accidents, but it is of importance to realise that the absence of bone damage does not eliminate the possibility of injuries to soft tissues. In severe contusions of the lower chest-wall the liver, spleen and kidneys may be involved and complicate the diagnosis. Rupture of the diaphragm followed by herniation of the abdominal contents into the chest is by no means rare, although this condition has often been overlooked for long periods after the injury.

*Diagnosis.*—Three main conditions have to be considered : shock, hæmorrhage and air leakage either into the pleura—pneumothorax, or into the tissues—surgical emphysema. *Shock* unassociated with other complications should improve under treatment, and if this does not ensue, hæmorrhage should be suspected. *Hæmorrhage* may result from damage to the intercostal or internal mammary vessels, from the lacerated lung or from rupture of vessels at the hilum of the lung. In all cases there will be signs of a pleural effusion, and this may or may not be associated with blood-stained expectoration, but will always be accompanied by the general signs of internal hæmorrhage when the loss of blood is severe. Radiological examination is essential to accurate diagnosis.



**Pneumothorax** or the escape of air from the trachea, bronchi or lungs into the pleura may likewise be negligible or of serious import. It is more likely to be serious if the trachea or bronchi are damaged, as lung lacerations rarely leak air to any degree owing to the sealing off of the pleural communication by effusion of blood into the pulmonary tissue. On the other hand, lacerations of bronchi or trachea may give rise to extensive pneumothorax sufficient in degree to displace the mediastinum to the opposite side and seriously to embarrass both respiratory and cardio-vascular functions.

The most marked condition of pneumothorax is that associated with valvular openings in bronchi, which permit the egress of air, on inspiration, from the bronchus into the pleura, but in which the opening becomes closed on expiration. This is termed "tension pneumothorax" and, unless recognised by its symptoms of increasing dyspnoea and cyanosis and rapidly relieved, may result in the death of the patient.

**Surgical Emphysema** or the infiltration of the cellular tissues with air may result from laceration of lung adherent to the chest-wall. This is rarely of much concern and is usually localised, being diagnosed by the presence of a swelling associated with a crepitant sensation on pressure.

**Mediastinal Emphysema**, which results from laceration of the trachea or main bronchi, may be much more serious in its results from pressure, particularly on the larger veins in the mediastinum. It shows itself by spreading upwards to the head and neck, and may in severe cases become generalised over the whole body.

In the generalised type it is especially marked in the eyelids, which become so swollen as to close the lids, and in the lower parts of the body may lead to considerable distension of the scrotum and labia.

Late complications of non-penetrating injuries may be infected pleural effusions, abscess of the lung from infection of a hæmatoma and pneumonia.

More severe injuries occur but are so rapidly fatal as to be beyond treatment. Such consist in severe lacerations of the pulmonary hilum, even to the extent of complete division of this structure, lacerations of major vessels such as the aorta or venæ cavæ and extensive cardiac damage.

*Treatment.*—The first treatment as in all severe injuries is that of the accompanying shock. This can be controlled by warmth, morphine if pain is severe and the administration of intravenous plasma. Oxygen administered by the B.L.B. mask or the author's spectacle frame carrier is of great value in crush injuries. Hæmorrhage may necessitate blood transfusion. When dyspnoea is considerable a slightly sloping position head upwards, except in the severely shocked patient, is advisable.

Increasing dyspnoea will indicate tension pneumothorax and the withdrawal of air from the pleura, which may require repetition or the retention of the needle *in situ* for several days. All blood and fluid effused into the pleura should be removed after twenty-four hours, and if reaccumulation occurs further aspirations will be required. Unless this is done infection of the intrapleural fluid may occur and has been observed as late as sixty days after injury.

When double fracture of a number of ribs occurs, a condition termed "stove-in chest," paradoxical movement may occur, *i.e.*, the falling in of the free portion of the chest during inspiration and vice versa on expiration. It is associated with much shock and should be quickly controlled by overlapping layers of adhesive strapping extending over the midline anteriorly and posteriorly.

The treatment of associated injuries of less vital importance must not be neglected as soon as the urgent phase has been passed. Fractures of the sternum usually result in some deformity and displacement, the lower fragment being displaced upwards and forwards over the upper fragment. Marked extension of the back over a sandbag, extension of the arms above the head and pressure on the lower fragment will usually result in reduction, although there is a tendency for the deformity to recur. In such cases operation and fixation by suture may be necessary.

**Blast Injuries of the Lungs.**—Owing to the blast of exploding bombs, especially in confined areas, a wave of high positive pressure is transmitted to surrounding objects including individuals. The pressure will vary with the distance of the individual from the exploding bomb and will be affected by intervening objects, but it has produced in numerous cases a newly described injury which is termed "blast injury" and leads to definite changes in the thoracic cavity all of similar type but of varying degree.

*Pathology.*—The impact of the positive pressure wave on the chest-wall results in multiple hæmorrhages into the substance of the lung and into the subpleural areas of the chest-wall, especially of the intercostal spaces. In the more severe cases mediastinal hæmorrhages are present and blood may be effused into the pleural cavity.

Naked-eye examination of the lung shows areas of intense congestion resembling early lobar pneumonia. Microscopically there is evidence of excessive transudation of red cells, of rupture of the capillaries and of elastic tissue, and in the later stages of secondary infection with streptococci leading to broncho-pneumonia.

*Symptoms.*—The main symptoms are grave shock, prostration and restlessness with respiratory difficulty and pain in the chest. Cyanosis of greater or lesser degree is common and hæmoptysis may occur. When mediastinal hæmorrhages occur deep chest pain is present and often severe, whereas in cases of lesser severity the pain is referred to the chest-wall and abdomen. As inhalation anæsthesia may be fatal it is important to differentiate this condition from abdominal lesions. The chest is held fixed in the inspiratory position and breathing is shallow. Râles may be heard in one or both lungs.

*X-ray Examination.*—Heavy mottling appears early and may vary from a localised patch in one lung to diffuse generalised shadowing in both lungs. Alterations occur relatively rapidly in the X-ray appearances and in slight cases the lungs may appear normal in ten to twelve days.

*Treatment.*—Warmth, morphine administration and the use of high concentrations of oxygen are all essential. Slow transfusions of plasma are advisable in some cases, and owing to the tendency to

infection sulphapyridine should be given as soon as shock has passed off, and continued for several days.

### PENETRATING WOUNDS

The onset of war in 1939 has given a new importance to penetrating wounds of the chest and the alteration from the static conditions of the last war to the mobile conditions of the present will probably increase the relative incidence of chest wounds, owing to greater exposure of the body than in trench warfare. It was computed that during the last war wounds of the chest comprised 6.2 per cent. of the total number of casualties reaching hospital, 2.6 per cent. involving the pleural cavity or lungs; of men who died on the field 20 per cent. had wounds of the chest, but during attacks this figure rose to 40 per cent.

The causes of death were chiefly wounds of the heart and large vessels and open pneumothorax.

In the survivors who reach aid posts, field ambulances or advanced operating units, the causes of mortality are shock and hæmorrhage and later infection.

The severity of chest injuries is variable, depending partially upon the missile, partially upon the site. Thus a perforating bullet wound in the middle portion of the thorax is usually associated with relatively little shock and rapid recovery; on the other hand a tangential wound of the lower left thorax caused by a fragment of shell is usually associated with indriven rib fragments, laceration of the diaphragm, damage to the upper abdominal viscera and open pneumothorax (sucking-wound) and is of grave prognosis.

In general terms, to which there are numerous exceptions, bullet wounds have a better prognosis than fragments of bomb or shell and wounds of the middle third of the thorax than wounds of the upper or lower third.

Lacerated wounds with open pneumothorax have a serious outlook unless the opening is rapidly closed, and when treated conservatively in the early days of the last war the mortality was 90 per cent.

**Simple Penetrating or Perforating Wounds.**—In such cases the wounds are relatively small, the missile either perforates the chest or is retained and as soon as the mild shock has passed off physical signs in the chest may be slight or absent. In other cases blood may be effused into the pleural cavity and give rise to signs of an effusion of greater or lesser quantity.

*X-ray* examination may show a shadow in the lung due to hæmatoma at the site of perforation of the lung or that of an effusion at the base.

*Treatment.*—The usual treatment of mild shock is indicated—warmth, the control of pain by morphia and rest in the lying position. Careful observation for forty-eight hours is essential even if symptoms are mild. The wounds should be excised if contaminated, otherwise the surrounding skin should be cleaned, the wound dusted with sulphanilamide powder and a dry sterilised dressing applied.

**Lacerated Wounds with Open Pneumothorax.**—Wounds of this type vary considerably in size but the serious factor is the associated pneumothorax, which causes symptoms of severe shock and respiratory embarrassment. The bony chest-wall is almost invariably damaged, and during respiratory movements air mixed with blood is expelled from the pleural cavity and air sucked in.

*Signs and Symptoms.*—Shock is always present and is often severe. Dyspnoea, restlessness and cyanosis occur in varying degree, depending upon the size of the opening and the loss of blood.

Attempt at elucidation of physical signs in the chest is a waste of time as the condition is obvious on superficial examination.

*Treatment.*—This may be divided into two parts: (1) Emergency and (2) Operative.

### 1. EMERGENCY

The essential of treatment is to close the opening into the pleura at the earliest possible moment, and for this two methods are available. The simplest method is to cover the opening with a dressing—vaseline or waxed gauze is excellent—cover this with a larger piece of jaconet and fix with overlapping layers of adhesive strapping as for fractured ribs. The other method is to close the opening by the application of sutures through the muscular layers but excluding the skin. Dressings and strapping are applied as above. When sulphapyridine is available it is powdered into the wound before dressing or suturing. Improvement after closure of the opening is often dramatic, and upon admission into hospital resuscitation must be undertaken before complete operative treatment is considered. X-ray examination should precede operation.

### 2. OPERATIVE TREATMENT

Choice of anæsthetic is important. The best is gas and oxygen given by intratracheal tube, but intravenous anæsthesia by pentothal with oxygen delivered through a tight-fitting face-piece is satisfactory.

The essential point in operative treatment is the careful and methodical excision of the wound. This is performed layer by layer and will include the trimming of jagged rib ends. As soon as the chest-wall wound is excised instruments and gloves should be changed. The opening in the pleura should be enlarged when its position permits of good exposure of the intrathoracic contents. All blood effused into the pleura is removed by suction, if apparatus is available, or by careful swabbing, any large foreign body or rib splinters are removed from the lung and pleura, bleeding lung sutured and an intercostal tube inserted into the ninth intercostal space near the angle of the rib. The chest-wall is then carefully sutured in layers after powdering with sulphapyridine. The chest-wall should be firmly strapped after the application of dressings. Under-water drainage which will prevent the entrance of air into the pleura is essential and will prevent wound disruption. Drainage should *never* be carried out through the

wound. If pleural infection does not supervene the tube may be removed after forty-eight hours.

**Hæmothorax.**—Simple hæmothorax, *i.e.*, in which the bleeding into the pleura is the chief lesion, often occurs with relatively simple wounds of the chest-wall.

Hæmorrhage may arise from the vessels of the chest-wall, intercostals or internal mammary, from the lung tissue or the larger vessels of the hilum. In many cases in which the latter are involved death is relatively rapid.

The amount of blood may vary from a few ounces to 2 or 3 pints, and is soon diluted by fluid exudate from irritation of the pleura.

The effusion collapses and compresses the lung and tends to restrict bleeding from this source, hence in the larger hæmothoraces the chest-wall is more commonly the source. Blood retained in the pleura is very liable to infection, either from organisms carried in with the penetrating missile or from the passage of organisms from the damaged lung and bronchi or bronchioles.

As this blood forms an excellent medium for the growth of bacteria and as the gradual deposition of fibrin on the pleural walls may result in considerable eventual fibrosis, the blood should be removed as soon as possible. It generally remains fluid and the formation of large clots in the pleura is a sign of infection.

*Signs and Symptoms.*—The signs are similar in all respects to those of a simple pleural effusion associated maybe with those of hæmorrhage. If the blood is not removed, pyrexia up to  $101^{\circ}$  is accompanied by increased pulse rate; cyanosis and dyspnoea may be evident in the larger effusions.

*Radiological Signs.*—The usual picture is again similar to that of a simple pleural effusion, *i.e.*, shadowing of the base of the chest up to a varying height depending upon the extent of the bleeding—but extending higher in the axilla than elsewhere. If air is present from damage to the lung a fluid level will be shown varying with the position of the patient.

*Treatment.*—If later complications are to be avoided early aspiration (twenty-four to forty-eight hours) should be performed. At the first aspiration a portion of the blood may be replaced with air in order to evacuate as much of the blood as possible. Subsequently, the effusion should be aspirated at forty-eight-hour intervals until the pleura remains dry.

Should infection supervene open drainage should be avoided at all costs and aspiration continued according to the principles of treatment of infected effusions (see p. 472).

## INJURIES TO THE DIAPHRAGM

Injuries to the diaphragm are often unrecognised in the early stages, but later may give rise to symptoms such as intermittent strangulation of a herniated portion of stomach or intestine. The diagnosis is made on radiographic examination associated with barium ingestion.

## INJURY TO THE HEART

This is not infrequent in stab wounds and in such gives rise to definite symptoms of hæmorrhage into the pericardium if the patient survives. The obstruction to the heart's action, due to the increasing pressure, is termed "cardiac tamponade."

The chief symptoms described by Beck are (1) silent heart, *i.e.*, diminution of cardiac sounds, (2) steady fall in arterial blood pressure and (3) raising of venous pressure due to interference with the filling of the auricles.

At a later stage cerebral symptoms such as stupor, general rigidity with muscular twitchings and occasional hemiplegia may result from cerebral anoxæmia. If the injury to the chest is associated with head injuries difficulty may be encountered in deciding whether the cerebral symptoms are due to direct injury to the brain or secondary to the cardiac compression.

Radiographic examination will generally show enlargement of the cardiac shadow and fluid in the pleura.

*Treatment.*—Cardiac compression resulting from cardiac wounds indicated by the falling arterial pressure, rising venous pressure with damping of heart sounds and, where X-ray is possible, cardiac enlargement, call for immediate exploration of the pericardium and suture of the cardiac wound.

In all other cases expectant treatment is advisable in the early stages, followed later by aspiration of the blood-stained effusion and its bacteriological examination. In no case should drainage be established until the fluid, should it be infected, has become frankly purulent.

When foreign bodies are retained in the chest careful radiological localisation is necessary, and the question of their removal will depend upon the type. Smooth foreign bodies, such as revolver and rifle bullets, in the lung tissue rarely cause symptoms sufficient to warrant their removal. Irregular foreign bodies, on the other hand, almost invariably result in abscess formation either early or maybe months or years later. Thus it is advisable to remove the latter type after careful radiological localisation.

## SUPPURATIVE PLEURISY OR EMPYEMA

Infection of the pleural cavity by pyogenic organisms is relatively common, and is therefore of considerable importance. It is not proposed to consider tuberculous effusions here.

*Etiology.*—Empyema occurs at all ages but is most common in children. Two organisms are generally found in infective pleural effusions—pneumococci and streptococci. Staphylococci are rarely encountered, and when present may be associated with pyæmic abscesses in other parts of the body, *e.g.*, joints. *Bacillus coli* may be found, especially when the primary source of the empyema is below the diaphragm—subphrenic abscess. Gas-forming organisms are occasionally present when the pleural infection is secondary to an abscess of the lung or bronchiectasis.

*Source of the Infection.*—1. Pulmonary conditions. Lobar pneumonia, due to the pneumococcus, is still the most common precursor of empyema. The pleural infection results from direct spread to the subpleural spaces and hence into the pleural cavity. The infection is usually recognised after the crisis—metapneumonic. Broncho-pneumonia spreads in the same way and the streptococcal effusion becomes purulent in the early stages of the disease in the lung—synpneumonic—and is usually much more severe than the former type.

Abscess of the lung may rupture as the result of a severe attack of coughing and cause infection in the free pleural cavity. This is a serious complication, and the patient immediately becomes gravely ill. In other cases the pleural layers overlying the abscess become adherent, and a localised empyema slowly forms between them. Bronchiectasis may likewise spread infection to the pleura, in which case the effusion has a foetid odour from the presence of anaerobic organisms.

2. Non-pulmonary causes. Blood-borne infection is rarely, if ever, a primary cause of empyema. In such patients the formation of small subpleural abscesses in the lung, which rupture into the pleura, is the more likely source of pleural invasion.

Lymphatic spread. It has been suggested that in broncho-pneumonia followed by infective effusions in the pleura the spread is lymphatic, but there is not sufficient evidence to justify this assumption. Lymphatic spread of infection from the subphrenic region either in the case of subphrenic abscess or of amoebic abscess of the liver certainly occurs, and it is probably the mode of spread from infections of the œsophagus with or without malignant changes in that situation. In the late stages of œsophageal carcinoma spread of the growth and direct involvement of the pleura often result in a terminal empyema.

Direct infection by penetrating wounds of the chest may result in immediate infection of the pleura or a hæmorrhagic effusion in the pleura, due to non-penetrating chest injury, may subsequently become infected from the blood stream.

*Pathology.*—Two distinct types of effusion are encountered in the pleural cavity: (1) The localised abscess, *i.e.*, a collection of pus of varying amount enclosed within adhesions which may be termed the true empyema; (2) the generalised effusion which occurs in the free pleural cavity and which, in the early stages, is unassociated with any adhesion formation. The former type follows an attack of acute pneumococcal lobar pneumonia, while the latter is more commonly seen with other types of organisms—streptococci, anaerobes, etc, and is associated with broncho-pneumonia, rupture of pulmonary abscess or arises secondary to bronchiectasis. There are other characteristics of the two types—in the first group the pleural effusion generally follows the pneumonic attack, the underlying pulmonary condition having partially or completely resolved and the fluid aspirated is usually thick, creamy pus. In the latter the effusion forms early, generally during the active stage of the broncho-pneumonic process, and is found on aspiration to be serous, seropurulent or, in the presence of hæmolytic streptococci, heavily blood-stained.



Fibrin formation is generally a marked feature of pneumococcal empyemata, and large flakes are found in the pus associated with a fibrinous lining to the cavity. In the streptococcal and other types these are not so common, but in the foetid types necrotic tissue may be found in the fluid contents and many pockets may be formed.

*Symptoms and Signs.*—The formation of empyema should be suspected if, after the temperature has settled in pneumonia, there is a recrudescence of fever. In broncho-pneumonia an increase in the pyrexia denotes further spread of the pneumonic process in the lung or the formation of an infected pleural effusion. Severe pain is occasionally encountered, more particularly in the streptococcal cases, and, in large effusions, increase in the respiratory rate may be associated with increased cyanosis.

Physical signs of pleural effusion are fairly characteristic. Dullness on percussion is associated with considerable impairment of movement. In young people, especially children, bulging of the intercostal spaces may be seen and, if the effusion has been present for some time, it is accompanied by redness and oedema over a portion of this area.

Auscultation is usually associated with diminution or absence of breath sounds, but occasionally bronchial or tubular breathing may be heard, a feature more often encountered in children. Tactile fremitus in



FIG. 220

An acute empyema on the right side before drainage.

always diminished in effusions and is of value in helping to decide whether the dullness, especially when associated with bronchial breathing, is due to consolidation of the lung or effusions in the pleura.

Vocal resonance is usually diminished and ægophony often elicited over the upper level of the effusion. The displacement of the cardiac apex beat towards the opposite side is a sign of cardinal importance. Constitutional signs are soon in evidence, wasting, sweating and general weakness associated with a swinging temperature and raised pulse rate.

An increased leucocytosis of the polymorphonuclear type is always present, and a white cell count up to 30,000 with 80 to 84 per cent. of polymorphs relatively common. Secondary anæmia is usually present. The presence of albumen in small quantities associated with casts in the urine is often found.

*Radiological Signs.*—It is advisable in every case of suspected empyema, where possible, to make a radiological examination of the



chest. The findings are so characteristic that the presence and type of the empyema will be shown (Fig. 220).

*Diagnostic Aspiration.*—The diagnosis is completed by the result of aspiration of the pleura; as it is advisable to employ a needle with a reasonable bore for such a purpose, one should induce local anaesthesia by infiltrating the proposed track with 1 per cent. novocain.

A distinct sensation is felt by the operator's fingers as the needle enters a fluid collection, and the piston is then withdrawn. If no fluid enters the syringe it is advisable to ensure that the needle has not become blocked either during its transit through the chest-wall or by a piece of fibrin in the empyema cavity. The latter is quite a common occurrence in pneumococcal empyemata where fibrin formation is usually abundant and sometimes leads to the erroneous view that the empyema is a small one if the needle gets blocked after a small quantity of pus is withdrawn.

*Complications.* 1. **Bilateral Empyemata.**—The involvement of the second pleural cavity is comparatively rare, but as would be expected is more frequent following broncho-pneumonia.

2. **Pericarditis.**—This complication is likewise more common with streptococcal infections than with those due to the pneumococcus. It may commence as part of a generalised infection synchronous with the pleural and pulmonary infection, or may arise as a result of spread of the infective process either directly or via the lymphatics from the pleura into the pericardium.

3. **Peritonitis.**—This complication occurs with both streptococcal and pneumococcal infections, but in a much higher proportion of the former type. In both types it is particularly fatal, and often is evidence of complete failure of resistance and generalised spread of the infection throughout the body.

In the subacute and chronic empyemata further complications may be encountered, viz. :

4. **Empyema Necessitatis.**—Perforation through the intercostal spaces and "pointing" of the empyema in the superficial tissues rarely occurs until the empyema has been present for several weeks, if not months. Unless this condition is borne in mind it may be mistaken for a superficial abscess in the chest-wall, and even after incision its true origin remains unsuspected owing to tortuosity of the sinus into the pleural cavity. Physical and radiological examination of the chest will make the diagnosis clear. Adequate drainage of the empyema, usually posteriorly, is required, as it rarely "points" at the most suitable area for drainage.

5. **Bronchial Fistula** occasionally complicates the acute pleural infection, especially when this has followed rupture of an abscess of the lung or a bronchiectatic cavity. In other long-standing empyemata sudden rupture of the pus into the lung associated with the expectoration of a quantity of pus may be the origin of a bronchial fistula.

6. **Meningitis** occasionally complicates acute pleural suppuration, but cerebral abscess is more usually associated with the chronic variety.

*Treatment.*—It has been shown by practical experience, and is to be expected from experimental observation, that the production of an open pneumothorax is a considerable strain in even the relatively normal individual. It necessarily follows that such disability is much greater if other toxic factors are already present.

The respiratory reserve of a normal individual is considerable, the vital capacity being on the average about seven or eight times the tidal air. All toxic conditions will invariably lower the vital capacity, and if an open pneumothorax is now produced by operative measures the danger point when the vital capacity is lowered to the limits of tidal air requirements may be reached. The practical effect of such considerations is seen when a pyogenic effusion is found in the pleural cavity, especially when the pulmonary lesion is still active and before pleural adhesions have formed.

The correct treatment in each case will therefore be determined by a consideration of the pathological condition in the chest, the nature of the fluid withdrawn by aspiration of the pleura being used as a guide. It should be pointed out in this connection that surgical procedures in the narrow sense are never urgently necessary in these cases. When the actual bulk of the pleural effusion itself appears to be an urgent factor, this can be relieved by aspiration of a sufficient quantity of fluid to relieve pressure symptoms, and thus time will be available for consideration of the findings from diagnostic aspiration. In general three types of fluid may be withdrawn: (1) Thin, serous or turbid or blood-stained fluid without odour; (2) thin, turbid, foul-smelling fluid; and (3) definite creamy pus.

In the two former groups the relatively small quantity of pus present in the fluid is evidence of the fact that localising adhesions have not yet formed. It corresponds to the period of diffuse cellulitis in soft tissues which occurs before the formation of the localised abscess. If the pleura is opened in this stage a condition of open pneumothorax is produced with its attendant disadvantages, failure to realise which was responsible for an operative mortality varying from 60 to 75 per cent in the influenza epidemic in 1918. The effusions of these groups when bacteriologically examined are found to contain streptococci, and particularly in the second group anaerobic organisms, often gas-forming, spirochaetes or spirilla. In the most acute forms almost pure blood may be withdrawn, which on examination is found to be swarming with hæmolytic streptococci.

It will be obvious from the foregoing remarks that other methods than open drainage should be adopted for the cases in Groups 1 and 2.

**Treatment of Group 1.**—It has been found that repeated aspirations will result in the fluid becoming steadily more purulent. They should be performed at intervals of twenty-four or forty-eight hours according to the rapidity of reaccumulation in the individual case. If a portion of fluid is put up on each occasion in a test-tube the relative proportion of pus present can be seen after settling. As soon as the fluid becomes frankly purulent, *i.e.*, seven-eighths sediment after standing in test-tube for twenty-four hours, treatment can be adopted as for Group 3.

**Group 2.**—There is a great tendency, during aspiration of foul anaerobic effusions, for slight leakage along the needle track to give rise to widespread gangrenous cellulitis of the chest-wall.

With the hope of preventing this, the author has for several years carried out the following modification with satisfactory results. As soon as the presence and nature of the effusion is determined, a vertical incision down to the ribs is made under local anaesthesia over the area where drainage is eventually to be established. This area is packed firmly with paraffin flavine gauze and a superficial dressing applied. When the next aspiration is required the gauze packing is removed, the aspiration carried out and a new pack inserted. This has two advantages, (1) the aspiration is less painful and does not require any local anaesthesia (repeated aspirations through unbroken skin are painful) and (2) a barrier of granulation tissue forms which prevents spread of infection from the area of aspiration into the tissues of the back.

*Intercostal Drainage* may be instituted after two or three aspirations and the further necessary aspirations carried out through the tube. In adults, although intercostal drainage or even repeated aspiration may result in cure of a small proportion of cases, it is inadvisable to rely on them entirely, as in each case drainage is rarely adequate owing to the presence of fibrin masses or necrotic tissue too large for removal by these means. Further, there is a distinct danger of producing a chronic empyema unless adequate drainage is established later. The temptation to be satisfied with intercostal drainage is great when the temperature and pulse have settled satisfactorily and the patient feels well, but the formation of a chronic empyema pocket is common, unless adequate drainage by rib resection is carried out later. Children and especially infants can, however, often be cured by intercostal drainage alone, and it is generally advisable to establish such drainage in infants and to leave rib resection until its necessity is obvious.

**Group 3.** *Rib Resection and Drainage.*—When patients in the two first groups have been treated on the lines stated above and the fluid withdrawn is definitely purulent, rib resection is indicated. In Group 3, which is generally due to the pneumococcus, thick creamy pus is usually withdrawn at the first aspiration and the pneumonic process in the lung has usually settled, and it is advisable to proceed to treat it by adequate drainage. An exception to this rule should be made when the amount of the pus in the pleura is excessive, an indication of which is considerable cardiac displacement and dyspnoea. Sudden decompression, which will result from operation, may result in cedema of the lung and cardiovascular disturbances, and it will be less dangerous to withdraw a reasonable amount, for example about 15 oz., and defer drainage for twenty-four or forty-eight hours.

Local anaesthesia is not only satisfactory and advisable, but in many seriously ill patients essential to recovery. In the localised atypically situated empyemata it is necessary to plan the incision over the area involved, but in the usual basal empyema the incision should be made posteriorly. The use of a vertical incision has advantages over the oblique one in that there is a tendency to

retention of infection in the lower lip of the oblique incision, and the vertical incision will also allow resection of a lower rib than was originally intended if it is found desirable.

If the portion of rib resected, which need be no longer than 1 to 1½ in., is removed at the angle it will result in drainage of the area of the costovertebral groove, an area in which a residual empyema is likely to form. The 9th rib is usually chosen, as the drainage through any lower opening is liable to be obstructed by the rise in the diaphragm which takes place in the ensuing few days.

In addition to adequate drainage there are three other requisites: (1) suction drainage, *i.e.*, the production of negative pressure within the empyema cavity; (2) irrigation; and (3) breathing exercises.

**Negative Pressure Drainage.**—This can be maintained by the use of flanged tubes for drainage of the chest and the continuous exhaustion of air from the drainage bottle. Although some doubt is cast upon the value of this procedure, as it can be carried out with little inconvenience, it should be employed (Fig. 221).

**Irrigation.**—The removal of debris and any stagnating pus can do nothing but good in any infected cavity, and the value of irrigation is probably more due to actual lavage rather than any antiseptic value of the fluid used. Care must be taken to exclude the presence of a bronchial fistula and to ensure that the outlet from the empyema cavity during irrigation is larger than the actual irrigating tube.

**Respiratory Exercises.**—The value of forced breathing exercises, such as is entailed by expiration against pressure, has probably been greatly exaggerated. All exercises, however, which encourage the use of the diaphragm and the lower intercostal musculature are of the utmost value, and should be commenced as soon as the acute phase is passed.

**Removal of Tube.**—An empyema is not healed until the two pleural surfaces are adherent and

the cavity completely obliterated. It has been advised that the tube should be removed as soon as the discharge becomes serous, regardless of the obliteration of the cavity. In some of these patients the superficial wound heals, the air in the remaining pleural pocket is gradually absorbed and complete healing of the empyema results. In others an air pocket remains, and in the course of weeks, months or maybe years a recurrent empyema results. In a certain proportion of patients the superficial sinus refuses to heal and they pass into the chronic stage. If treatment is carried on and the tube not removed until obliteration of the cavity is complete,

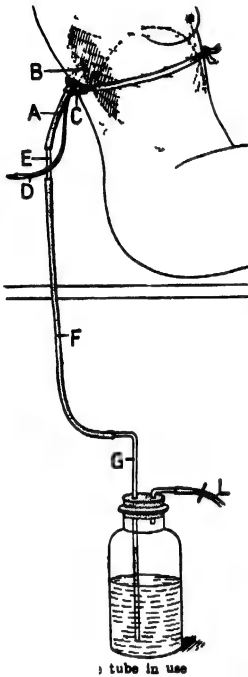


FIG 221

The author's method of negative pressure drainage.

A, the main drainage tube; B, its internal flange; C, its external flange; D, lateral catheter for irrigation; E, F, and G, glass and rubber connections to bottle, maintained at negative pressure via tube L.

as shown by filling with an opaque fluid and then radiographing the chest, recurrent empyema is extremely unlikely to result in the future. The treatment of the acute empyema under these circumstances will be more prolonged but more certain in its results.

*Prognosis* will depend upon several factors, such as age, primary infection and the occurrence of complications. Infancy and old age both show increased mortality in all groups, and it is recognised that empyemata due to the streptococcal infections are more virulent than those due to the pneumococcus. It is an interesting fact that the statistics show that the mortality from empyema follows closely year by year that due to the pneumonia itself, each being virulent one year and perhaps mild the following year.

This mortality is likewise associated with complications which are more frequent in the more severe pneumonias than the less severe ones.

It is probable that the mortality rate associated with streptococcal broncho-pneumonias is higher than it should be, owing to the better surgical judgment required in these cases, particularly as to when to temporise by aspiration and when to drain.

**Sulphapyridine**, which will have been given in the acute stages of the lung infection, should be continued in the acute stages of the pleural infection.

### CHRONIC EMPYEMA

Chronic empyema is generally the result of incomplete treatment or non-diagnosis of the acute condition. Other causes of chronic empyema are unrecognised tuberculosis or actinomycosis of the pleura or the unsuspected presence of malignant disease in the underlying lung.

In chronic empyema due to the common pyogenic organisms four types may be recognised: (1) chronic undrained empyema; (2) chronic empyema with an external sinus; (3) chronic empyema with bronchial fistula; (4) chronic empyema with external sinus and bronchial fistula.

**The Chronic Undrained Empyema** is by no means rare. The gross thickening of the pleura greatly limits the absorption of toxins. The symptoms will necessarily vary, but the most constant are chronic non-productive cough, pain in the chest, feeling of malaise and clubbing of fingers. A slight degree of pyrexia may be present in some cases, in others the temperature may be normal and only raised from time to time. In many cases emaciation and sweating are prominent features, and in younger patients flattening of the chest and scoliosis can be observed. Associated with evidence of fluid in the pleura a considerable degree of leucocytosis is found. Radiography and exploratory aspiration will confirm the diagnosis.

**Chronic Empyema Cavity with External Sinus** is probably the most common type. It is often associated with the presence of foreign bodies, most commonly small rib sequestra, but occasionally portions of rubber tubing and gauze. However, this type usually results from inadequate drainage of the acute and subacute types or follows the too early drainage of the streptococcal variety. The

sinus may be quite small, a regular daily discharge of from 1 dr. to 1 oz. or more of pus being found on the dressings, but occasionally the sinus may heal and discharge intermittently. When examined radiologically these cases show either an area of increased density due to pleural thickening or a cavity partially filled with fluid and air above. If lipiodol is introduced into the sinus the outlines of the cavity are demarcated (Fig. 222).

**Chronic Cavity with Bronchial Fistula.**—Such cases result from rupture of a chronic undrained empyema into the lung and thence into the bronchi. Patients with such a condition have a history of expectoration of a large quantity of pus, which is followed by relief of the more urgent symptoms associated with the chronic undrained type. Expectoration of pus, although in smaller amounts, continues until treatment is instituted. The condition is occasionally confused with the rupture of a pulmonary abscess, but radiography will generally enable a diagnosis to be made.



FIG. 222

The X-ray appearance of a chronic empyema with external sinus after injection with lipiodol.

**Chronic Empyema with External Sinus and Bronchial Fistula.**—These cases result from drainage of a chronic empyema which has already ruptured into the lung, particularly after drainage of the former group. The introduction of radio-opaque fluid into such cavities results in its leakage into the bronchi on the same side and, occasionally, on the opposite side. The bronchial opening, of

course, varies in size considerably in different cases. This type is also found following drainage of a pulmonary abscess which has ruptured acutely into the pleura, or in which localised empyema has followed its slow penetration into the adherent pleura.

Such complications as empyema necessitatis and cerebral abscess are much more common in the chronic varieties of empyema than they are in the acute. Clubbing of fingers and osteo-arthritis are also seen in all varieties of chronic empyema. Complications such as gross pulmonary fibrosis, with or without bronchiectasis, may be a late sequela.

**Diagnosis.**—The first essential before considering treatment is full investigation of the type of empyema present. In the closed variety this will entail, of course, the use of exploratory aspiration to confirm the diagnosis, whereas in the other varieties the extent and outlines of the cavity can be delineated by the introduction of lipiodol,

either through the sinus or through the trachea, followed by X-ray examination from several angles. In this way the position of the cavity can be accurately determined and treatment decided upon.

The intratracheal introduction of lipiodol will often result in a portion of the opaque fluid passing into the empyema cavity through the bronchial fistula, although the fact that this does not happen is no proof that such a condition is not present.

*Treatment.*—Methods of treatment to be adopted will depend to some degree upon circumstances, but the principles enunciated in the consideration of treatment of the acute variety should likewise be adopted here. It is therefore probable that every case of chronic empyema will primarily require adequate drainage which may, or may not, be possible through the old sinus, when present. Associated with this, irrigation and respiratory exercises are of considerable importance, but in chronic empyema relatively high negative pressure drainage is of great advantage. No major surgical procedure should be undertaken until such treatment has been carried out over a long period, during which the size of the cavity remains stationary. It is advisable, therefore, to carry out routine filling of the cavity with radio-opaque material at intervals of three weeks to determine the results of treatment. So long as the cavity is getting smaller this treatment is persevered with, and it is only when two or three successive radiological examinations show the cavity to be stationary in size that other surgical procedures should be undertaken.

*Further Surgical Procedures.*—These are of two types, (a) decortication of the lung by multiple incisions in the thickened visceral pleura, and (b) plastic operation on the chest-wall, designed to permit the falling in of the chest-wall by the removal of as many ribs as is necessary. The latter procedure can be done in one or more stages, and, although the time entailed may be long, the results are excellent (cf. Fig. 229, B).

## PULMONARY SUPPURATION

Pulmonary suppuration may disclose itself as one of several conditions—abscess, gangrene, diffuse suppurative pneumonitis—and should include such conditions as bronchiectasis in which the parenchymatous portions of the lung are often involved. It will be advisable, however, to describe these main conditions as separate entities with a realisation that often they are associated; for instance, in chronic abscess of the lung it is almost invariable to find a greater or lesser degree of associated bronchiectasis present in the same pulmonary lobe.

## PULMONARY ABSCESS

The term pulmonary abscess denotes the formation in the parenchyma of the lung of a localised suppurative lesion. It is preceded by a more diffuse involvement of a certain area of the lung tissue in an infective process, best termed pneumonitis, which later becomes more localised by the breaking down of the central portion and the



formation of pus, surrounded by a pyogenic membrane. The pathological process is closely analogous to the condition of infective cellulitis, followed by local abscess formation in other soft tissues of the body. However, there are other factors concerned, more especially the communication sooner or later with the bronchial system and the great vascularity of the pulmonary tissue. More accurate diagnosis has been responsible in recent years for the apparent increased frequency of this condition in all countries.

*Causation.* 1. **Aspiration of Infected Material.**—This is probably the most common cause of pulmonary abscess, as infection of and around teeth, in the tonsils and sinuses of the nose is relatively frequent. It is also occasionally responsible for those cases which arise after operation, not only upon the upper air passages but upon other parts of the body, *e.g.*, abdomen. Obviously the inhalation of foreign bodies such as pieces of a tooth or bone during mastication, or pea-nuts and small metal toys by children may result in abscess formation.

2. **Embolism.**—Suppuration may occur subsequent to infarction of the lung, the infection spreading into the infarct from the bronchial tubes. In other cases infective emboli will result in the formation of one or more abscesses in the lung, which may be widespread and fatal or localised to a relatively small area of lung tissue, finally coalescing to form a single multiloculated abscess.

3. **Pneumonia.**—It is doubtful whether pneumonia is so common a precursor of pulmonary abscess as was formerly thought to be the case. Certainly, lobar pneumococcal pneumonia is rarely complicated by pulmonary abscess, and in broncho-pneumonia due to streptococcal infection, when abscess formation results, the foci are generally small and multiple and are rarely of the type that is amenable to surgical treatment. The stress laid upon pneumonia as a prior cause in earlier descriptions probably results from the fact that the formation of a pulmonary abscess is necessarily preceded by a larger area of pneumonitis in the lung, which causes similar physical signs to those recognised as due to pneumonia.

4. **Carcinoma of Lung.**—Two types of pulmonary abscess are encountered in association with carcinoma. The first occurs as a result of carcinoma of one of the larger bronchi, which becomes obstructed. The pent-up secretions, if infected, will then tend to cause ulceration in the bronchi and pulmonary tissue. In such cases the walls of the abscess cavity do not consist of breaking-down growth. In the second type the abscess results from degeneration of the central portions of a carcinoma, arising in the bronchioles, and hence the walls themselves consist of new growth. The former type is often regarded as a pyogenic abscess and drained as such, and its true nature is only later recognised.

5. **Direct Spread.**—Hepatic amœbic abscess, subphrenic abscess and rarely perirenal abscess may penetrate the diaphragm and, when the lung is adherent, burrow into the lower lobe, forming an abscess which is eventually expectorated following secondary rupture into a bronchus. Occasionally in the late stages of carcinoma of the



œsophagus, following the formation of adhesions, direct ulceration into the lung occurs with formation of a pulmonary abscess.

6. **Injuries.**—Severe, non-penetrating contusions of the chest may, especially in younger subjects, result in the formation of a hæmatoma in the pulmonary tissue, which later becomes infected from the blood stream. Penetrating injuries likewise may convey infection directly into the pulmonary tissues. Suppuration is relatively rare owing to the great vascularity of the lung tissue. Missiles, such as shell fragments or bullets, may cause pulmonary abscess many years after they have apparently been “sealed off” in the lung tissue.

7. **Specific Infections.**—Actinomycosis may be the cause of a pulmonary abscess, and occasionally infection of a hydatid cyst results from rupture into the bronchial tree with secondary infection of the cyst cavity.

*Pathology.* **Bacteriology.**—The rôle of the various organisms found in the sputum of patients suffering from pulmonary abscess is still somewhat ill-defined. Likewise the pus obtained direct from such abscesses is commonly found to contain several different types of organisms. More recently the frequency of occurrence of anaerobic organisms has been recognised generally in association with streptococci and occasionally staphylococci. The anaerobic organisms almost constantly present in foetid pulmonary abscesses are often found in the mouth, tonsillar crypts and other parts of the upper air passages. It is also of significance that various types of spirilla and spirochætes are frequently present in foetid lung abscess.

**Naked-eye Appearances.**—In the early acute stage of pulmonary abscess, the lung shows a localised area of consolidation, œdema and often extravasation of blood. At a later stage the centre liquefies and discharges partially through the bronchus, resulting in a cavity varying widely in size, having a wall composed of necrotic pulmonary tissue and a surrounding area of consolidated lung. On occasions, minute secondary abscesses may be seen in the surrounding consolidated zone and the cavity found to contain a semi-solid, foul-smelling, yellowish material, similar to that occasionally seen in the tonsillar crypts. Strands of tissue are seen traversing the cavity, and these often contain blood vessels which may be thrombosed.

In the chronic abscess the walls will be more fibrous in type, and a considerable degree of fibrosis be present in the surrounding pulmonary tissue. When the abscess is of long standing, its walls may be completely epithelialised and a definite degree of bronchiectasis found in the adjacent bronchi.

In both types one or more bronchi open into the abscess cavity.

*Symptoms.*—In the early stages the symptoms consist in fever, cough, raised pulse rate, occasionally pain in the chest, rigors and sweating. Expectoration is usually absent until the abscess ruptures into the bronchus. When this occurs there is often some degree of hæmoptysis associated with the expectoration of a quantity of pus, which is often foul-smelling and tasting. Following this there is a rapid fall in the temperature and pulse, and an amelioration of the general condition of the patient. In many cases there occurs, however,

re-collection of the pus with recrudescence of symptoms followed by another copious discharge of purulent expectoration. This may be termed the "intermittent discharging" abscess.

Another type is encountered in which there is a more continuous expectoration of purulent sputum, and in such cases the constitutional symptoms are less marked. This type is often associated with clubbing of the fingers and other signs of chronic absorption of toxins from a septic focus, and may be mistaken for bronchiectasis.

Physical signs in pulmonary abscess are unreliable. In the early stages a diagnosis of pneumonia is not uncommon as the signs are often similar. Occasional râles and crepitations may be heard, and

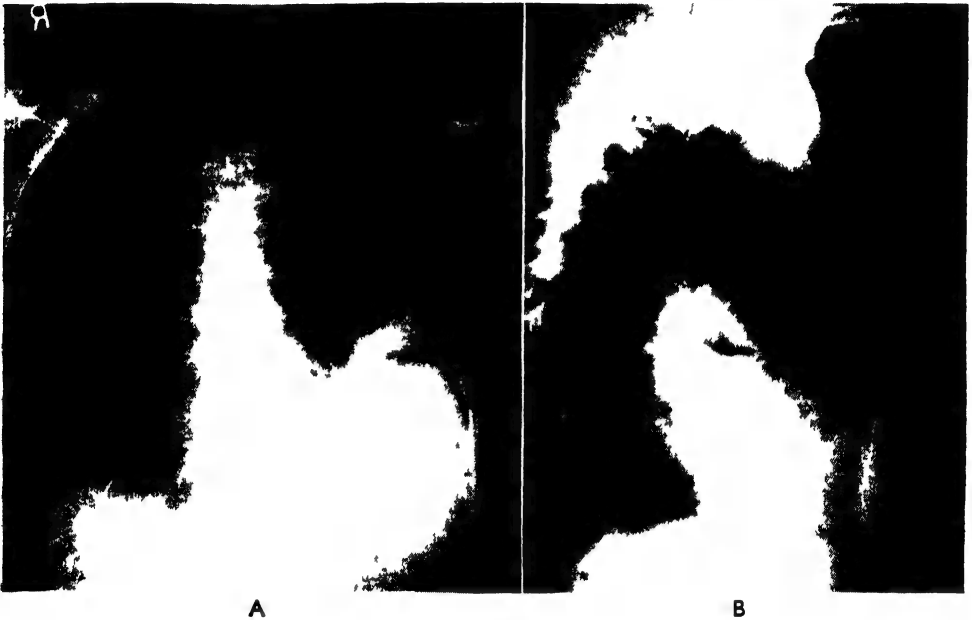


FIG. 223

A, anteroposterior view. B, lateral view of left lung abscess.

Note the fluid level, especially in B.

localised tenderness can occasionally be elicited by finger pressure over the site of the abscess.

A leucocytosis with a polymorphonuclear predominance is relatively common, but its absence does not exclude the diagnosis. Anæmia is usually present.

The quantity of sputum does not depend upon the size of the abscess cavity, often being greatest when the cavity is relatively small. A large proportion of the expectoration arises from irritation of the mucous membrane of the bronchi by the contents of the abscess. Colour may be yellow or greenish but it is often altered by the blood content into brown or red. In most cases of pulmonary abscess the sputum is foul-smelling at some time or other, but occasionally is so small in quantity that fœtor of breath may be the predominating symptom. Microscopic examination of the sediment from sputum left standing usually discloses the presence of small portions of necrotic lung or elastic fibres, especially when the abscess is extending.

**Radiological Examination.**—Pulmonary abscess before rupture of the contents into the bronchial tree gives rise to a rounded or triangular shadow in the translucent lung field. In the latter case the base of the triangle is towards the periphery of the lung, and in both cases the shadow is most dense in its central portion, gradually fading off to normal lung tissue. As soon as rupture occurs the central portion may show a fluid level with air above. This fluid level alters with the position of the patient (Fig. 223).

The majority of pulmonary abscesses are situated peripherally in the lung, but radiograms in various directions are essential in order to establish accurate localisation.

**Aspiration.**—This should never be carried out in suspected cases of pulmonary abscess. Should the pleural layers be non-adherent it will result in infection of the pleural cavity—a serious complication.

**Treatment.**—As in all acute respiratory infections rest in bed associated with good nursing and nourishing food are essential.

**Postural Drainage.**—This is the most effective non-operative measure in the treatment of the acute abscess after bronchial rupture and is often effective in the early chronic abscess. In a proportion of cases, probably in the neighbourhood of 25 per cent., cure will result from the use of this procedure, and even in the chronic cases great improvement may follow, which will make later operation safer.

It is essential, however, that the treatment should be carried out adequately, and in most cases this will require the use of a postural bed to enable the patient to be kept in the optimum position for drainage for considerable periods without undue fatigue.

The optimum position is the one in which cough is most readily induced, and cannot always be determined by the position of the abscess in the lung field.

**Drugs.**—The presence of spirochaetes in many lung abscesses has led to the use of arsenic in treatment. It appears to lead to definite improvement of the foetor, but only rarely cures the abscess without other measures. Likewise emetine has been used, especially in France, with only occasional success unless the abscess is amœbic in origin. There is a tendency, except in the gangrenous type, for the abscess to pass from the acute into the subacute and chronic stage and it is therefore essential not to regard improvement in the constitutional condition as necessarily associated with disappearance of the abscess. The progress of every patient should be controlled by regular radiological examination, and after six to eight weeks, if the radiological picture is not showing very considerable and steady improvement, alternative forms of treatment should be considered. The disappointment associated with surgical treatment many years ago was largely due to delay in its institution, with the result that the patient was very gravely ill or the abscess had passed into a chronic stage with secondary changes in the surrounding lung and bronchi before operation was undertaken.

**Artificial Pneumothorax.**—The introduction of air into the pleural cavity will result in compression of the lung and, theoretically, further evacuation of pus retained in the abscess cavity in the lung. There have been undoubted successes, but there are very definite dangers in its use. Not uncommonly, adhesions overlying the abscess cavity in the lung are torn and the pleural cavity is suddenly flooded with the abscess contents,

which condition may lead to rapid death. In other cases, compression of the lung may interfere with drainage of the abscess by kinking and obstruction of the communicating bronchus, and result in spread of the infective process in the lung.

**Bronchoscopy.**—There is no doubt that bronchoscopic examination should be carried out in every case in which the aspiration of a foreign body is suspected. Some authorities claim a high percentage of success in the cure of pulmonary abscess by bronchoscopic aspiration.

The *treatment* consists in the aspiration of pus from the lobar and secondary bronchi, and the destruction of any visible granulations obstructing drainage. It is rarely, if ever, possible to introduce the aspirating tube directly into the abscess cavity.

Injection with antiseptic solution is of doubtful value and considerably increases the risk of spread of the disease to other parts of the lobe or lung.

Bronchoscopic aspiration should generally be used after a trial of postural drainage, but if it does not produce reasonably rapid improvement should not be continued until the condition becomes chronic.

**Phrenicectomy and Thoracoplasty.**—The end results of these two procedures are not sufficiently good to justify their use.

**Pneumonotomy and Drainage.**—The external drainage of pulmonary abscess is the treatment of choice when medical measures and bronchoscopy have failed to cure the condition. There is increasing evidence in favour of early operation for the "fœtid" abscess. The main essential before operation is the most careful localisation of the abscess. Operation is advisable under local anæsthesia in order to prevent flooding of other portions of the lung when relaxation takes place. The operation should generally be done in two stages, the first stage with the intention of producing adhesions, if not already present and firm, between the two layers of pleura. It entails resection of portions of two ribs and the intercostal structures overlying the most superficial portion of the abscess. The parietal pleura is not incised unless it is firm and fibrous, but a strip of gauze, wrung out in iodine and containing a small metal ring, is placed *in situ* and the superficial tissues sutured without drainage.

After an interval of seven to fourteen days and further radiological examination the wound is reopened, the pack removed and an aspirating needle inserted into the subjacent lung. When pus or foul gas is aspirated the abscess is opened alongside the needle by diathermic cautery.

In a superficial abscess the whole of the outer wall is excised and the cavity "saucerised"; in deeper abscesses a large opening is made, and in either case the cavity is packed with gauze and the superficial tissues left unsutured. At a later stage, drainage by soft-walled tube is instituted, and this is maintained until the cavity is obliterated and only a tube track and residual bronchial fistula remain, when the tube may be shortened and later removed.

**Lobectomy.**—In old-standing chronic abscess, which is always associated with a greater or lesser degree of bronchiectasis, the only hope of complete cure may depend upon removal of the affected pulmonary lobe.

### PULMONARY GANGRENE

Gangrene of the lung, as in other situations, denotes massive death. It depends upon several factors, the chief being infection with very virulent organisms in an individual with lowered resistance, and associated therewith may be thrombosis of blood vessels. In the lung

it is always of the moist type. Some authors describe the foetid abscess as gangrene in addition to the more massive deaths of lobe or whole lung, generally designated pulmonary gangrene.

*Pathology.*—Apart from the intensity of the infection the causes are similar to those of pulmonary abscess. The rapid spread of the gangrenous process leads to early and intense infection of the pleura, and portions of the lung slough off, and may be found in the pleural exudate, which resembles thin muddy liquid most foul in odour.

*Symptoms.*—The onset is often sudden, associated with symptoms of an intense general infection, rigors, high temperature and rapid pulse. Dyspnoea, cyanosis, expectoration of foul sputum and most penetrating foetor of breath are characteristic symptoms.

*Signs.*—Multiple rhonchi and râles can be elicited over the affected lung on auscultation. Dullness at the pulmonary base on percussion occurs relatively early as fluid forms, and the high-pitched resonance of pneumothorax is often found in the upper part of the chest in front and behind. This pneumothorax is produced by leakage of air from the gangrenous lung and the presence of gas-forming organisms.

*Treatment.*—This consists in the use of all medical measures, including blood transfusion and an oxygen tent, directed to the maintenance of the strength of the patient. Drainage of the pleura should be carried out at first by means of the introduction of an intercostal tube and later by rib resection. The prognosis is always bad. If the patient recovers sufficiently it may be necessary to remove a slough from the pleural cavity later.

### BRONCHIECTASIS

This condition denotes dilatation of the bronchial tubes with or without superadded infection.

There are various theories about its causation, such as traction on the tubes from fibrosis of the lung, nutritional changes in the bronchial walls, forced inspiration and prolonged cough, this being forced expiration against a closed glottis. Other factors concerned are bronchial obstruction due to foreign bodies, intrabronchial neoplasms, pressure of tumours outside the bronchi, previous pulmonary abscess and lastly congenital malformation.

Congenital bronchiectasis is now recognised as much more common than formerly. The dilatations are so considerable, often in quite young infants, and symptoms are so minimal that there can be little doubt that they have been present since birth. Upon a developmental basis it has been suggested (Sauerbruch) that a bronchus may be "tied off" by the enveloping pleura. If a large bronchus is involved a large cyst of lung should result, but if the process is delayed until late in the developmental phase, multiple small cysts or congenital bronchiectasis may eventuate. Primary infection of the congenital condition may follow whooping-cough, measles, etc., during infancy. This group is really related to true cystic disease of the lung (Fig. 224).

In congenital atelectasis with bronchiectasis the latter condition is probably secondary. The lobe is often lacking in pigment, and the most attractive theory of its development is that it results from the tendency of the bronchi to dilate during inspiration in a portion of the lung, which is not taking its full part in the normal chest movements. In the adult types many factors take part, such as foreign bodies or tumours in the bronchi, chronic bronchitis with changes in the bronchial walls—especially loss of elastic tissue—syphilis, pulmonary fibrosis and abscess, tuberculosis and certain extrapulmonary factors such as enlarged lymph glands, mediastinal tumours, aneurysms, empyema and bronchopleural fistula.

*Pathology (Schneider's Classification).—A.* Atrophic bronchiectasis, sometimes called cylindrical, in which the dilatation extends through-



FIG. 224

A congenital cystic lung on the right side, with displacement of the heart to the right. Three tomograph views, A and B showing the multiple cystic appearance, and B and C, at a deeper level, the apex of the left upper lobe which has prolapsed across the midline and fills the upper part of the right side of the chest.

out the whole bronchus. The epithelium may be normal, the elastic tissue fibrillated and the cartilage destroyed. Cellular infiltration is absent, there is no laying down of new connective tissue, and the bronchial muscles are not hypertrophied. *B.* Atrophic bronchiectasis with inflammatory change in the bronchial wall. This type resembles the former, except that inflammatory changes can be seen in the subepithelial layers and the lung may show induration. *C.* Sclerosing bronchiectasis shows fibrosis in the bronchi. *D.* In saccular bronchiectasis there are two types, in one of which the cavities have retained their elastic tissue, while in the other there are rigid walls.

In all these types pleural adhesions are present over the affected areas in about 60 to 70 per cent., being somewhat more frequent in the sacculated than in the cylindrical type.

The disease is essentially more common in the lower lobes of the lungs and is usually unilateral. There is little difference between the incidence of bronchiectasis in the two sexes, and the great majority are diagnosed between the ages of 20 and 40 years. Many patients, however, give a history dating several years and recently children are being diagnosed as sufferers from it.

*Symptoms.*—Cough and expectoration are characteristic of the disease, the sputum varying in quantity from a few ounces to over a pint in the twenty-four hours; it generally increases in the winter and is commonly foetid. On standing, the sputum separates into three layers, the upper consisting of greenish yellow pus, mucus and air bubbles, the middle being thinner and containing less air, while the third layer contains pus, cell debris, fatty substances and Dietrich's plugs (small foul-smelling aggregations of bacteria and fatty acids). In children the cough is the more characteristic feature, expectoration being absent unless postural drainage is carried out, but vomiting of pus is by no means rare.

Hæmoptysis is relatively common and may be the only symptom in the so-called "dry" bronchiectasis. It arises from granulations, but may be excessive or fatal from ulceration into larger vessels.

Attacks of fever with rigor are often evidence of extension of the infective process into the surrounding lung parenchyma. Repeated attacks may result in excessive fibrosis in the involved area. The same symptoms sometimes arise from blocking of bronchi with granulations and infective cedema of the bronchial walls, leading to retention of infective products in the affected portion of the lung.

Pain is generally the result of attacks of pleurisy over the affected lobe. Indigestion and diarrhoea are usually signs of toxic absorption, but may be the first evidence of amyloid disease.

*Diagnosis.*—The diagnosis entails not only the fact that bronchiectasis is present but its exact distribution, the presence or absence of atelectasis and fibrosis, the effect on the mediastinum, the inciting cause if possible, *e.g.*, foreign body or obstruction by growth, and the presence or absence of sepsis in the upper air passages.

The detailed medical history is of particular importance in this disease, not only in reference to the organs of respiration but in general assessment of the patient.

*Physical Examination.*—It is well to remember that no physical signs may be detected in a patient suffering from bronchiectasis. In other cases they may be present on one examination and absent at the next; when present they are generally more obvious at the pulmonary base, but necessarily depend upon the extent of the disease. They may consist in diminished mobility, fremitus and resonance, dullness on percussion, tubular or amphoric breath sounds and coarse râles. These râles may disappear after vigorous coughing, and occasionally vocal resonance may be increased.

Clubbing of fingers and toes is almost constant in cases of bronchiectasis, although varying greatly in degree. It has been suggested that it is due to diminished oxygen tension in the blood



owing to cutting off of the function of a portion of the lung by disease. This would result in diminished tissue respiration, which should be more obvious in the extremities such as the finger-tips and toes. This explanation is not, however, adequate, as, after removal of a lobe of the lung or even the whole of an infected lung, there is often considerable improvement or even actual disappearance of the clubbing.

*Sputum Examination.*—It is essential to eliminate all possibility of tuberculosis by repeated examinations of the sputum, as these conditions are not uncommonly associated.

Other organisms commonly found in bronchiectasis are various types of streptococci, hæmolytic, non-hæmolytic and viridans, staphylococci and micrococcus catarrhalis. Anaerobic organisms, fusiform bacilli, spirochætes and a specialised organism—*bacillus melanogenicus*,

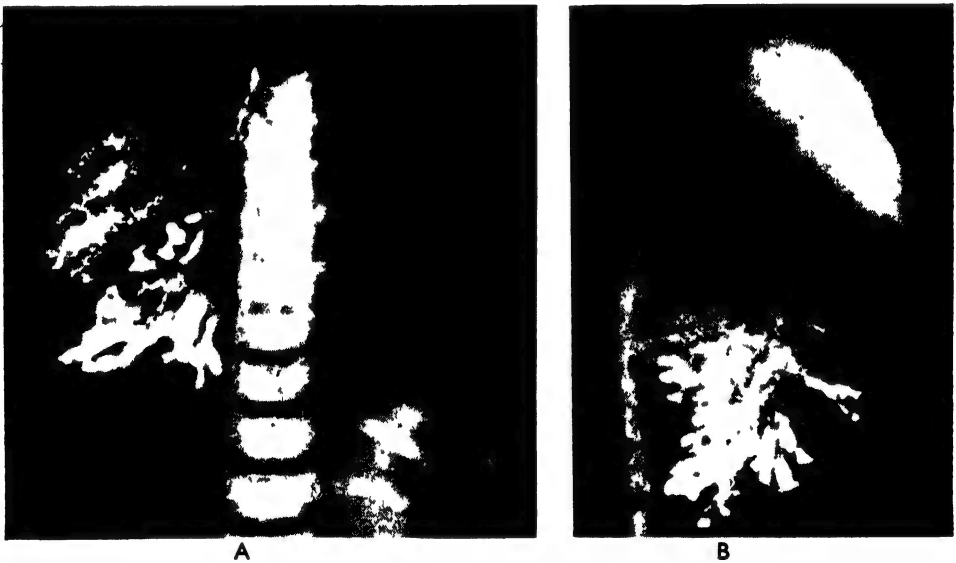


FIG. 225  
Bronchogram of moderate degree of bronchiectasis.  
A, anteroposterior, and B, lateral views.

which on culture gives off the typical odour of foetid bronchiectasis or abscess—have been regularly found.

*Radiological Examination.*—When atelectasis is present, a definite shadow corresponding to the airless lobe will be demonstrable in the film. In the absence of atelectasis some thickening of the structures from the hilum to the base may be seen.

*Bronchography.*—The introduction of opaque oil into the bronchi permits visualisation of the bronchial tree (Fig. 225). Before its introduction an attempt should be made by postural drainage to evacuate the purulent contents of the bronchi.

It is essential to fill all the lobes of each lung individually, and this will necessitate the filling of each on different occasions. As elimination occurs much more rapidly on the less affected side, this should be filled first and anteroposterior and lateral exposures



taken. About a week or ten days later the affected lung should be similarly filled. An excessive quantity of opaque oil tends to flood into the alveoli and 9 to 10 c.c. are quite sufficient for each lung. One of two preparations may be used for bronchography, neohydriol or lipiodol, both of which contain 40 per cent. of iodine in a basis of vegetable oil. By bronchography the site and extent of the lesion can be accurately determined and the question of treatment decided.

*Bronchoscopy.*—This method of investigation is of considerable value as it permits a direct view of the lesion, *i.e.*, the bronchi discharging pus, and eliminates the presence of a foreign body, new growths or strictures. It is essential in adults where atelectasis is present.

*Complications of Bronchiectasis.*—**Empyema.**—This is probably the most common complication, and the pus in the pleura is generally foetid. The pleural lesion is often subacute, and in a proportion of cases after drainage of an empyema the pulmonary symptoms disappear, often for long periods.

**Cerebral Complications.**—Cerebral abscess or meningitis may arise at any time in a bronchiectatic subject. Two views are held for the predilection of the brain as a site for abscess in pulmonary suppuration, (1) the embolic theory, in which it is suggested that the infected emboli pass into the left heart and thence are shot up to the brain via the carotids, and (2) the specially favourable soil provided by brain tissue for development of the specialised organisms—*spirochaetes* and *fusiform bacilli*—found in pulmonary suppuration. These organisms have been demonstrated repeatedly in the walls of secondary cerebral abscess, although not in the pus.

*Treatment.*—This may be divided into general and local treatment.

General treatment is directed to the improvement of the general health of the patient, and may be grouped under the headings of rest, nutrition, change of climate and treatment of sepsis elsewhere. Rest in bed is almost invariably followed by improvement in bronchiectatic subjects, but the benefit is only temporary. Nutrition can be improved by nourishing diets, especially in the poorer classes of the community, but no specific effect has been obtained by the use of vitamins.

Change of climate will generally benefit bronchiectatic subjects. The seasonal remissions found in damp climates during the winter encourage removal of the patient when possible to warm, dry climates. Heliotherapy has no specific effect. Elimination of sepsis elsewhere in the body is obviously of advantage to these patients. This applies especially to infections in the upper air passages, mouth, throat, nose and its sinuses. Many attempts have been made to prove the origin of bronchiectasis from nasal sinusitis but without definite results. On the other hand the removal of oral and especially nasal sepsis is frequently followed by improvement in the bronchiectatic symptoms.

Local treatment may be (1) non-operative and (2) operative, but it must be realised that all non-operative and certain forms of operative treatment are palliative, and the only cure is the entire removal of the diseased tissue.

**Non-operative Treatment.**—*Postural Drainage* is the most valuable non-operative measure at our disposal, depending upon the effects of gravity for the evacuation of the contents of the bronchial tubes. This treatment is best carried out on a specially constructed double-inclined plane or postural bed (Nelson), either of which allows complete inversion of the upper half of the body along one plane and the thighs and legs along the other. Treatment morning and evening for half an hour will often permit considerable freedom from cough during the rest of the day and night and, by increasing drainage, will often rid patients of the associated fœtor of breath.

*Treatment by Drugs* has been carried out for many years. Of these, creosote has been employed most frequently; iodides have also been used in order to produce a mild bronchitis, and thereby help to wash out the purulent contents by increasing the secretions of the mucosa.

In recent years, as a result of the finding of spirochætes in the lesions, arsenic by the intravenous route has been given an extensive trial. The results are sometimes striking, especially as regards the fœtor of the expectoration, but it has no effect upon the bronchial secretions.

Vaccine therapy has no curative value but may act in a prophylactic manner and prevent the repeated reinfections which, as a result of acute coryzal infections, spread down from the upper air passages.

**Operative Treatment.**—*Bronchoscopic Aspiration* permits almost complete evacuation of the bronchial contents and will rarely fail to afford relief, especially when combined with postural drainage. It may be carried out at weekly intervals at first, and these increased gradually to once a month.

*Artificial Pneumothorax* has been used for the relief of bronchiectasis but without success.

*Thoracoplasty* indicates the removal of extensive portions of ribs from the 1st to the 11th; relaxation of the rigid chest wall is produced and compression of the affected lobe or lung obtained.

This procedure, which is carried out in several stages, has in many ways the same disadvantages as pneumothorax, in that the brunt of the compression falls on the relatively soft normal lung. However, the particularly older patients with extensive unilateral disease experience considerable relief, and occasionally disappearance of symptoms may be obtained.

*Phrenicectomy* has been advocated and much practised. The results are uncertain and rarely justify this procedure. The danger of diaphragmatic paralysis in patients with large quantities of sputum is evident.

**Radical Extirpation.**—After many trials over many years associated with such a high mortality as to make it prohibitive, it may be said that lobectomy or removal of the affected lobe for bronchiectasis, is now put on a satisfactory basis, the mortality varying from 3 to 14 per cent. in different groups of statistics.

It has further been found possible to remove the right middle lobe when it is involved, together with the lower one, and to remove the corresponding portion of the left upper lobe, the lingula, with the lower under similar circumstances.

During the last three or four years a number of bilateral lobectomies have been carried out successfully, and likewise, in complete unilateral

disease, total pneumonectomies have been recorded in a number of patients.

For the exact technique of the operation the various monographs on the subject should be consulted, but there are certain essentials which may be mentioned here. Firstly, it is necessary that satisfactory bronchograms filling all lobes of both lungs should be obtained to determine the exact distribution of the disease. Secondly, at least one month should be allowed to elapse between the time of the bronchographic examination and the operation, during which time regular postural drainage should be carried out. The presence of iodine oil in the remaining lobe appears to delay its post-operative expansion. Thirdly, an exact assessment of the patient is necessary as regards the cardiovascular and renal systems, and should include blood examination and grouping for possible transfusion. Neurological examination may determine the presence of a cerebral abscess and should not be neglected. Fourthly, the induction or attempted induction of artificial pneumothorax on the affected side about a fortnight before operation, will sometimes temporarily diminish the sputum, and by gradually allowing the other lung to take up the full respiratory function permits more easy adjustment and diminishes the effect of freely opening the pleura at operation.

Again discussing general principles, two types of lobectomy may be carried out—the one-stage and the two-stage operations. In the former the operation is completed at one stage regardless of the presence or absence of general adhesions in the pleura. In this type the operative closure of the lobar bronchi is expected to remain sufficiently long for the expansion and adhesion to the chest-wall of the remaining lobe (usually the upper). Should an empyema, without or with a bronchial fistula, form it will then be localised to a small pocket at the base.

In the two-stage procedure, which is used only when adhesions are absent over the normal lobe, the first stage is used for separation of any adhesions present over the diseased lobe, the division of the ligamentum latum pulmonis and the roughening of the parietal pleura over the upper lobe with gauze. The chest is then closed and re-opened about four to six weeks later. By this measure adhesions are produced and post-operative total empyema is prevented.

*Poudrage.*—Adhesions over the portion of the lung to be left behind may be produced without open operation. After induction of artificial pneumothorax a powder consisting of sterilised talc with  $\frac{1}{4}$  per cent. iodine is insufflated through a cannula over those parts of the lung which it is desired to make adherent. All air is then withdrawn from the chest. Adhesive formation takes three weeks to become firm.

The one-stage method is being more generally adopted not only in this country but on the Continent and in the United States.

*Complications.*—*Reactionary hæmorrhage* due to incomplete hæmostasis at the hilar stump.

*Secondary hæmorrhage* may occur from sepsis in a residual pocket near the hilum, and may prove fatal.

*Pericarditis.*—Suppuration in the pericardium occasionally occurs, probably due to lymphatic spread from a residual empyema.

*Suppurative Pneumonitis* may arise in the residual lobe or in the opposite lung. It is probably a primary atelectasis due to filling of the lobar bronchi with pus from the affected lobe during operation. Almost invariably the lobe sooner or later breaks down into multiple abscesses. The only hope of recovery if the condition does not show evidence of clearing up after a week to ten days is the removal of the remaining lobe.

## TUMOURS OF THE LUNG

**Benign Tumours in the Bronchi** are more often encountered than those in the parenchyma of the lung, the adenoma being the most common (Fig. 226, A). Until 1930 only twenty-six cases of benign growth had been recorded, but since that date a considerable number have been reported, the increase being due to more frequent use of the bronchoscope. Fibroma, papilloma and localised enchondroma have all been described.

*Symptoms.*—The only symptom, before the tumour is large enough to obstruct the bronchus in which it originates, is recurrent hæmoptysia. Later evidence of bronchial obstruction appears with symptoms of

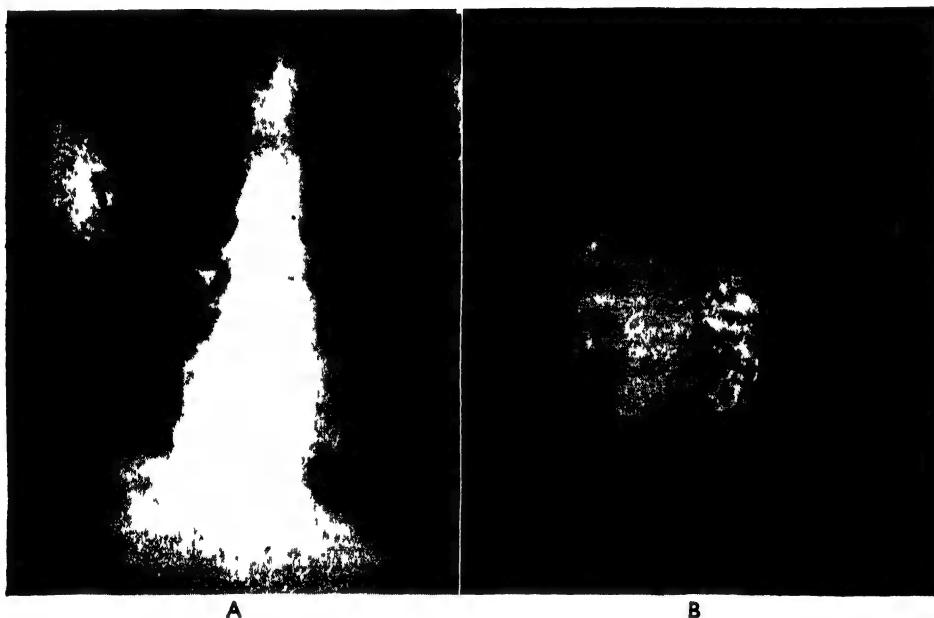


FIG. 226

A, bronchogram of adenoma of right lower bronchus (note the lumen is unaltered above the obstruction). B, the appearances of a carcinoma of the bronchus showing the "fat-tailed" appearance.

pneumonia or pleurisy. Clinical signs are absent except for a whistling sound over the affected bronchus. When obstruction is present, signs of lobar or pulmonary collapse will be found.

*Bronchography.*—Appearances will differ if the obstruction is complete or partial. In the former the site of the tumour is shown by a line, often convex upwards, and an absence of narrowing above the obstruction. When it affects only a part of the bronchial wall a "filling defect" may mark the position of the tumour. The diagnosis is confirmed by a bronchoscopic biopsy.

*Treatment.*—Piecemeal removal through a bronchoscope, followed by the introduction of radium to the site, is probably the most effective treatment. When the lung is disorganised by pent-up infective products the removal either of the lobe or of the lung is the only method likely to offer hope of permanent relief.

**Benign Tumours in the Lung.**—Various tumours have been reported, but they are all pathological curiosities—lipoma, angioma, neurofibroma and chondroma, the last being the most frequent. They give rise to neither symptoms nor signs until extension to the pleural surface causes pleuritic pain.

**Malignant Disease of the Lung.**—The incidence of malignant disease of the lung diagnosed during life has increased considerably in the past twenty years, and statistics suggest that lung carcinoma comprises 10 per cent. of all cancer cases. The disease is more common in males, the proportion being three men to one woman; it occurs chiefly between the ages of 40 and 60 years.

*Inciting Causes.*—Tuberculosis is not rarely associated with carcinoma in the lung, but there is no evidence of any specific connection between the two diseases.

Chronic pulmonary irritation has been put forward as an inciting factor. Evidence of proliferative and metaplastic changes has been produced after influenzal infections, and after the injection of irritant substances into the bronchi. Although smoking, inhalations of dusts and exhaust gases from motor cars have all been held responsible for carcinoma of the lung, no incontestable proof exists of their effects in the production of malignant disease of the lung.

The most striking evidence of the effect of a certain type of dust in the incidence of pulmonary cancer is produced by Rostocki. This particular dust, found in Schneeberg mines, contains minute particles of arsenic and is slightly radio-active. In the course of time it produces pneumoconiosis, and a very high proportion of these miners die from carcinoma of the lung. In many other types of pneumoconiosis due to inhalation of silica, coal dust, etc., there does not appear to be any increased incidence of cancer.

It is probable that many chronic irritants may act as the inciting factor where the predisposition exists.

*Site.*—The great majority of pulmonary cancers arise in the larger bronchi, the most common site being the division of the main lobar bronchi into their secondary branches. The right lung appears to be involved more often than the left, but the upper and lower lobes are almost equal in frequency.

*Pathology.*—The early descriptions of carcinoma divided them into two chief groups, the bronchial and the alveolar, according as to whether they arise in the larger bronchi or towards the periphery of the lung.

It is now considered that all primary pulmonary carcinomas are bronchogenic, and that they take their origin from the basement cells of the bronchial epithelium. This theory accounts for the gross differentiation of many of the carcinomas and their polymorphic appearance. Thus it is not uncommon to find bronchogenic carcinoma showing squamous cells in one portion and oat cells or an adenocarcinoma in another portion. It is almost characteristic of the "oat cell" type to produce submucous infiltration in the bronchus, causing narrowing of the lumen and relatively late ulceration; hence mediastinal glandular involvement is usually present when the

diagnosis is made. The other types ulcerate earlier and symptoms, such as hæmoptysis, may be encountered before metastasis has occurred. The production of bronchial obstruction will lead to atelectasis of that portion of the lung aerated by the involved bronchus, and a relatively large shadow may be seen in a radiograph, but it is well to remember that the primary growths may be quite small in such a case.

*Symptoms and Signs.*—The chief symptoms are cough, either dry or productive, hæmoptysis, pain and dyspnoea. In the later stages general symptoms such as loss of weight, weakness and loss of appetite. The former symptoms, especially if persistent, in individuals after forty should give rise to suspicion of malignant disease of the lung.

Physical signs are often absent in the early stages, but later when bronchial obstruction is complete, the physical signs of lobar collapse, *i.e.*, of consolidation, with displacement of the heart to the affected side, of pleural effusion or of mediastinal obstruction may be elicited.

Pyrexia, either due to toxæmia from the growth or from the absorption of toxins from retained secretion, is common. Enlargement of the supraclavicular glands and of the axillary glands is sometimes present, but is evidence of late disease. Congestion and œdema of head, neck and upper extremities indicate obstruction of the superior vena cava. Paralysis of the left recurrent laryngeal nerve leading to hoarseness, of the phrenic nerve leading to diaphragmatic paralysis, and Horner's syndrome (ptosis, myosis, anidrosis and enophthalmos), due to sympathetic paralysis, are all evidence of mediastinal involvement.

Clubbing of fingers and toes is not rare.

*Sputum.*—The sputum is occasionally blood-stained, often purulent and, on rare occasions, portions of growth may be discovered. Examination of the sputum by the wet-film method of Dudgeon gives a high proportion of positive results in cases of carcinoma of the bronchus, and is a very important method of investigation.

*Radiological Examination.*—This method is not only valuable but essential in all suspected cases of carcinoma. The shadow may be either that of the tumour itself or the result of obstruction, effusion, or of a secondary mass in the mediastinum (Fig. 227, A).

In the peripheral carcinomata, the shadow is generally somewhat rounded, homogenous and clearly demarcated from the surrounding lung. Occasionally the central portion necroses and may give the appearance of a pulmonary abscess with a thick wall. In the more frequent large bronchial carcinoma, which causes bronchial obstruction, the shadow will correspond to the collapsed lobe. The occasional so-called "hilus" carcinoma gives a shadow in the hilum with radiating shadows into the lung tissue.

The picture of miliary carcinoma is somewhat similar to that of miliary tuberculosis, *i.e.*, shows diffuse mottling of one or both lungs.

*Bronchography.*—The introduction of neohydriol into the bronchial tree will demonstrate bronchial obstruction. In carcinoma the shadow above the obstruction is somewhat tapering—"rat-tail" (Fig. 226, B).

In the peripheral neoplasms bronchial obstruction generally shows up better in the lateral view.

*Bronchoscopy.*—The diagnosis of carcinoma involving the main lobar and adjacent portions of the secondary bronchi can be made with precision by this method of examination in association with biopsy. The appearances vary somewhat from a definite proliferating, easily bleeding mass to a submucous infiltration narrowing or obstructing the lumen. A portion should be removed for histological examination.

*Examination of Effusion.*—When an effusion is present it may be due to metastases in the mediastinal glands, causing lymphatic



FIG. 227

Appearance of carcinoma of the lung. A, before, B, after dissection pneumonectomy.

obstruction, or to metastasis in the pleura itself. It should be aspirated and centrifuged, the supernatant fluid poured away and the sediment hardened in formalin and later treated for histological section like a solid tumour, when groups of malignant cells may be found in the sections (Mandlebaum).

*Treatment.*—According to the general condition of the patient, the extent and site of the neoplasm and the presence or absence of metastases, treatment may be radical or palliative.

Radical treatment is possible in an increasing proportion of cases owing to earlier diagnosis and consists in total pneumonectomy. It is essential to carry out this operation by ligature of the pulmonary artery and two pulmonary veins of the affected side and to divide the main bronchus near its origin in order to remove the hilar glands and those beneath the bifurcation of the trachea (Fig. 227, B).

Apart from general conditions which may contraindicate any major operative procedure, extension of disease either direct or by metastasis



should be eliminated. Radiological examination may show a mediastinal mass or a pleural effusion, radioscopy a paralysed diaphragm. Changes in voice may be evidence of recurrent laryngeal paralysis and clinical examination may disclose enlarged glands in the supraclavicular or axillary regions or enlargement of the liver. A careful neurological examination should be carried out.

Obviously, the discovery of any secondary deposit contraindicates radical operation.

The results of radical operation are increasingly hopeful, and patients are still living and well after operations of over seven years ago.

Palliative treatment consists in the use of radiotherapeutic methods—radium or X-ray therapy.

In bronchial carcinoma involving the larger bronchi radon seeds in special containers (Tudor Edwards) may be inserted through the bronchoscope, left *in situ* for five days and then removed, or individual seeds may be inserted directly into the carcinoma via the bronchoscope. In the more peripheral types, thoracotomy is carried out and the radon seeds implanted directly into the tumour. Both these methods have led to occasional cures, and in others the bronchus has been recanalised and the effects of retention of secretions overcome with great improvement in the general condition of the patients.

The value of X-ray therapy depends upon the experience of the radiotherapist. Cure is rare but life may be prolonged by its use.

Other palliative treatment consists in aspiration of large effusions, the drainage of associated abscess or empyema and the relief of pain, when it is a marked feature.

*Metastases* are stated to occur earlier in peripheral than in more central types, possibly owing to the greater quantity of cartilage shutting off the latter and the thin-walled vessels surrounding the former.

The most common site for metastases is the mediastinal glands, but later liver, pancreas, suprarenals and kidneys become involved.

Brain metastases result in 10 per cent. of cases, and occasionally the first evidence of any abnormality in the patient may be the symptoms and signs of a cerebral tumour.

*Prognosis* is invariably grave, but occasional and more frequent recoveries are resulting from operative treatment.

**Endothelioma of the Lung.**—A few cases of this condition have been described.

The diagnosis is generally made after the removal of the lobe and microscopic section of the tumour.

They generally give rise to hæmoptysis as the chief symptom, and by X-ray can be seen as well-defined homogenous shadows in the lung tissue without any apparent lung reaction around.

**Sarcoma of the Lung.**—This malignant neoplasm is now considered to be rare as a primary tumour in the lung. At one time the "oat-celled" carcinoma was described as sarcoma, and this accounts for the considerable number of cases described in the old literature. Bronchial stenosis is rare.



### MEDIASTINAL TUMOURS

**Tumours of the Thyroid.**—Such goitres may be partly intrathoracic, partly cervical or wholly thoracic. In the vast majority there is a definite connection with the thyroid gland in the neck, although this is sometimes much attenuated. The goitre is generally nodular, although occasionally a large single adenoma or cyst-adenoma may comprise the intrathoracic portion, and there is a great tendency to calcification.

In many cases the mass rises in the superior mediastinum when the patient swallows, and this may be seen on radiological screen examination, but the absence of this sign does not exclude the diagnosis.

Intrathoracic thyroid tumours usually give rise to signs of pressure on the trachea, and are occasionally diagnosed as asthma. In some cases thyrotoxic signs are present.

These tumours should be removed surgically through the usual collar incision, and it is possible in the majority to deliver the sub-sternal mass without median sternotomy.

**Teratoid Tumours.**—These tumours are congenital and vary in type from the dermoid cyst to the solid teratoma, which may contain portions of almost any structure. Likewise, they vary considerably in size and in many cases do not cause symptoms until middle age. Their site of origin is always the mediastinum, usually the upper portion, and often in close relationship with the innominate veins to which they may be firmly attached. As they enlarge they spread outwards covered by mediastinal pleura in front of the pulmonary hilum or between the pulmonary lobes to which they often become firmly attached.

The pressure on the bronchus may result in erosion of its wall and one or more cavities of the more solid teratomas may discharge sebaceous material and hair into the bronchial lumen and be coughed up. Secondary infection may follow and the condition be confused with chronic pulmonary abscess. In other cases rupture of the cyst may flood the pleura with sebaceous material. Malignant changes may take place.

*Symptoms and Signs* may be absent and diagnosis may follow routine radiological examination (mass radiography). In other cases persistent non-productive cough, with or without dyspnoea on exertion, may predominate. When rupture into a bronchus has occurred purulent expectoration is associated and occasionally foetor of breath and sputum is present.

The physical signs are only obvious in the larger tumours or when secondary complications occur. In the uncomplicated tumours dulness on percussion in the anterior parasternal region is associated with absence of breath and voice sounds.

*Radiological Examination* will demonstrate a rounded shadow in the superior mediastinum, extending outwards and downwards into the lung fields. Bone or teeth may be visible in the tumour and in the dermoid cysts calcification of the wall is occasionally seen.

*Treatment.*—Removal of the tumour is indicated on diagnosis as the smaller the tumour the less the operative risk, and the possibility of complications including malignant changes must be borne in mind.

**Tumours of Thymus.**—SIMPLE ENLARGEMENT or hypertrophy of the thymus is generally found in infants, and occasionally gives rise to urgent dyspnoea. There are records of thymectomy in such cases, but it would appear to be unjustifiable as X-ray therapy results in rapid diminution in size.

The diagnosis is confirmed by radiological examination, showing a dense shadow in the region of the thymus.

THYMOMA or true thymic tumour is almost invariably malignant, although varying in its rapidity of growth. In some cases they are soft and vascular, in others there is considerable fibrosis with lobulation. The former rapidly infiltrate all structures while the latter often show capsule formation. Invasion and compression of large vessels and air passages occur fairly early, and death takes place from asphyxia. The symptoms are those of mediastinal obstruction to air passages and blood vessels, viz., increasing dyspnoea, venous engorgement and oedema of the upper limbs, head and neck. The prognosis is bad.

**Tumours of Nervous Tissue.**—These comprise the ganglion neuroma and the neurofibroma. GANGLION NEUROMATA are relatively rare and arise in the region of the costovertebral groove, probably originating from the primary ganglionic crest, and show typical ganglioma cells. Horner's syndrome is often present and localised pain a symptom. They occasionally attain a large size, and should be removed by thoracotomy.

NEUROFIBROMATA are often found in the same situation as the ganglion-neuromata, and arise from the intercostal or sympathetic nerves. They may be rounded or oval in outline, and either wholly within the thorax, or occasionally partly within the vertebral canal and partly in the thorax—"dumb-bell" or "hour-glass" tumours. The single type may be symptomless or give rise to pain, the dumb-bell type almost always causes pain and occasionally signs of pressure upon the spinal cord, and usually erodes the transverse process, vertebral body and often the rib on which they are situated. They have the same histological picture as such tumours elsewhere. Surgical removal is indicated.

**Connective Tissue Tumours.**—LIPOMATA arise in the fatty tissue of the mediastinum or extrapleural space. They may be partly extra and partly intrathoracic or wholly intrathoracic, and occasionally attain an enormous size (one weighing 17½ lbs. has been recorded). They are rare.

FIBROMATA, except in combination with carcinomatous changes, are rare.

CHONDROMATA arise in the cartilages of the ribs, the ribs themselves, the sternum and vertebral column. They have a tendency to undergo malignant change but otherwise enlarge slowly. In the majority of cases, except those arising from the vertebral column, the tumour involves the external and pleural surfaces of the chest-wall. In view of their tendency to malignancy, they should be widely resected.

**Tumours of the Œsophagus.**—Other than carcinoma, tumours of the Œsophagus are rare, but occasionally diverticula of some size are found in the lower Œsophagus and have been treated by operation.

**Tumours of the Lymphatic Glands.**—Enlargement of the lymphatic glands in the mediastinum may be due to causes such as acute inflammation and tuberculosis, which will not be considered here.

Progressive enlargement occurs in lymphadenoma, lymphosarcoma and in secondary carcinoma, all of which are by no means rare. Blood conditions such as leukæmia also produce mediastinal glandular enlargement.

In LYMPHADENOMA or Hodgkin's disease the primary glandular enlargement may be in the mediastinum. It is often accompanied by relapsing attacks of pyrexia, a secondary anæmia and later by increase in size of lymphatic glands in other parts and by changes which cause splenic enlargement.

LYMPHOSARCOMA grows much more rapidly and steadily encroaches on the surrounding structures, thereby giving rise to more urgent symptoms of mediastinal obstruction at an early stage. Both these tumours respond to X-ray treatment, in lymphadenoma more slowly, in lymphosarcoma often dramatically. In the former, however, life is usually much more prolonged, whereas in the latter recurrence takes place relatively soon and is then usually radioresistant.

SECONDARY GLANDULAR CARCINOMA occurs in the mediastinum from primary carcinomata of the bronchus, Œsophagus, breast and thyroid.

### PULMONARY TUBERCULOSIS

The increasing employment of surgical measures in the treatment of pulmonary tuberculosis necessitates a short description of the various types of procedure employed, the indications for their use and the rationale upon which they are based.

It is not intended to discuss the etiology, pathology, diagnosis or treatment of pulmonary tuberculosis in general, for which purpose textbooks on medicine should be consulted.

In pulmonary tuberculosis the aim of the surgeon is largely to overcome certain mechanical disabilities consequent on the relatively rigid thoracic cage, which encloses the lungs, and to increase the possibilities of maintaining the lung at rest, reducing pulmonary movement to a minimum and permitting relaxation. The value of rest in tuberculosis is well illustrated by the use of external splinting in joint disease and, in selected cases, of internal splinting by bone grafts in vertebral tuberculosis. In the lung, rest is not so easily brought about as in the more simple joint lesions, and, hence, specialised methods are necessary in certain cases.

*Pathology.*—This is again a wide subject which will only be touched upon in so far as it is essential to the understanding of the reasons for the application of surgical measures.

The type of lesion produced in the lungs from invasion by tubercle bacilli will vary according to the resistance of the host and the virulence of the organisms. When the response is poor, a general

miliary or acute pneumonic tuberculosis will result, while with a slightly higher resistance an infiltrative process is produced, portions of which may caseate and discharge their contents into the bronchi, cavity formation being a feature of the condition. This type may later, as a result of treatment on general lines, show evidence of fibrosis around the cavity or cavities, fibrocavernous disease; the most resistant patients may have an excessive formation of fibrous tissue, when the condition is termed "fibroid phthisis."

The aim of treatment in the more acute type is to build up individual resistance to such a degree as to arrest the disease, or at any rate convert it into the more chronic type.

*Treatment.*—It must be sufficiently obvious that the attainment of complete rest even to one lung is impossible, but the aim and basis of all sanatorium treatment depends upon complete bed rest in the early stages of the disease, so that a minimal pulmonary movement is attained. By this means the lung is rested, the lymphatic and vascular currents in the lung are slowed and in many instances the process ceases to spread. In such cases no accessory means of treatment are required, but in others there may be slow and steady deterioration, and it is in these patients that the employment of some form of relaxation or collapse therapy has its indications.

Even in the fibroid group the contraction of the fibrous tissue may result in certain mechanical disabilities which will necessitate operative procedures. Thus, the pull of the contracting fibrous tissue may cause narrowing of the intercostal spaces, raising of the diaphragm and, most important of all, displacement of the mediastinum with the heart and main vessels, to the affected side. The dyspnoea and tachycardia resulting from this displacement may be sufficiently crippling to prevent the patient undertaking even minimal exercise, although the disease itself has been arrested.

In other cases the contraction of fibrous tissue proceeds further and gives rise to dilatation of the bronchial tubes, producing a secondary bronchiectasis with its associated expectoration.

In the intermediate group is a large number of patients in whom the persistence of a cavity or cavities exposes them to the risk of a bronchogenic spread of the disease to other parts of the lung, and in whom hæmoptysis may occur and either prove fatal or be followed by an acute spread of disease to other areas in the same or opposite lung. Apart from the risk to themselves, such patients persistently expectorate a sputum containing tubercle bacilli, cause spread of the disease to others and, unless the cavities are closed, provide a serious economic problem.

Closure of these cavities is often prevented by the adhesion of the lung to the relatively rigid chest-wall, and if this is permitted to collapse, the cavity walls are brought into apposition and healing may be attained.

This closure of pulmonary cavities will show itself by the disappearance of the expectoration: cough and fever will cease, dyspnoea improve and the risk of hæmorrhage from cavities be eliminated. It can be confirmed by radiological examination.

It is an elementary principle of treatment that the most simple measures should be employed first, and only when these fail should more radical procedures be undertaken.

**Artificial Pneumothorax.**—The introduction of air, under sterile precautions, into the pleural cavity will result in collapse of the lung in the absence of adhesions between the pleural layers. In the treatment of tuberculosis the air is gradually introduced by repeated refills over a period of ten to fourteen days, by which time complete collapse should be attained. It is carefully controlled by regular manometric readings of the pleural pressures and by X-ray screen examinations. Subsequent refills will be required at intervals suitable for the individual patient in order to keep the lung collapsed.

Treatment by artificial pneumothorax is continued for periods of two to four years, depending upon the state of the lung preliminary to the treatment and upon the views of the physician concerned.

Complications of treatment are pleural shock, hæmorrhage from the chest-wall due to the needle penetrating vessels, mediastinal hernia, *i.e.*, bulging of the mediastinal pleura towards the opposite side and, lastly, pleural effusions, clear or purulent, which may result in gradual obliteration of the pneumothorax in spite of repeated refills.

It is found in a proportion of patients that, although artificial pneumothorax can be induced, collapse of the lung is only partial and that portions of the lung are held out to the chest-wall by adhesions between the pleural layers. As one would expect, the adhesions are most commonly encountered over the more grossly diseased areas of the lung, especially cavities, and the object of the artificial pneumothorax—the closure of the cavities—cannot be successful without other measures. When the adhesions are fibrous in type, and of varying shape, bands, straps or films, it is possible to divide them and permit the lung to collapse completely.

**Cauterisation of Adhesions.**—The operation is carried out by the passage of a thoracoscope through a cannula introduced into an intercostal space under local anæsthesia. The adhesions which have been localised by previous radiological examination are thus viewed and their suitability for division decided. Extensive surface adhesions or those containing lung tissue are unsuitable for division. If it is decided to divide them, another cannula is introduced at a suitable intercostal space and through this a cautery is passed. It is advisable to use a cautery at a dull red heat, and to have available a diathermic current to coagulate any bleeding vessels, as many adhesions contain quite large adventitious blood vessels. The adhesions are thus divided under direct visual control.

If suitable cases are chosen and the operation is done with due care, complications are rare. If pulmonary tissue is damaged, the operation may be followed by tuberculous effusions, occasionally associated with secondary infection. Hæmorrhage rarely occurs if adequate precautions are taken and the cautery used at a relatively dull red heat.

Both the previous methods are intrapleural operations and depend upon the absence of extensive adhesions between the pleural layers,

but in a proportion of cases adhesions are generalised and other methods which are extrapleural must be adopted in order to bring about pulmonary collapse.

**Extrapleural Operations.**—In the acute, subacute or early chronic cases of pulmonary disease, two relatively minor methods are available.

1. **DIAPHRAGMATIC PARALYSIS.**—This will produce collapse of the lung to a definite extent, although naturally if the pleura is adherent the effect will largely be upon the base and lower mid-zone of the lung. The operation is carried out, under local anæsthesia, by exposing the phrenic nerve in the neck as it crosses the scalenus anticus beneath the sternomastoid muscle. It can be crushed by artery forceps and any accessory fibres divided, an operation which will produce a temporary paralysis lasting from four to nine months ; or the paralysis can be made permanent by division of the nerve and slow extraction of its distal end from the mediastinum, by which means all accessory branches are ruptured.

The operation has been employed for cavitation at the base and in the mid-zone of the lung and as a temporary procedure for soft-walled apical cavities ; when these close the operation is made permanent before diaphragmatic movements return. It can be used for pleural effusions which return rapidly after aspiration, and for diaphragmatic pain in association with artificial pneumothorax, both of which are to be regarded as temporary operations.

At the termination of artificial pneumothorax treatment in patients in whom there has been extensive destruction of the pulmonary tissue, it is unreasonable to expect a contracted lung to fill the same space, on re-expansion, as a normal lung. Therefore, as expansion occurs the intercostal spaces become narrower, the mediastinum is drawn over to the affected side and the diaphragm rises slightly. If this is insufficient an increasing strain comes upon the healed lung and there is a tendency for cavities to reopen and the disease to become reactivated.

If phrenic evulsion is carried out at the beginning of pulmonary re-expansion, the high rise of the diaphragm may be sufficient to compensate for the decreased size of the affected lung and healing will be maintained.

2. **EXTRAPLEURAL PNEUMOTHORAX.**—This operation depends upon the presence of a thin cellular layer which lies between the deep surfaces of the periosteum of the ribs and intercostal muscles on one side and the parietal pleura on the other.

Thus, when the visceral and parietal pleural layers are adherent, the lung and its pleural layers can be separated from the chest-wall through this layer—the endothoracic fascia. The operation is generally performed at the apex of the lung by resection of a portion of the 3rd or 4th rib posteriorly. The correct layer is determined and the apex of the lung separated from the chest-wall down to the level of the posterior ends of the 5th, 6th or 7th ribs according to indications. The superficial tissues are sutured and the extrapleural air-space maintained by repeated introduction of air.

Cavities can in this way be closed as by intrapleural pneumothorax, and although the operation has as yet only been regularly employed

for about three years, it has nevertheless a definite field of usefulness, especially in cases in which the disease is subacute and at a time when thoracoplastic procedures are generally contraindicated.

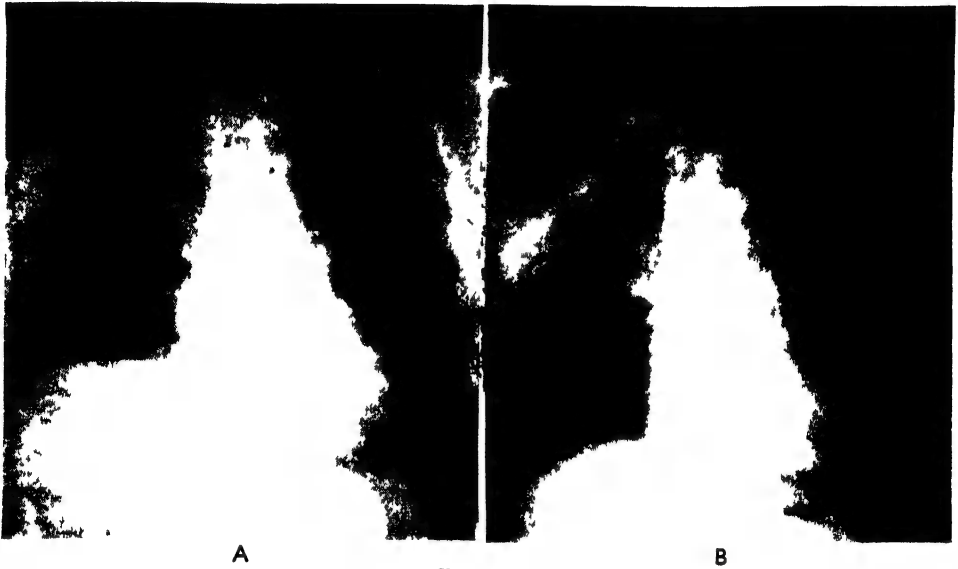


FIG. 228

Right-sided pulmonary tuberculosis of the upper lobe  
A, before, B, after partial thoracoplasty.

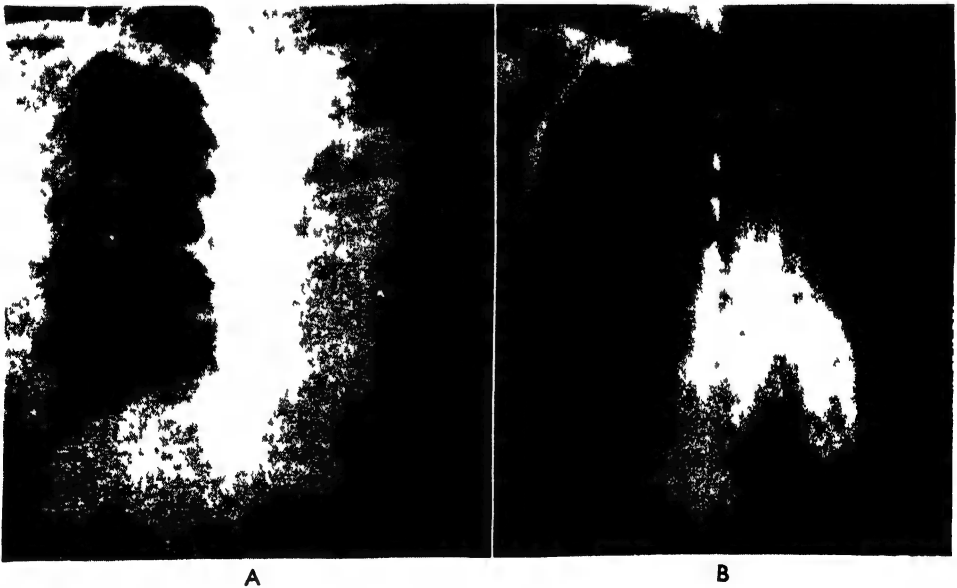


FIG. 229

Left-sided fibrocavernous phthisis A, before, B, after complete thoracoplasty.

**Thoracoplasty.**—Thoracoplastic operations consist in resection of extensive portions of ribs to enable the lung to collapse by mobilisation of the chest-wall. As the resections are subperiosteal, the ribs reform in the course of time in a new position, leaving a narrowed hemithorax.



The operation may be partial, *i.e.*, confined to the upper five or seven ribs, or may be complete from the 1st to the 10th ribs, according to the extent of the disease (Figs. 228 and 229).

Partial thoracoplasty entails very extensive removal of the upper ribs, the whole of the first up to its costal cartilage, the 2nd rib just short of its cartilage, and the 3rd and 4th about  $1\frac{1}{2}$  in. short thereof. The ribs are divided posteriorly at the costotransverse joints. If the extent of the disease necessitates, as it generally does, resection of five ribs or more, the operation should be divided into two stages with an interval of fourteen days between them, the upper three or four at the first and the 5th, 6th and 7th, if required, at the second stage.

Apicolysis or separation of the apex of the lung from the parietes is now often combined with the upper stage of thoracoplasty when cavitation is a feature; this permits concentric closure of such cavity or cavities (Semb's operation).

Bilateral apical thoracoplasty is occasionally successful. When the complete operation is required, three stages are usually employed to permit gradual pulmonary collapse.

During the ensuing months the mobile chest-wall is gradually compressed by the use of bags of shot, of varying weights, graduated to the rigidity of the individual chest-wall, which are placed upon the chest for fixed periods daily. Those after complete thoracoplasty usually wear a form of firm jacket which holds in the operated side until rib regeneration is firm. Necessarily, thoracoplastic procedures, particularly the total, require a more rigid examination of the contralateral lung. It is rarely completely clear of old healed disease, but any except minimal active lesions at the apex should be regarded with suspicion; a review of previous radiograms is of considerable value in a doubtful decision.

Minimal tuberculous lesions in bone, kidney and larynx are not considered contraindications, but intestinal lesions are generally of more grave prognosis. Cardiac and renal disease, other than tuberculosis, are also contraindications to operation.

Thoracoplasty has been carried out in tuberculous patients with diabetes, provided this is well controlled.

The mortality of thoracoplasty will depend upon the interpretation of the absolute and relative indications and contraindications. If cases of advanced disease with active lesions on the other side are included, although the absolute number of patients restored to health may be greater, the operative and subsequent mortality rate will be higher. When operative indications are kept rigid the mortality should be 3 to 4 per cent., but on widening the indications may reach figures of 16 to 20 per cent.

### TUBERCULOUS PLEURAL EFFUSIONS

Tuberculous effusions in the pleura may be primary, or secondary to pulmonary disease. Three types are encountered, the yellowish clear effusion, the tuberculous purulent and the tuberculous purulent



with secondary pyogenic infection—pneumococcal, staphylococcal or streptococcal. The two former are often found to be sterile on culture, except upon special media, and no organisms may be seen in films.

Serous effusions after one or two incomplete aspirations often absorb and the pleural layers become adherent. In other cases gas replacement may be carried out, *i.e.*, the aspirated fluid is partially replaced by filtered air. These occasionally pass into the tuberculous purulent type. Any of these types may arise during the course of treatment by artificial pneumothorax.

The tuberculous purulent type is usually accompanied by the presence of definite tuberculous granulations on the pleura. Occasionally, perforation occurs directly through the chest-wall, usually anteriorly, but in other cases the communicating pleural opening is at a great distance from the presenting point, the abscess having tracked along a rib.

These effusions may be aspirated and the pleura irrigated with normal saline and later with flavine 1 : 1000 or Carrell Dakin's solution. They should never be drained. If no pulmonary disease is present, full expansion of the lung may be attained and the disease arrested. Where disease is present in the underlying lung the attainment of pulmonary expansion is likely to lead to reactivation of the disease there, and it is wiser to proceed to thoracoplasty if the condition of the contralateral lung permits, continuing the pleural lavage between the operative stages and after its completion until the pleural space is obliterated.

If a tuberculous purulent effusion is allowed to continue, sooner or later external sinuses or an internal bronchial fistula will appear, both conditions being more difficult to cure.

Secondary pyogenic infection is a very serious complication of tuberculous pleurisy and every possible means should be taken to avoid it by careful aseptic precautions during aspiration of the simple tuberculous types. In many cases it reaches the pleura either by a bronchial fistula or by spread from the lung without obvious communication.

In the less acute types, pleural lavage may be employed and, when hæmolytic streptococci are present, can be combined with oral or intramuscular administration of prontosil.

The majority of cases of secondary infection require external drainage, which should always be carried out by the insertion of an intercostal tube. As all these patients, if they recover sufficiently, will eventually require a complete and extensive thoracoplasty, it is advisable to insert the intercostal tube below the 10th rib, or in the anterior axillary line, to avoid the future operative field as far as possible.

The prognosis is serious.

A. TUDOR EDWARDS.

## CHAPTER XXV

### THE BREAST

**A***NATOMY*.—The breast lies in the subcutaneous tissues covering the chest-wall. In its fully-developed virgin state it is roughly hemispherical, and extends from the 2nd to the 6th rib and from the edge of the sternum to the mid-axillary line. It has a well-defined prolongation along the lower margin of the pectoralis major muscle, the axillary tail, which extends into the lower part of the axilla. It lies mainly on the pectoralis major muscle, but overlaps on to the serratus magnus and abdominal external oblique muscles. It is ensheathed by the superficial fascia which splits to enclose it, and from the laminae thus formed fibrous processes spread into the gland to support its lobes. From the anterior lamina, particularly in the upper half, fibrous strands extend to the skin and are known as the suspensory ligaments of Cooper. Just below and internal to the centre of the gland is the nipple, placed in the middle of a circular area of pigmented rugose skin—the areola, the surface of which is studded with numerous small projections due to specialised subaceous glands, the follicles of Montgomery. The summit of the nipple is perforated by the orifices of the lactiferous ducts. The breast is composed of from fifteen to twenty lobes each of which is pyramidal with its apex at the nipple. Each is complete in itself and consists of lobules of secreting tubules, and is drained by one main duct, viz., the lactiferous duct, which on approaching the surface dilates to form an ampulla, and then narrows again to reach the nipple. After lactation, the breast loses its firm compact structure and becomes more fleshy, more bulky and more pendulous. After the menopause its tubules atrophy and it becomes shrunken.

The blood supply is derived from the external mammary branch of the axillary, the internal mammary artery, and branches from the intercostal arteries of those spaces over which it lies. The venous return is to the axillary and internal mammary veins.

The lymphatic drainage may be divided into four groups :—

- (a) A central subareolar plexus, the efferents of which pass to the pectoral group of axillary glands.
- (b) The outer and lower quadrant drains into the central axillary glands, some channels passing direct, others going to the pectoral group first.
- (c) The upper and outer quadrant drains into the axillary glands via the pectoral group, while some lymphatic vessels pass direct through the costocoracoid membrane.
- (d) The inner quadrant efferents penetrate the intercostal spaces to enter glands along the internal mammary vessels.

The development of the breast is from a small circular thickening in the epidermis of the chest-wall during the second month of intra-uterine life. From the deep surface of this area solid columns of cells make their way into the subjacent tissue, and then produce lateral offshoots. The main

columns are the future lactiferous ducts, and from the lateral offshoots are formed the lobules and acini. The surface cells of the thickened area develop into the nipple and areola. The breast remains in this rudimentary state until puberty, when great activity occurs and the secreting tissue is fully developed.

#### ANOMALIES IN DEVELOPMENT

These may be classified as :

Anomalies in number—Deficiency, *i.e.*, amasia.  
Excess, *i.e.*, polymasia.

Anomalies in situation.

Anomalies in function.

Anomalies in size.

There may be complete absence of one or both breasts, known as *amasia*, or an absence as well of nipple and areola, a condition termed *athelia*. *Polymasia* means the presence of additional breasts on the anterior surface of the body along a line drawn between the midpoint of the clavicle and the pubic spine, a state of affairs normally seen in certain animals. *Polythelia* is a similar condition in which nipples without breasts are found. In addition, supernumerary breasts are recorded in many parts of the body, *e.g.*, the labium majus and the outer surface of the thigh. Anomalies of function include absence of secretion in women after delivery and that rare condition in which the male breast has secreted milk.

Anomalies in size are illustrated by *hypertrophy*, infrequently seen in young women. It is usually bilateral and the breasts grow to great size, causing embarrassment not only by their dimensions but by giving rise to pain, dyspnoea and palpitations. It may occur as an unrestrained overgrowth during pregnancy, but is most common in young non-pregnant women. The change is mainly a hyperplasia of the fibrous tissue and not of the glandular elements. Treatment is some form of plastic operation or complete amputation.

*Gynaecomastia* is the term applied to the development of the breast in the male, in whom at puberty an abortive attempt to form a breast normally takes place. This is represented by a slight enlargement beneath the areola and some throbbing and tenderness. This stage may give rise to parental anxiety, but the activity soon ceases and the swelling subsides. In rare cases, growth continues and a small virgin breast is formed on one or both sides. It may be thought difficult to justify an operation, but it is better to remove the breast than that the boy should be the subject of so definitely embarrassing an anomaly.

#### CLINICAL EXAMINATION OF THE BREAST

The breast is first examined by inspection, for which purpose the patient should be sitting up in bed or in a chair. Swelling, shrinkage or deformity of the breast is apparent, as also are abnormalities of the nipple or areola, and invasion and fixation of the skin. For palpation the patient is placed flat on the back in bed, and the observer sits beside her on the affected side. The physical characteristics of any

tumours are investigated by palpation with the flat of the hand, and between the fingers and thumb. Its mobility is determined with regard to the breast tissue, the skin and the underlying deep fascia. If the swelling is surrounded by the fingers and thumb of the left hand, and moved about by the right index finger, then, if it is attached to any part of the breast tissue, a pull will be communicated to each finger in turn as the tumour moves. If, however, it is free and unattached to the breast, it will move so freely as to communicate no pull on the surrounding fingers. Surface fixation may be demonstrated by attempting to lift the skin or to make it slide across the tumour. In testing for fixation to the deep fascia, the underlying muscle must first be put into action and the tumour tested for mobility along the long axis of its fibres. The pectoralis major is rendered taut by the patient placing the hand on the iliac crest and pressing strongly against it; the serratus magnus is put in action by instructing the patient to touch the back of the head, which movement is resisted by the observer. The axillary contents are then examined and the number, size, position, consistence and fixation of any enlarged glands noted. And finally, before any opinion can be given, the spine, supraclavicular triangles, opposite breast and axilla, abdomen and chest, must be examined for the presence of secondary growths.

## DISEASES OF THE NIPPLE AND AREOLA

### RETRACTION OF THE NIPPLE

This may be congenital in that the adult prominence is not developed, and the position of the nipple is marked by a pit in the centre of the areola (inversion of the nipple). This may lead to difficulties in suckling and predispose to infection. Acquired retraction is a classical sign of carcinoma of the breast, but can occur in any condition leading to scarring within the breast, such as a breast abscess.

### SIMPLE ECZEMA

This is typical of eczema as seen in other parts of the body, and is caused by the *Staphylococcus aureus*. It occurs at any age after puberty and is often associated with lactation. It yields readily to treatment and is of importance only if it complicates lactation, in which it may act as the starting point of an acute mastitis. Apart from this it makes suckling a matter of doubtful propriety as the milk is heavily infected, and any applications to the eczema will further contaminate the milk.

*Treatment* consists in bland ointments such as boracic, and exposure to the infra-red lamp. Before suckling, the area must be carefully cleansed and a nipple shield used. In severe lesions the child should be weaned. In those cases occurring apart from lactation mild mercurial ointments will readily effect a cure. Cracks and fissures follow injury during suckling, due to failure to harden the nipple

during the last month of pregnancy. If this is done and retraction corrected, the nipple should be able to withstand the pressure of the infant's alveolar margins. Proper hygiene both before and after delivery should ensure the complete absence of cracks and fissures, and so do away with the chief etiological factor of breast infections.

### PAGET'S DISEASE

This is a chronic persistent eczematous condition which does not yield to treatment and which is associated with a carcinoma of the breast.

It occurs in elderly women, rarely before 50 years, and is always unilateral. It has been described in men.

*Naked-eye Appearance.*—A small area on the nipple or areola is affected by a papular eruption, which soon breaks down and spreads over the whole areola and later to the surrounding skin. In time, the process being a slow one, the nipple and areola are destroyed and are replaced by a condition resembling simple eczema. The colour is a particularly vivid scarlet on a granular surface, with little points of pus oozing from it. The edge of the skin is thin, purple and well defined. Some cases have crusts over the whole or part of the area, and the typical appearance is seen only on removing these (Fig. 230).



FIG. 230

Paget's disease of the nipple.

*Microscopic Detail.*—There is a proliferation of the epithelium with desquamation of the surface layers, and a dense, round-celled infiltration of the corium with increased vascularity. In the surface epithelium certain cells undergo swelling and vacuolation, forming "Paget bodies" (Fig. 231). The ducts may be dilated and their lining membrane shows some proliferation (Kettle).

*The Nature of the Condition.*—In the original description it was accepted that carcinoma developed at some period as a result of the chronic inflammatory lesion in the skin. Sampson Handley, however, holds that the carcinoma exists previously in the breast and is the cause of the eczema. He has shown that a small atrophic scirrhus may cause sufficient lymphatic obstruction to bring about a water-logging of the skin, a shedding of the surface epithelium and the production of the typical appearance. It may be years before the growth is clinically recognisable, and it is not always in that part of the breast immediately subjacent to the areola. His theory is accepted to-day and treatment is based upon it.

*Treatment.*—In the face of an eczema of short duration and without a palpable tumour, doubt may exist as to the nature of the

condition. In such cases the usual treatment for simple eczema should be adopted, and if no improvement has occurred within one month the diagnosis of Paget's disease is established. If no tumour is palpable a resection of the breast disc with an elliptical area of skin is sufficient, but if the growth can be felt, the radical operation should be performed.

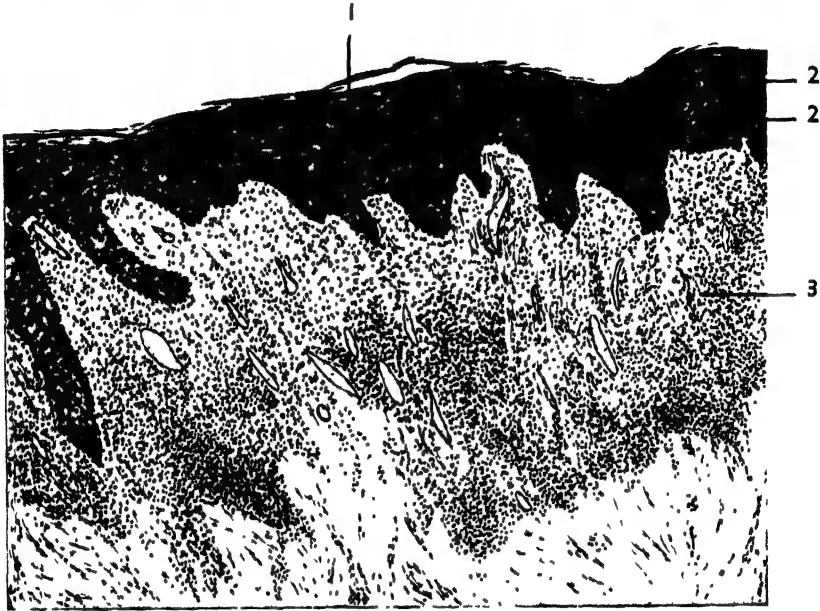


FIG. 231

Microscopic appearance of Paget's disease.

1 is the hypertrophied epithelium showing 2, Paget's bodies, and 3, the subepithelial zone of round-celled infiltration and congestion. (*Kettle.*)

### DISCHARGE FROM THE NIPPLE

A serous discharge from the ducts may be present in duct papilloma, duct carcinoma, and rarely in chronic mastitis. No abnormal condition may be palpable, though careful examination may prove that the discharge is produced by pressure over one isolated segment of the breast and always from the same orifice. A persistent discharge is always to be regarded as of great importance as suggesting a probable duct papilloma, and treated accordingly.

A blood-stained discharge is not necessarily a sign of a malignant process, but the same importance will attach to it as to a simple serous discharge.

## DISEASES OF THE BREAST

### ACUTE MASTITIS

**Mastitis Neonatorum** is a condition seen occasionally in infants after birth, when one or both breasts become slightly swollen and tender; only rarely will suppuration ensue, in which case an incision will be needed. Apart from this, no treatment is required.

**Mastitis of Puberty** is not an inflammatory process. At puberty the secreting part of the breast is developed, and its growth to normal young virgin size is rapid. When this occurs simultaneously on both sides, the condition is rightly regarded as normal, but not infrequently the development of one breast precedes the other. As some tenderness and throbbing will be present, the girl may be brought for advice. A simple explanation of the facts suffices. In boys a transient abortive effort at breast growth may also require similar sympathetic explanation.

**Acute Mastitis in the Adult.** *Etiology.*—The great majority occur during lactation, the remainder being due to spread from surface infection, suppurating hæmatomata or to pyæmia. During lactation there are certain predisposing factors, such as retracted or soft nipples, which may give rise to cracks and fissures, and when these have occurred, only the most meticulous cleanliness will prevent infection spreading into the breast. The infection, which is usually staphylococcal, travels either along the lymphatics in the interstitial planes or, rarely, along the ducts. Three stages in the process may be recognised.

**A. Milk Engorgement** is a condition best exemplified by the cessation of suckling, as when the child is weaned or kept from one breast for some local reason. The whole gland is then swollen, firm and tender, and throbbing pain may become severe. It occurs also as the first stage of breast infections, and affects either the whole organ or certain lobules only. The infection causes an inflammatory reaction around the ducts and also a swelling of their lining membranes. The flow of milk from these ducts is obstructed, and milk engorgement occurs in the lobules drained by them. A sector-shaped area of induration appears which is throbbing and tender. The use of the breast is possible but distinctly painful. There will be slight malaise, and the temperature will be about 99° to 100° F.

**B. Acute Non-suppurative Mastitis.**—If the condition of milk engorgement is not relieved the infection will progress deeper into the lobule, and the acini, filled with stagnant milk, afford an admirable culture medium for the infecting organisms. The affected area becomes more indurated, more definitely localised, and the pain, throbbing and tenderness increase. The skin will be hot and red, and enlarged veins will be seen coursing over the breast. The temperature is up to 101° to 102° F., and the patient is now definitely ill.

**C. Acute Suppurative Mastitis or Breast Abscess.**—The condition changes only in that softening occurs in the centre of the indurated area and fluctuation becomes apparent. The general condition of the patient rapidly deteriorates. Three types of breast abscess are described according to their position :

1. The *premammary* (or areolar) abscess occurs when the infection remains localised around the main ducts beneath the areola. The skin is early involved and the pus remains superficial.

2. The *intramammary* abscess is the usual type in lactational mastitis. It is commonly limited to one or two lobes, a sector-shaped area of induration resulting, but the whole breast may be affected. There is a tendency, particularly in deep-seated abscesses, for the pus to track, which results in a loculated cavity (Fig. 232).



3. The *retromammary* abscess occurs behind the breast and often behind the deep fascia. It is due to infected hæmatomata or rarely to a neglected empyema spreading through the chest-wall. A chronic abscess in this position is due to tuberculous disease of the ribs. These abscesses give a very characteristic clinical picture; the breast, which is normal, is pushed forward and appears to be floating on the surface of a fluid cushion.

*Treatment.*—Acute mastitis during lactation is due to neglect and uncleanness and should therefore never be allowed to occur. Efficient antenatal care and proper attention after every feed would succeed in abolishing breast abscesses altogether.

The treatment of milk engorgement during the weaning of the child needs no description here. When it is due to cracked nipples or threatened infection, the first decision concerns the continuation of milk secretion. If the usual time for weaning is near at hand, if the supply has never been good and the child is not thriving, or if the mother's condition is poor, then lactation should be terminated. If, however, the mother is strong and the infant is only a few weeks old and thriving well, every effort must be made to preserve the milk supply. The child will be confined to the unaffected breast and any deficiency made good by

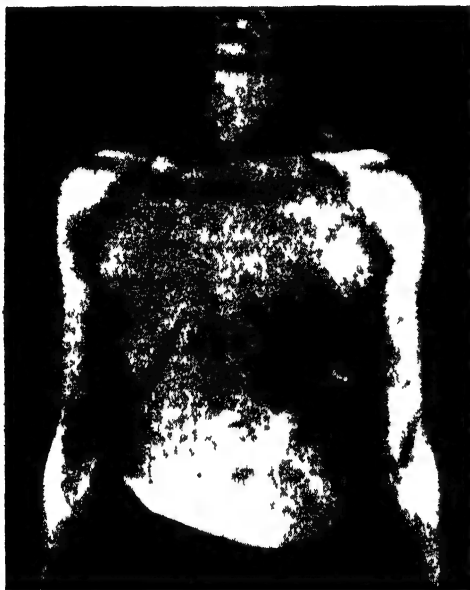


FIG. 232

A long-standing lactational infection of the left breast, accompanied by extensive eczema of the skin

one or two bottles. The affected breast must be emptied *completely* every four hours by a breast-pump, and in the intervals swathed in hot dressings and firmly supported. The patient should be kept in bed and adequately, but not severely, purged.

With the onset of acute mastitis, the same treatment is continued. The desirability of weaning the child becomes more urgent, and a careful watch is kept for signs of suppuration. When pus forms lactation must be stopped and an incision made over the swelling radially from the nipple. The septa dividing the cavity into loculi must be broken down with the finger and a tube inserted to drain its most dependent part. Retromammary abscesses are drained by a curved incision below the breast in the line of the skin reflection on to the chest-wall.

### CHRONIC MASTITIS

*Etiology.*—Chronic mastitis of inflammatory origin is known to follow the incomplete resolution of an acute mastitis. Clinically, it is



indistinguishable from chronic interstitial mastitis, but the history of previous infection and a small round-celled infiltration define the latter sharply from the former. The term "chronic mastitis" is commonly restricted to the non-inflammatory condition.

The etiology of such chronic mastitis is imperfectly understood. Infection has no part in its causation, and it is probable that it is the result of a disturbance of those endocrine factors which control the activity of the mammary epithelium. From puberty to menopause the female breast is in varying stages of activity. Apart from pregnancy, the breast epithelium undergoes proliferation and regression during each menstrual cycle. At the menopause widespread atrophic changes occur. Functional activity is known to be influenced by both ovarian hormones, oestrin and lutein, which are under the control of the anterior pituitary hormones. These latter may also possibly exercise direct control upon the breast epithelium. Repeated injections of oestrin will produce chronic mastitis in experimental animals. It seems probable, therefore, that some error in hormonal control plays a leading part in the causation of this disease. It does, however, occur in men, and in both sexes it is known to follow direct injury to the breast. The variety of the microscopic appearances suggest that many influences are at work, and at present the etiology remains obscure. It occurs most commonly during the years preceding the menopause, but is not infrequent in young women following injury. The incidence is slightly higher in women who have borne children, and miscarriage seems to be a predisposing factor.



FIG. 233

Chronic mastitis with multiple cysts.

*Naked-eye Appearance.*—A cross-section shows a thickened area of breast tissue, dull ivory-white in colour, with or without cysts. When present these vary in size and number; sometimes one large cyst dominates the picture, while in others multiple small cysts are scattered throughout the tissue. They are filled with a brownish fluid and when exposed have a blue colour, giving rise to the name "blue-domed cysts" (Fig. 233).

*The Microscopic Appearance* is very complex. The condition so well defined clinically includes widely differing cellular changes. Essentially there is a proliferation of interstitial connective tissue leading to interference with the acini and their ducts, some of which may become occluded, causing the acini to distend behind them and form cysts. The epithelium reacts in different ways. In some cases the acini are so compressed that they undergo atrophy, and slit-like spaces lined with a flattened epithelium are seen lying in a dense mass of fibrous tissue. In others the epithelium proliferates, filling up and distending the acini. In many specimens these two processes of atrophy and hyperplasia occur side by side.

*Relationship to Carcinoma.*—Considerable conflict of opinion exists

concerning the precancerous status of chronic mastitis. Some observers deny the existence of any etiological association, which has undoubtedly been exaggerated in the past. Nevertheless it is wise to regard chronic mastitis as a possible, if infrequent, precancerous condition.

*Clinical Features.*—In young women a history of injury, often during some game, will be obtained. In all, the earliest and only symptom is pain. At first it may be merely discomfort, but later a dull, aching pain becomes constant. Its severity varies widely in different patients. It may be felt in one segment only or referred to the whole breast, and occasionally shoots up into the axilla and down the arm. It is worse just before and during the menstrual period, and is aggravated by severe work or exercise, entailing prolonged use of the pectoralis major muscle. If the patient notices a lump in the breast it is because her attention has been directed to it by the pre-existing pain.

The findings on palpation depend on the degree of cyst formation. Chronic interstitial mastitis without cysts produces one or more indurated lumps in one or both breasts. The mass can hardly be felt with the flat of the hand, but when it is picked up between finger and thumb a coarse or knotty feeling can be appreciated as if a ball of inextricably tangled thick string were being palpated beneath the skin. The presence of cysts of small size make the nodular feeling more pronounced, and a single large cyst forms a definite tumour surrounded by typical mastitic tissue. More rarely the condition may be limited to one or more contiguous lobes, when a sector-shaped wedge of induration results. This is described as chronic lobar mastitis. In all cases the indurated areas are tender.

*Diagnosis.*—The non-cystic mastitis can never be mistaken for anything else, once its typical feeling has been learnt, the occasional case of tubercular mastitis providing the only difficulty. But the cystic type may readily be confused with carcinoma and less frequently with fibro-adenoma. The diagnosis of a cyst can be quickly verified by aspiration (see p. 523).

*Treatment.*—The possibly precancerous character of chronic mastitis has some bearing upon treatment. In young women it is never justifiable to waste time on palliative measures, and a course of X-ray treatment is begun at once. Its results are excellent in early cases, but in those which fail to respond, an excision of the indurated area must be advised. In older women, in whom no cysts can be felt and no doubt exists as to the diagnosis, treatment is symptomatic. Pain is often relieved by a course of X-ray treatment and the breasts need firm support. Potassium iodide and thyroid extract are given by the mouth. In mild cases the breasts must be properly supported by a really well-designed brassiere, and never allowed to hang down. If no improvement takes place, if the pain is said to be intolerable, if cysts are present, if any doubt as to the diagnosis exists, and if the patient is consumed with the fear of cancer (which is often the case) then an operation should be performed. If the area is strictly localised a local excision of the lump may suffice, but if, as is often the case, the condition affects the whole of the breast, the breast disc should be removed.

### CHRONIC NON-TUBERCULAR ABSCESS

This condition is occasionally seen as the result of a low-grade infection in a hæmatoma, in a previously existing retention cyst, or in connection with an imperfectly resolved acute mastitis which did not form an acute abscess at the time. It forms a slowly increasing swelling which is painful and tender. The breast is enlarged and pushed forward, and there is an indurated area occupying a large part of the organ. The thickness of its wall may make fluctuation difficult to obtain, and this may be so misleading as to suggest a rapidly growing carcinoma. The entire abscess with its walls should be dissected away if possible ; otherwise it is opened and drained.

### TUBERCULOUS MASTITIS

*Etiology.*—Tuberculous infection of the breast is rare. It occurs between the ages of 20 and 35, is unilateral, and in over 60 per cent. of cases is secondary to a known infection elsewhere, *e.g.*, the cervical or axillary glands or lungs. It may reach the breast by the blood stream, the lymphatics, or by direct spread. It has been reported as having spread from the nipple, but has never been known to occur in men.

*Pathology.*—At the beginning the disease is limited to one or two lobules, the acini being surrounded by tuberculous granulation tissue. More fibrosis is seen in the breast than is common in tuberculous lesions elsewhere. The process slowly spreads, caseation occurs in the centre and later multiple abscesses are formed with walls of typical tuberculous tissue, surrounded by dense fibrosis. Eventually a small shrunken breast results with several sinuses.

*Symptoms.*—Dull, aching pain first calls attention to the breast of a patient who has usually had a history of tuberculosis elsewhere. The clinical signs fall into three stages. First, there are areas of induration impossible to distinguish from those of chronic mastitis. Secondly, the fibrosis has increased to such an extent that the tumour is very hard, the nipple retracted and the skin fixed, while enlarged axillary glands may be felt. The mimicry of carcinoma is complete. In the third stage caseation and possible sinus formation reveal the true nature of the condition. In most cases the diagnosis is not difficult if care is taken to obtain a proper history ; nevertheless, it provides many mistakes.

*Treatment* consists in the removal of the breast and axillary glands, followed by general treatment for tuberculosis.

### SYPHILIS OF THE BREAST

This is very rare. A primary chancre of the nipple and areola may be seen, and condylomata occur on the lower part of a pendulous breast. Gummata are occasionally met with, forming dense areas of induration which become fixed to the skin and later break through to form a typical gummatous ulcer.

### ACTINOMYCOSIS OF THE BREAST

This is also very rare. It may reach the breast by direct implantation, by spread from the pleura or by a blood-stream infection. It produces one or more areas of induration and is indistinguishable from chronic mastitis until it breaks down and involves the skin, when the typical yellow granules in the discharge reveal the nature of the condition.

### FAT NECROSIS

This interesting condition is not so rare as is generally taught. It is invariably the result of injury, which is usually in the nature of a direct blow, but which, less commonly, is indirectly communicated to the breast by violent contractions of the pectoralis major muscle; still less frequently it follows subcutaneous saline infusions into the chest-wall. As a result of trauma fat is released from its enclosing membrane and is set free in the interstitial tissues, where it is acted upon by enzymes and converted into fatty acid salts. A round-celled and foreign body reaction is called forth and a palpable tumour is formed. Clinically, fat necrosis of the breast presents itself in two ways.

**Acute Form.**—This is well illustrated by a young woman of 25 years of the leisured classes who suddenly started to dig furiously for victory in heavy soil. After an eight-hour day she was awakened in the early hours of the following morning by a severe pain in the left breast. Within twenty-four hours so severe were the pain and tenderness that a breast abscess was diagnosed. When first seen the swelling in the upper quadrant was so tender that no adequate examination was possible, but no other sign of inflammation was present.

Local heat and intensive short-wave therapy led to complete resolution within ten days.

**Chronic Form** is much more common than the acute. A hard mass is formed by the fibrous tissue reaction so that it is frequently mistaken for carcinoma. If rapid improvement does not follow short-wave therapy, the swelling should be excised.

### CYSTS OF THE BREASTS

*Etiology.*—Cysts are commonly found in the breast in association with many of its diseases. The following classification is the simplest :—

1. Those connected with the main ducts : Galactoceles ; simple retention cyst.
2. Those connected with the small ducts and acini : Cysts of chronic mastitis.
3. Those connected with benign new growths : Duct papilloma ; fibro-adenoma ; cystadenoma.
4. Those connected with malignant new growths : Degeneration cysts.
5. Lymphatic cysts : Cystic lymphangiomata.
6. Blood cysts : Serous cyst following hæmatoma.
7. Parasitic cysts : Echinococcal.

**Galactoceles** is a retention cyst occurring during lactation and containing milk. The cause of the obstruction is unknown, but scarring from injury or from fissures may account for some of the cases. The cyst is situated beneath the areola and neighbouring skin and rarely exceeds 3 in. in diameter. It may persist after lactation has ceased and then the contents vary from milk to a thick, inspissated, cheesy material. It may increase in size during suckling and exude a milky discharge from the nipple on pressure. No treatment is indicated during lactation unless the cyst becomes very large. Repeated aspiration will cure many, while others will require complete removal through a radial incision.

**Simple Retention Cysts** are similar to galactoceles except that they do not occur during lactation. They contain clear fluid and are often accompanied by a slight induration in the corresponding lobe of the breast. They are very rare, and if any doubt exists as to the presence of a duct papilloma, they should be excised and examined microscopically. Obvious simple cysts may be aspirated.

Cysts arising from other causes will be described under the appropriate disease.

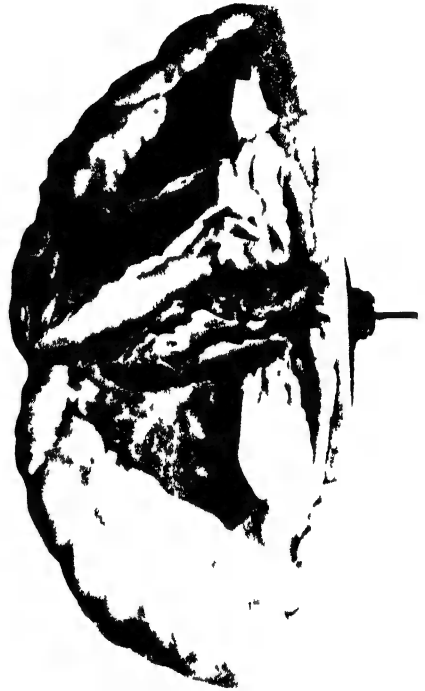


FIG. 234

Duct papilloma of the breast.

### GROWTHS OF THE BREAST

The new growths of the breast may be classified thus :

	<i>Epithelial.</i>	<i>Connective Tissue.</i>	<i>Mixed.</i>
Benign . . .	( Duct papilloma. Pure adenoma.	Fibroma. Lipoma.	Fibro-adenoma. Cystadenoma.
Malignant . .	Carcinoma.	Sarcoma.	Teratoblastoma.

#### DUCT PAPHILLOMATA

These are small pedunculated growths (Fig. 234) arising in the larger lactiferous ducts, and may be multiple. They are covered with columnar or cubical epithelium and give rise to a serous discharge from the nipple without pain or thickening in the breast. The discharge may be blood-stained at times ; in some cases it will cease altogether, after which a small tumour can be felt in the affected lobe, owing to cyst formation occurring in the obstructed duct. On examination it will be seen that the discharge always comes from the same orifice and pressure over one localised area will produce it.

Papillomata show a tendency to become malignant and for this reason, and because they may be multiple, no local removal should be considered. The breast disc is removed with the nipple and areola.

### PURE ADENOMA

This is a rare tumour seen in girls and young women, forming a soft, smooth, rounded swelling in the breast. It is encapsuled and consists of normal-looking breast tissue, except that the tubules have no lobular arrangement and there are no ducts. It may grow to a large size and is better removed.



FIG. 235

Microscopic appearance of an intracanalicular fibro-adenoma of the breast.

[ The benign connective tissue tumours are all described as occurring in the breast, but are more or less pathological curiosities. A fibroma may occur within the breast or as a pedunculated tumour from beneath the skin of the areola. A lipoma may be found in the breast either as a localised or diffuse type.

### FIBRO-ADENOMATA

These are the commonest benign tumours in the breast. Two varieties are described, "hard" and "soft."

**Hard Fibro-adenoma.**—This, the common type, occurs in women between 20 and 45 years of age, the majority being between 20 and 30 years. It is an encapsuled and lobulated tumour with a smooth surface, having a small vascular pedicle and being separated from

surrounding breast tissue by loose areolar tissue. Microscopically two types are described, pericanalicular and intracanalicular. The pericanalicular shows a dense overgrowth of connective tissue around the acini, which are compressed and appear as small tubules with flattened epithelium lying in a mass of fibrous tissue. In the intracanalicular type the fibrous tissue hyperplasia affects the acini, which are distorted and drawn out into narrow chink-like spaces (Fig. 235).

Clinically it forms a tumour which is painless, and which the patient notices accidentally when washing. It is firm, but elastic, and has a smooth lobulated surface. It is so freely movable that it constantly slips away from the examining finger, and this movement is independent of the breast tissue. They may be multiple in the one breast or present in both.

Fibro-adenomata should be removed as they are always a source of anxiety, and carcinoma has been known to arise in them.



FIG. 236

Soft fibro-adenoma of the breast.



FIG. 237

A cyst-adenoma of the breast.

**Soft Fibro-adenoma** is a rare growth. It is soft, rapidly growing, and its cells are of an embryonic type. It consists of large acini lined by tall columnar cells lying in a fibrous tissue matrix, which is unusually cellular. So rapidly do they grow that they may break through the skin by pressure atrophy and fungate on to the surface (Fig. 236). It is suggested that this tumour may arise in the intracanalicular fibro-adenoma, and it has been given many names, *e.g.*, the serocystic sarcoma of Brodie. It is, however, unquestionably benign. The diagnosis from cystadenoma, encephaloid carcinoma and sarcoma may be extremely difficult. In every case the breast should be removed.

**Cystadenoma.**—It has become customary in recent years to describe this tumour as belonging to a special group, though it is doubtful if this can be upheld histologically. It is distinguished by its tendency to form large cysts filled by rapidly growing intracystic papillomata (Fig. 237). Essentially it is fibro-adenomatous in origin. Clinically it is a rapidly-growing encapsulated tumour behaving like the soft fibro-adenoma, and if left it bursts through the skin, although remaining benign throughout. It is



seen in women between 35 and 45 years of age, advances rapidly to vast size, but remains freely movable and gives rise to no enlarged glands.

*Treatment* is removal of the breast.

### CARCINOMA

*Etiology.*—In susceptibility to cancer, the breast is second to the uterus; in every twenty-seven cases of cancer in women eight are in the breast. The epithelium of the mammary gland is constantly undergoing changes of activity and involution, and it is possibly these changes which render the breast so susceptible to carcinoma. There is some slender evidence that growth may follow an injury, and long-continued irritation from an unduly prominent corset bone may be a causative factor. The hyperplastic and cystic types of chronic mastitis and duct papillomata are recognised as precancerous conditions. It is seen most frequently between the ages of 40 and 60, but after 20 no age is exempt. It is as common in married as in unmarried, in parous as in nulliparous women. The left breast is more often affected than the right, and the commonest situation is the upper and outer quadrant, the other areas in order of frequency being the outer and lower, the upper and inner, the lower and inner quadrants and the axillary tail. Age has a marked influence on the rapidity and virulence of growth, the younger the patient, the more rapid and more deadly its advance, whereas in the aged the growth may be locally very slow and spread to the glands may take months or even years.

*Varieties.*—Histologically it is possible to divide carcinoma of the breast into groups. They are:

A. The spheroidal or polygonal-celled carcinoma scirrhus, atrophic scirrhus, and encephaloid.

B. The columnar-celled or duct carcinoma.

As these are also clearly defined clinically they will be described separately.

**The Spheroidal or Polygonal-celled Carcinoma.**—The great majority fall into this group, and in it three types are described: atrophic scirrhus, scirrhus and encephaloid. The differentiation is based on the amount of fibrous tissue in the growth, and a clear understanding of the processes involved is essential. Fibrous tissue is the response of the body to attack, its main line of defence. With the earliest onset of carcinoma a round-celled infiltration is immediately mobilised around the cancer cells. If the virility of these cells is so low that the round cells are given time to consolidate into fibrous tissue, then the cancer cells may be completely encapsuled. This constitutes an *atrophic scirrhus*. When, however, the cells of the growth have intense activity, they divide and spread so rapidly that each newly laid-down zone of round-celled defence is swamped long before it can form fibrous tissue, and this is the *encephaloid* type. The middle group is intermediate in every sense, in that the activity of the cancer cells is not sufficient to prevent fibrous tissue being laid down, but is enough to prevent their being completely cut off. This is the *scirrhus* group, which includes the majority. It will be appreciated, therefore,



that there is a multitude of tumours varying in the proportionate amounts of growth and fibrous tissue, forming in this way a long series, beginning in the encapsuled atrophic scirrhus and passing through many intermediate stages to end in the rapidly fatal encephaloid cancer. The division into three groups is convenient but arbitrary, and it does not permit of every growth being relegated to one or other group with the expectation that it will behave in an identical manner with every other member of that group.

#### *Naked-eye Appearances*

—**The Atrophic Scirrhus** is a small spherical nodule of dense ivory-white tissue with minute yellow or white areas in the centre. The breast in which it occurs is usually also small and atrophic (Fig. 238).



Fig. 238

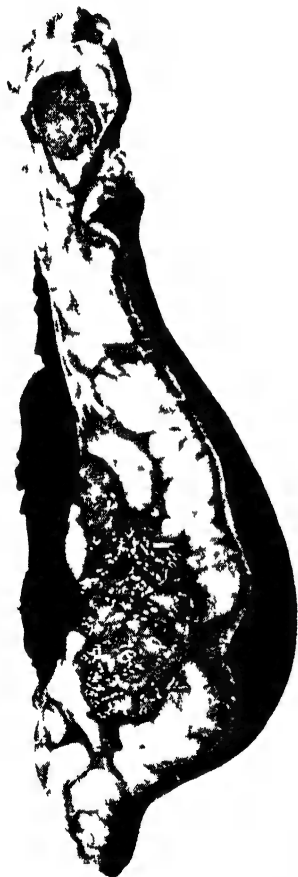


Fig. 239

of a breast showing retraction of the nipple, the typical appearance of the cross-section of this type of growth and the invasion of the axillary lymph glands

**The Scirrhus** has so typical an appearance that it allows a diagnosis to be made with certainty in early cases in which clinical methods leave it in doubt. On cutting such a growth open, the knife is gripped in a curious way, the impression being gained that the cut surfaces are attempting to hold the sides of the scalpel, so that a fine vibratory thrill is communicated both to the fingers holding the scalpel and to those supporting the tumour behind. When the section is complete, the cut surfaces become concave. The growth is pinkish white and radiating processes are seen passing into the surrounding fat. The centre shows white and yellow areas due to necrosis, and, when the edge of the knife is scraped across the surface, irregular gritty areas can be felt, and a thick, yellowish fluid collects on the blade (Fig. 239).

**The Encephaloid Carcinoma** is a soft, grey, homogeneous growth said to resemble brain tissue. It has large vessels running over and through it.

*Microscopic Detail* —The amount of fibrous tissue varies greatly, as has already been described. The growth is of the carcinoma simplex type, the cells forming a closely packed mass without any attempt at differentiation. The shape of the cells is governed by pressure, and they take on irregular polygonal forms. Sometimes they show some indication of a regular arrangement, and

papillary, alveolar and adenocarcinomatous forms are described. The rapidity of the growth can be judged by the amount of fibrous tissue and the number of mitotic figures present (*vide* Fig. 39, p. 108).

*Method of Spread.*—Carcinoma of the breast spreads by (1) infiltration, (2) permeation, (3) embolism and (4) transcœlomic implantation. These are fully described on p. 85.

Carcinoma of the breast spreads by these methods to :

1. Regional lymph glands in the axilla and above the clavicle ;
2. Skin and opposite breast ;
3. Bones especially ribs, sternum, dorsal and lumbar vertebræ and humerus ;
4. Lungs and mediastinal glands ;
5. Liver ; and
6. Any part of pleura and peritoneum by transcœlomic implantation.

**Scirrhus Carcinoma.**—*Clinical Features.*—In over 95 per cent. of cases the patient first notices a lump in her breast. This discovery is made quite accidentally during washing. There has been no pain to call attention to the breast. In a few cases sudden and rapid increase in size is the first noticeable symptom, while in others it is a small ulcer or a peculiar dimpling of the skin.

The picture of a moderately advanced growth will be described first, and the early signs and late complications discussed later. On inspection it is seen that the affected breast is :

- (A) Smaller than the other.
- (B) Raised to a higher level than the other, and
- (C) The nipple is retracted.

These signs are due to contraction of the new fibrous tissue of the growth infiltrating the ligaments of Cooper.

- (D) The tumour may be visible.
- (E) The skin may be dimpled, puckered or retracted (Fig. 240).

On palpation of the breast, a tumour will be felt which has the following characteristics :

1. It is either regularly spherical or flattened from back to front.
2. It is very hard ; comparable to a stone.
3. Its surface is irregular, rough and craggy.
4. Its edge is indefinable as it fades imperceptibly into normal tissue, and it is impossible to say where one starts and the other ends.
5. It is fixed to the skin and underlying deep fascia.
6. It is accompanied by enlarged glands in the axilla.

*Early Signs.*—The above description has been accepted as the typical textbook description of a carcinoma of the breast. It is, however, characteristic of a moderately advanced type in which the hope of lasting cure is small. Carcinoma of the breast can be, and should be, diagnosed when no sign exists except the presence of a lump,

when the breast is not yet distorted and displaced, when no fixation and no enlargement of glands has occurred. The physical characteristics of the lump, viz., its hardness, its rough, craggy surface and its indefinable edge should provide a diagnosis. If any doubt exists the tumour should be explored.

*Late Signs.*—A. The skin may be affected in several ways. *Peau d'orange* or "pigskin" (Fig. 241) is produced by blocking of the lymphatics draining the skin by cancer cells or by fibrosis. In this way the affected area of skin becomes swollen except where the hair follicles and sweat glands penetrate it, and as a result their orifices



FIG. 240

Carcinoma of the breast showing fixation to the skin. The surface has been painted with fluorescein prior to X-ray treatment.

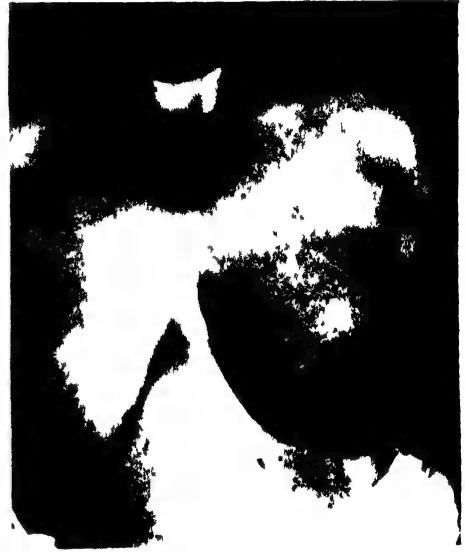


FIG. 241

Carcinoma of the breast showing *peau d'orange*.

become accentuated and the orange-skin dimpling is closely mimicked. The skin becomes thickened and leathery. "*Cancer en cuirasse*" is the name given to a more advanced condition in which the leathery texture of the skin has become much more marked, the colour has turned to purple and crusts have formed on the surface. Hard nodules of growth are present on the skin, the cells having reached this level by permeating the cutaneous lymphatics. A carcinomatous ulcer may form as a result of direct spread from an underlying growth (Fig. 242) or from the ulceration of a secondary nodule (Fig. 243). It presents the typical hard, everted and raised edges.

B. "*Brawny arm*" is due to widespread lymphatic obstruction around the shoulder region, preventing any collateral lymph circulation being established. Blockage of the main axillary trunks alone is not sufficient to produce it. The oedema at first pits on pressure, but soon becomes hard and brawny. The arm swells to twice or thrice its normal size and the skin is tense and shiny. Pain becomes continuous and paralysis finally occurs.

*Diagnosis.*—This rests between hard fibro-adenoma, chronic mastitis and carcinoma. The differentiation is shown in tabular form (p. 523).

*Prognosis.*—In those cases in which lymphatic glands are palpably enlarged the chances of complete cure are not good. When, in addition, the skin is ulcerated or the chest-wall infiltrated, then a fatal issue is likely. In very old women quite a large superficial growth can be present without any metastases, and the prognosis is good. But in every case, particularly in the early cases, it is most unwise to give a prognosis until the pathologist has reported on the material. The amount of fibrous tissue, the number of mitotic figures and the presence of cancer cells in the lymphatic glands are by far the most reliable guide.



FIG. 242

Ulcerating carcinoma of the breast.

*Treatment.*—1. Definition of operability. A case is operable unless the growth has infiltrated the chest-wall,

caused cancer *en cuirasse* or produced a fixed mass in the axilla, enlarged fixed supraclavicular glands or definite metastases at a distance.

2. Exploration of the small doubtful tumour. The early cases with no signs except the lump in the breast may cause uncertainty. In these cases no words can condemn sufficiently strongly the attitude of "wait and see." Such a policy may result in a curable growth being allowed to become incurable (though not necessarily inoperable). The growth must be explored, and permission obtained for the performance of the radical operation if found necessary. Under general anæsthesia the tumour is either cut into *in situ* or removed completely and cut open outside the body. The naked-eye appearances are pathognomonic. If they prove carcinoma, a small swab soaked in pure carbolic acid is placed in the wound and the skin sewn up over it. Towels, gloves and instruments are changed and the radical operation is performed.



FIG. 243

Ulcerating secondary nodule in skin after radical mastectomy.

3. The radical operation. The principles governing this operation are based on the study of lymphatic permeation. A small area of skin needs removal, but a wide one of deep fascia, as this is the main plane of lymphatic spread. For this reason, skin flaps need to be cut sufficient to expose the necessary area beneath. This is bounded by the clavicle above, the opposite edge of the sternum internally, the

## DIFFERENTIAL DIAGNOSIS OF SWELLINGS IN THE BREAST

	FIBRO-ADENOMA.	CYSTIC MASTITIS.	CARCINOMA (Early).	CARCINOMA (Moderately Advanced).	SOFT FIBRO-ADENOMA; CYSTADENOMA.	ENCEPHALOID CARCINOMA; SARCOMA.
Symptoms	Lump noticed by chance.	Pain.	Lump noticed by chance.	Lump.	Lump.	Lump.
Size of Breast	Normal.	Normal or a little enlarged.	Normal.	Shrunken.	Enlarged.	Enlarged.
Position of Breast	Normal.	Normal.	Normal.	Elevated.	Pushed forward.	Pushed forward.
Nipple	Normal.	Normal.	Normal.	Retracted.	Pushed forward.	Pushed forward.
Skin	Normal.	Normal.	Normal.	Puckered, dimpled <i>peau d'orange</i> .	Thinned. Gives way. Fungating mass not attached to skin edge.	Early <i>peau d'orange</i> Thinned and gives way. Fungating mass attached to skin edge.
Consistency	Solid, firm, elastic.	Solid, elastic.	Stony hard.	Stony hard.	Soft. Cystic in parts.	Soft. Very vascular.
Surface	Smooth always. Lobulated often.	Cyst smooth;ropy. knotty covering.	Irregular. Craggy.	Irregular. Craggy.	Smooth. Lobulated.	Smooth. Lobulated.
Edges	Clearly defined.	Fairly defined.	Indefinable.	Indefinable.	Well defined.	Fairly defined.
Fixation	Nil.	To breast tissue.	To breast tissue.	To breast tissue, skin, deep fascia.	Nil.	To everything.
Glands	Not enlarged.	Not enlarged; occasionally large, soft, tender.	Not enlarged.	Enlarged.	Not enlarged.	Great enlargement in encephaloid. None or moderate in sarcoma.

first fibrous intersection of the rectus abdominis muscle below and the anterior border of the latissimus dorsi externally. Such an incision enables an extensive clearance to be made, with ease of skin apposition afterwards. The tissues to be removed are :

- (a) An area of skin between 4 to 6 in. in diameter according to the size of the growth, which is at its centre. This area must include the nipple and areola.
- (b) The whole breast.
- (c) The sternal part of the pectoralis major muscle.
- (d) The whole of the pectoralis minor muscle.
- (e) The fascia over the serrations of origin of the serratus magnus and external oblique, and the anterior sheath of the rectus abdominis muscle as far as the first intersection.
- (f) The whole of the fibro-fatty-lymphatic contents of the axilla.

The apposition of the skin is rarely attended with much tension, and drainage through a separate stab incision in the axilla prevents accumulation of blood and serum beneath the flaps. The full technical details can be found in textbooks of operative surgery.

4. Palliative removal. In old women a slowly growing carcinoma may reach some size before they seek advice. At first sight its size and the evidently imminent ulceration of the skin lead to an opinion that it is inoperable. On closer examination no glands can be detected, and the degree of fixation to the pectoral fascia is not advanced. In such cases it is certainly justifiable to advise a local removal of the breast without attempting the radical operation. The danger, discomfort and pain of an ulcerating growth are thereby obviated, and it is surprising how good are the results.

5. After treatment. In every case prophylactic X-ray treatment should be advised. Although it may not be absolutely necessary in the early case, nevertheless it should be adopted as the routine procedure.

6. The position of radium. A great deal of research work has been done in connection with breast cancer. The results are not encouraging so far as final prognosis is concerned. Early growths are destroyed as successfully as in the tongue, but the five-year results are not as good as those following radical removal, which still remains the treatment of choice.

**ATROPHIC SCIRRHUS.**—This type of mammary cancer provides the best example of the human body's attempt to destroy a malignant process and achieve a natural cure. The cancer cells are of such low vitality and divide so slowly that an extensive fibrous reaction surrounds them. Patients may live for many years with such a growth and die from intercurrent disease, or, after several years and for no accountable reason, widespread dissemination may occur. Such an example is in the St Mary's Hospital museum; the primary growth (Fig. 238) being a tiny atrophic scirrhous in a shrunken breast which, after many years, gave metastases in almost every bone in the body. These growths may form so small a tumour that the patient remains unaware of their presence until puckering of the skin or

ulceration draws attention to them. Some remain in this condition, while in others fibrosis spreads throughout the breast, which becomes shrunken to such an extent that it resembles the male breast. Sampson Handley advises removal of the breast disc in every case, for, as he says, no one can foretell when an atrophic scirrhus may blossom forth into active dissemination.

**Encephaloid Carcinoma.**—In these growths the intense activity of the cells allows no time for a defensive fibrosis to be laid down. A very rapidly growing soft homogeneous mass resembling the grey matter of the brain is formed. It consists of a pure polygonal-celled carcinoma simplex with no attempt at alveolar arrangement. It is fortunately a rare condition occurring in younger women before the age of 35 years, in whom a rapid enlargement of the breast is noticed. None of the typical signs of scirrhus are present. The skin early develops *peau d'orange* and is quickly broken through, a fungating mass being formed which differs widely from the ulcerating scirrhus (Fig. 244).



FIG. 244

A fungating encephaloid carcinoma.

Glands in the axilla are enlarged to great size, and the growth disseminates early and widely to all parts of the body, leading to a fatal issue within a few months. It is doubtful if any attempt at operative treatment is justifiable. Heavy radiotherapy may check the speed of growth, but does not alter the inevitable result.

**Acute Inflammatory Carcinoma.**—This rare type is similar to the encephaloid, but it is even more rapidly growing, and so great is its vascularity that the breast becomes red, hot and throbbing. It is usually, but not invariably, seen in lactating breasts. It may be mistaken for a breast abscess so sudden is its onset, so rapid its growth and so suggestive are the signs of inflammation. These cases are inevitably fatal and no treatment is of any avail.

**Peripheral Carcinoma.**—Carcinomata of varying degrees of activity, particularly at the atrophic scirrhus end of the series, sometimes arise in the terminal parts of the alveoli at the periphery of the breast. As these may lie outside the apparent gross limits of the breast, the condition is not always recognised as mammary. It is for this reason that this group is specifically mentioned. Growths of the axillary tail are comparatively common, the crease where the skin of the breast is reflected on to the chest-wall may provide examples of these peripheral growths, and Handley has recorded cases exactly in the midline of the sternum between the breasts. They are all polygonal-celled tumours of the atrophic scirrhus type. With regard to prognosis, they are nearer to the main lymphatic fields, and dissemination is apt to occur earlier

than in the case of a central mammary cancer of equal growth rate. They usually attract attention as little ulcers or by puckering of the skin, and the underlying tumour may be very small. It is imperative that these points should be understood, as such clinical signs may be passed over as of little importance, because they are not apparently connected with the breast.

**Columnar-celled Carcinoma.**—This tumour probably arises from the ducts and is therefore termed a "duct carcinoma." It is believed to arise from the malignant degeneration of a benign duct papilloma. Microscopically, dilated duct spaces will be seen filled with cellular debris and containing one or more papillary growths, the surrounding breast tissue being invaded by a columnar-celled cancer. Other forms of this growth produce an adenocarcinoma with well-formed acini, but even in them all the infiltrating cells tend to revert to the simple polygonal form. Clinically the duct carcinoma gives a discharge from the nipple, which is probably blood-stained, and a small nodule may be felt in the breast near the nipple. They are of slow growth, and although the skin may be affected early they disseminate late. The treatment is the same as for the polygonal-celled growths.

#### SARCOMA OF THE BREAST

Mammary sarcoma is very rare, the incidence being well under 1 per cent. of all breast tumours. It occurs usually before the menopause and sometimes in young girls. There is some evidence to suggest that it follows a blow. It may be a highly malignant round-celled type, or a less malignant spindle-celled growth. It is a rapidly growing tumour without pain and without any shrinkage of the breast or retraction of the nipple. It grows to a large size, filling the whole breast, and finally the skin gives way, a fungating mass being formed. The axillary glands are sometimes normal, but occasionally they are enlarged even before secondary infection is brought about by invasion of the skin. The tumour is very vascular, and severe, even fatal, hæmorrhages occur after fungation. Generalised dissemination occurs rather later than would be expected in a case of sarcoma. If seen early, a radical operation should be performed as for scirrhus carcinoma.

**Teratoblastoma** of the breast is a pathological curiosity and is of no clinical importance.

#### THE MALE BREAST

The male breast is very rarely the seat of pathological processes, but it may be the subject of any of the diseases which attack the female.

#### CHRONIC MASTITIS

Reference has already been made to the so-called mastitis of puberty, in which the boy is brought for advice because of a swelling in one breast. This attempt to develop a breast is more common than is generally supposed.



In later life true chronic mastitis may occur in the male, and in every respect is similar to that seen in the female.

*Treatment* is removal.

### CARCINOMA

This is very occasionally met with in the male. It may be either a scirrhus carcinoma simplex or a columnar carcinoma. The former is a small, hard tumour below the nipple or areola and has all the clinical appearances of the corresponding tumour in the female; the latter is somewhat more common, growing as it does from the rudimentary ducts attached to the nipple. It presents as a swelling from the surface and pushes the nipple and areola forward. Ulceration occurs in the later stages. In men these growths become evident in the early stages and the prognosis is usually good.

*Treatment* consists in radical removal.

R. M. HANDFIELD-JONES.

## CHAPTER XXVI

### THE GENERAL SURGERY OF THE ABDOMEN AND PERITONEUM

**S**URGICAL ANATOMY.—The anatomy of the abdominal wall, its muscles, nerve supply and general arrangement are described in Chap. XXVII under the heading of hernia. The abdominal viscera are described in their respective chapters and the anatomy of the peritoneum is dealt with later in this chapter.

#### ABDOMINAL INJURIES

The most prolific cause of abdominal injury throughout the centuries has been the warring instinct of mankind. Historical museums display portions of the lumbar spine with impaled flint arrow heads, some of which, imbedded in the anterior aspect of the vertebral bodies, limn with unerring surety the transabdominal course of the missile shot by some hand of the Pleistocene period, a dual monument to victor and slain.

Abdominal injuries have swayed and shaken international politics ; the gunshot wound of the abdomen which killed an Austrian Arch-duke at Sarajevo set alight the great European conflagration of 1914, while an assassin's bullet penetrated the stomach and pancreas of a famous President of a powerful transatlantic people.

When combatant armies fight at some distance from each other, as in modern war, injuries of the abdomen are no more frequent than those of other regions ; where proximity between foemen becomes closer, the belly constitutes a target and the tale of abdominal injuries must increase. Penetrating wounds are the prerogative of passion ; accident or "blast" or the capricious effects of air bombardment engender those injuries to the entrails in which no breach of the belly wall exists ; yet even in the sphere of accident the abdomen appears to be under the special protection of the wonderful muscular reflex of its encompassing parietes.

From time immemorial the belly has been regarded as a vulnerable area ; it has been hopefully explored by suicides from the days of the Roman Empire ; it is traditionally disembowelled in the Orient ; it has been utilised by murderers, sometimes in the guise of religious enthusiasts, and the most heinous crimes have often been committed in the name of the Church. It seems ironical to speak of "bowels of compassion," when the contents of the belly have been exposed to public view, even before death ; and when St Erasmus' intestine was coiled around a windlass by fanatics.

### NON-PENETRATING INJURIES

The abdominal parietes may suffer along with the subjacent organs, and ecchymoses and effusions of blood may be found in any of the anatomical layers superficial to the peritoneum. When visceral lesions are present, actual rupture of the abdominal muscles is rare, whereas the blow that ruptures the powerful abdominal muscles is often too spent to injure the intestinal tract. It is convenient to describe non-penetrating injuries as affecting (1) the abdominal wall, (2) the hollow viscera and (3) the solid organs.

**Injuries of the Abdominal Wall** deserve special consideration by reason of the difficulties which beset the diagnosis from concomitant damage to the subjacent abdominal organs. The lesions produced by subcutaneous injury, which demand special notice, include rupture of the rectus abdominis muscle, possibly with damage to the deep epigastric artery, and, more serious still, those contusions in which the peritoneum is torn as well as the superjacent musculo-aponeurotic structures, and a traumatic hernia is present under the intact skin.

### INJURIES OF THE HOLLOW VISCERA

The potentialities of a belly-blow in respect of damage to the alimentary canal are not negligible, and the various lesions resulting can be classified as contusions and ruptures. Statistics show that the small bowel is preponderantly liable to trauma, the jejunum being hurt far more frequently than the ileum; the duodenum takes third place in vulnerability, but the appalling mortality associated with its rupture affords eloquent testimony to the frequency with which the duodenal lesion is unsuspected in diagnosis and overlooked at operation.

**Contusion of the Intestinal Wall** may be single or multiple and, although spontaneous healing may result in mild cases, more serious damage may determine subsequent perforation from the separation of a gangrenous patch, the development of mucosal ulcers or the late advent of cicatricial stenosis. The first-mentioned complications are seen between the fourth and the fifteenth days, and their possible occurrence must be kept in mind.

**Rupture of the Intestine** may be complete or incomplete. In the latter one or more of the intestinal coats is torn, but the lumen of the gut does not communicate with the peritoneal cavity. The inner tunics are more frequently damaged than the outer; any subsequent necrosis or rupture of the thin remaining layer will lead to peritonitis. In complete rupture the opening in the bowel wall may be no larger than a pinhead, but on the other hand the gut may be completely divided.

The rupture may be caused by a compression or a crushing injury, by a bursting or a tearing force. In *compression* injuries the lesion is either small and rounded or elongated, in which latter case the tear runs transversely round the bowel and may constitute a complete section of the gut. The edges of the rupture are contused and crushed; but temporary protection against the escape of intestinal contents is

frequently afforded by the prolapse and eversion of the mucous membrane and by the contraction of the circular muscle fibres, to which Jobert de Lamballe first drew attention. The duration of this defensive mechanism varies between nine and thirty-six hours ; in many patients, however, the escape of intestinal contents is immediate, and this is especially true of the large bowel.

The uppermost portion of the jejunum is that most frequently ruptured in these non-penetrating injuries, the great majority occurring in the first, second and third coils where they cross the lumbar vertebræ. At the other extremity of the small intestine, the two terminal coils of the ileum come into contact with the sacral promontory and the right sacro-iliac synchondrosis, and rupture of the ileum, though less frequently encountered than the jejunal tears, is found to increase in proportion to the proximity of the coil to the ileocæcal junction.

Rupture by *bursting* is rare. Complete transverse severance of the bowel is never seen in this injury ; the wound usually has its long axis parallel to that of the intestine and is found on the antimesenteric border. Rupture by *tearing* is still more rare and is always across the long axis of the gut, so that total or subtotal tears are the rule.

*Symptoms and Signs*, arranged in order of their frequency, are :—abdominal pain and tenderness, rigidity, vomiting, rising pulse, shock, bruising of the abdominal wall and dulness in one or both flanks. The protective mechanism referred to above may be so perfect for the first few hours that there will be, in effect, no symptoms ; in many patients there will be a concomitant bruising and tenderness of the abdominal wall. It is these two factors which make the diagnosis of non-penetrating injuries of the intestine so difficult in many patients.

*Diagnosis*.—Zachary Cope emphasises the extreme importance of excluding injury to the spinal cord, chest and kidney ; and then urges that rupture of the intestine should be suspected (1) when the pain persists for more than six hours after injury, especially if accompanied by bilious vomiting, a gradually rising pulse, persistent local rigidity which tends to spread, and deep local tenderness ; and (2) when abdominal pain is absent or slight, but the pulse rate rises steadily and the patient is restless or listless. Grant Massie first drew attention to the great value of radiography in diagnosis in the early hours after injury. Free gas in the peritoneum can be seen by this means.

*Prognosis* depends on the time that is allowed to elapse before treatment is undertaken ; Siegel states that of those patients operated upon in the first four hours the mortality is only 15 per cent. ; those who underwent laparotomy between the fifth and eighth hour had a death rate of 44 per cent., and after the twelfth hour no less than 70 per cent. succumbed.

*Treatment*.—Perhaps the most cogent reason for laparotomy is *doubt* in the mind of the medical attendant. It is far better to look and find nothing than to waste valuable time in speculation until definite evidence of peritonitis, the harbinger of an early dissolution, has appeared. The intestinal injury is sought for and the wound closed ; simple suture may suffice, or resection and anastomosis may be needed

in total section of the bowel. Care must be taken to ensure that a second lesion does not coexist. The treatment of the peritoneum varies with the amount of the soiling; if the effused contents are strictly limited, dry sponging only should be employed and the belly wall may be closed without drainage; if the escaping contents are widely spread, gross infective material should be wiped up, the fluid aspirated by suction and drainage employed. The local and oral employment of sulphanilamide is to be encouraged.

### INJURIES OF SOLID VISCERA

Non-penetrating injuries of the solid viscera present the picture of internal hæmorrhage, which has been described fully in Chap VIII.

**Tears of the Liver** are most often the consequence of blows on the right hypochondrium or the lower part of the right side of the thorax; they may be produced also by contre-coup in the case of falls from a height. The degree of damage varies from a central rupture or sub-capsular hæmatoma to severe disruption and even complete separation of a portion of the liver, which may be loose in the peritoneal cavity. It is affirmed that the right lobe is damaged six times as frequently as the left and that the convexity of the organ is torn twice as often as the concave surface. The liver takes first place among the solid abdominal viscera in its liability to injury.



FIG. 245

Method of repair of the liver after rupture. Note the technique for preventing the coaptation sutures cutting out.

*Clinical Picture.*—In the grave form of injury shock is severe, there is tenderness over the liver area, diffuse rigidity of the abdomen and diminished amplitude of respiratory excursion. The signs of cataclysmic internal hæmorrhage will dominate the picture.

In less grave cases there may be only slight shock and localised tenderness over the hepatic area; the pulse will be slow and signs of mild jaundice develop subsequently. There may be the complaint of pain in the subscapular region when the convexity of the liver is torn or in the epigastrium when the tear is in the concave surface. Rectal examination may demonstrate that the pouch of Douglas is becoming progressively fuller (Lecène).

*Prognosis and Treatment.*—The minor degrees of injury recover spontaneously; the more severe demand instant surgery. It is said that between 60 and 80 per cent. of patients sufficiently injured to require immediate operation perish. Grey Turner issues the warning that the hepatic hæmorrhage is partly controlled by contraction

of the abdominal muscles and that as soon as this is relaxed under anæsthesia bleeding may become torrential. A preliminary drip-transfusion of blood should be started at once and allowed to continue throughout the operation. A midline incision is rapidly made and the bleeding controlled by compression of the structures in the free edge of the gastrohepatic omentum. Finally the bleeding is stopped by coaptation of the edges by mattress sutures (Fig. 245) or by packing the cavity with gauze impregnated with paraffin and flavine.

Rupture of the extrahepatic bile ducts is uncommon, and symptoms are due to intraperitoneal extravasation of bile. The treatment consists in drainage of the extravasated bile; possibly drainage of the gall-bladder may be superadded. Suture or drainage of the duct itself may be possible in the early cases.

**Rupture of the Spleen.**—Damage to the spleen may occur at any age of life, even in a new-born babe dropped on the floor in precipitate labour, and is not uncommon in children. The organ has been injured by violence of every degree, but the spontaneous rupture of a normal spleen has never quite satisfied critical inquiry. The proclivity of the abnormal spleen to spontaneous rupture is, of course, well known, and to the enlarged spleen of malaria attaches a special liability to this dramatic complication (Fig. 246).



FIG. 246

A spleen showing multiple radiating tears.

The *clinical signs* are those of grave intra-abdominal hæmorrhage, which may be catastrophic in its severity and suddenness, and shoulder pain, especially in the left side when the patient lies down or tries to sit up. Rigidity is not always present and is more common in children than in adults. There are also cases of prolonged slight hæmorrhage, in which the bleeding goes on slowly into the peritoneal cavity. There is a further group in which a latent period has lasted for more than forty-eight hours and been followed by the abrupt onset of a most severe hæmorrhage.

*Treatment* consists in immediate operation and removal of the spleen with ligature of its vessels.

**Injuries to the Pancreas** are very rare, but are also of interest from the possibility of the subsequent development of a pseudo-pancreatic cyst.

## PENETRATING WOUNDS OF THE ABDOMEN

The diagnosis of intestinal injury due to a penetrating wound in the abdomen is not beset with the same difficulty that obtains in deciding whether the bowel has been damaged by contusions or non-penetrating violence, and the indications for treatment are more clear. Whatever the traumatic agency, a stab with a knife, dirk or dagger, a sword or bayonet thrust, a wound from bullet, shell or bomb, a laceration by spike, stake or animal's horn, the general

principles are alike. A penetrating wound of the abdomen probably means a penetrating wound of bowel or solid viscus, and demands the earliest surgical interference (Fig. 247).

*Symptoms and Signs.*—An escape of fæcal material or flatus from a wound involving the abdominal parietes or even a part of the body remote from the cœlom is self-evident proof of an intestinal lesion. An abundant and persistent discharge of blood from wounds in the back, flank or belly-wall, an ebb which flows faster and with greater force when the patient coughs or makes an effort, will suggest some deep visceral injury. No comment is needed upon those cases where a portion of the abdominal contents projects from a wound, and still more significant will be the diagnosis if the patient is shocked and blanched, passes blood from the rectum or has a hæmatemesis.

The situation of the wound may not at first suggest an involvement of the peritoneal cavity or its contents. The wound of entry may be, for example, in the thorax or the buttock, injuries fraught with the gravest danger.

In addition to obvious evidence the following signs and symptoms assist in the diagnosis. Rigidity, pain and tenderness, vomiting, a rising pulse rate and an expression of profound anxiety are all suggestive. No one sign or symptom individually is diagnostic, but when they are present in combination the clinical picture becomes clear. An X-ray examination will give valuable information as to the direction of the missile and may furnish useful suggestions as to the best mode of approach in a particular case.

*Treatment.*—It may truly be said that “in the abdomen there are no insignificant wounds.” Every wound must be explored, excised and disinfected. Experience has taught that early operation improves the result immensely, but that the treatment of the intestinal lesions should be as conservative as possible. Wherever possible the gut should be repaired by suture and intestinal resections reserved for such injuries as must inevitably lead to leakage and peritonitis, unless dealt with radically.

**Wounds of the Large Intestine.**—Suture must always be preferred to resection, unless the latter seems inevitable. The whole experience of war demonstrates that this suffices in an overwhelming number of cases, since by reason of its size, wounds of the colon are usually tears and not complete divisions. Moreover, the absence of numerous loops and coils makes multiplicity of wounds unlikely. On the other hand, it must be remembered that it is extremely probable that the

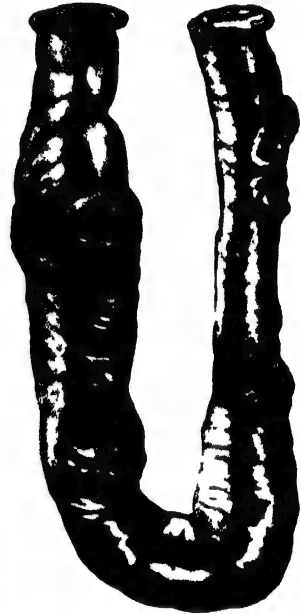


FIG. 247

Multiple gunshot wounds of the small intestine. Note the pouting of the mucous membrane.

wound is of a perforating nature, and the surgeon must satisfy himself as to the existence, or absence, of a retroperitoneal wound in addition to the more obvious and accessible peritoneal injury. These retroperitoneal injuries with consequent infection of the extraperitoneal cellular tissues are much more fatal than the intraperitoneal wounds.

Resection of the colon is reserved for those cases in which the blood supply is so damaged that a portion of its wall is infarcted; a colostomy may be needed in certain cases to prevent or ameliorate a retroperitoneal infection. The work of Vaughan Hudson, Rodney Smith and Selbie demonstrated the value of local sulphanilamide therapy.

**Penetrating Wounds of the Stomach** are not very common and are usually associated with severe intra-abdominal bleeding, which may come from the vessels on the stomach wall or along its curvatures. Vomiting and hæmatemesis constitute a feature of these wounds. The stomach and spleen are likely to be involved in abdomino-thoracic injuries and may sometimes be advantageously explored and treated across the thorax and diaphragm.

**Penetrating Wounds of the Liver.**—The dimensions of the missile play a considerable rôle in determining the type of hepatic lesion, which is almost protean in character, varying from a perforation, possibly with cracks and fissures radiating therefrom, or a superficial score, to a ragged wound or a crateriform cavity. The liver is damaged in almost every abdomino-thoracic wound on the right side and may be badly shattered in such injuries as the "stove in" chest. The whole organ may be disrupted even by a bullet wound, and large fragments may be found loose in the peritoneal cavity.

In many cases of liver injury there are few arresting clinical signs, and such patients are often wisely left alone; it is significant that out of all the abdominal wounds of the war which recovered without operation the majority lay in the liver area. The danger of bleeding depends mainly upon whether large veins have been broached, for if these have escaped hæmorrhage ceases spontaneously. There is usually little oozing of bile, unless main ducts are severed. Early jaundice has no serious portent, but late icterus has a grave significance and indicates serious infection.

*Treatment* consists in suture of the rent in the liver after the instillation of sulphanilamide powder or paste.

**Penetrating Wounds of the Spleen.**—In considerably more than half the cases of splenic injury there will be no damage to the other abdominal viscera. The symptoms are those of internal hæmorrhage, the amount of which mainly depends upon whether the splenic vessels are injured. The pulp is said to cease bleeding within ten hours, but this time may be prolonged to forty-eight hours.

*Treatment.*—Cuthbert Wallace advised that the spleen should not be excised unless it is badly damaged or the vessels torn, but it must be remembered that interference with the blood-clot may restart bleeding and necessitate ablation.

Rupture of the urinary bladder, the kidney and the pancreas will be found described in the chapters dealing with these structures.



**Recent Advances.**—The present conflict has added to our knowledge of abdominal injury. In the “total warfare” of to-day when most of the injuries on the “home front” are the result of collapse of buildings or the impact of baulks of timber, masses of stone or fragments hurled with explosive force against old and young, woman and babe, warrior and cripple, operations for injuries of the belly due to enemy action have been remarkably infrequent. The tale of abdominal operations amongst casualties in ships or “in the field” is also a small one. It might have been thought that many of those killed in air bombardments of cities perished from abdominal lesions; such a surmise finds no confirmation on post-mortem investigation of fatal casualties. The remarkable fact emerges from the analysis of over 600 operations for abdominal injury in this war that 50 per cent. recover, despite the fact that the cases have not been selected for surgical interference after painstaking scrutiny. Operators have indeed vied with each other in their efforts to bring the resources of surgery within the reach of every wounded abdominal case who is not already obviously beyond all mortal aid. This great attainment, despite the fact that many abdominal injuries have been complicated by other lesions, even a fracture of the femur, a broken pelvis, or a wound of head or chest affords heartening proof that British surgery has not stood still since 1918.

“Total war” has changed the sex-incidence of the casualties: women and children are nowadays to be found in the lists of killed and wounded after air bombardment of cities, villages and hamlets. The youngest victim in a recent series of over 600 operations for intra-peritoneal injury was aged 3 years; the mortality of injuries to the belly in babyhood and early childhood is naturally very high, although a baby of 10 months recovered from a through-and-through wound of the abdominal wall which implicated the muscles, but did not penetrate the peritoneum.

**Nature of the Projectiles producing Injury.**—In a certain number of cases of abdominal injury produced by air bombardment the lesion is subparietal or non-penetrating in character. Most of the casualties in the present series had been wounded by fragments of *bomb-casing*, but many had been injured by flying spicules of *glass*, which may sometimes produce extensive visceral damage. *Mud, debris*, pieces of *brick* and *stone* may be found in the belly, and as in other campaigns, *fragments of bone* may be driven into the belly and damage abdominal organs. The greater frequency with which *incendiary bullets* have been employed has led to the more frequent need for intestinal resection because of the necrosis of the gut-wall produced. The rarity of successful operation for injury due to *bayonet thrust* is in conformity with the experience of previous wars.

**Blast.**—The effects of “*blast*” attracted little attention in the last Great War, although Sir Leonard Hill affirmed that the air in the pulmonary alveoli might even be compressed by blast acting upon the abdomen. Recent clinical and post-mortem evidence demonstrate that primary blast effects upon the abdomen do not often menace life. I have elsewhere recorded fatal cases due to this mechanism under the

care of Mr D. H. Patey, Mr Blacow Yates and others ; minor degrees of "blast abdomen" may be recovered from (O'Reilly). In experimental work, Zuckerman found that the abdomen was indubitably less vulnerable than the thorax ; of the hollow viscera, the colon was more liable to show changes than the small gut. Both in experimental work and in human pathology, perhaps the most characteristic blast effects are retroperitoneal hæmatomata, hæmorrhages between the leaves of the mesentery and subserous and submucous hæmorrhages in the bowel. The spleen may be torn in persons exposed to "blast" ; the liver has been bruised or torn, or its right lateral surface may be diversified by lines corresponding to the ribs. Perhaps the youngest living thing to perish in this war was a foetus, 6 cm. long, whose placenta was partly detached from the uterine wall of its mother, who had been diagnosed clinically as a "blast injury of the lung"—a diagnosis confirmed at autopsy. Multiple lesions of the abdomen were found, including the uterine damage referred to.

For an account of the physics of blast in water (immersion blast), reference must be made to the work of Surgeon-Commander Rex Williams, R.N. His work also showed the vulnerability of the lungs as compared with the abdomen, thereby confirming the work of Zuckerman. Protection of the abdomen in animals subjected to experimental immersion blast rendered the pulmonary lesions less severe than in those animals that were either unprotected or had received chest protection. It seems clear, therefore, that while pulmonary blast injuries may be due to the direct impact of the pressure wave on the thorax, the upthrust to the diaphragm through the abdomen may be equally, perhaps more, dangerous.

The clinical picture presented by those exposed to blast in water varies from a transient ileus to a rupture of the gut, complicated by various other lesions. The reader is referred to the writer's Bradshaw lecture for 1942.

**Multiple Wounds.**—Multiple wounds were common in the last war ; the severity of so many of the individual lesions in multiple wounding has been a marked feature of the casualties of to-day.

Each succeeding war demonstrates the increasing frequency of *the buttock* as a portal of entry for penetrating injuries of the abdomen ; the necessity for a careful inspection of the gluteal region must never be overlooked by those in charge of reception wards.

As in other conflicts, the wound of the belly produced by a large *fragment of metal* is almost always fatal ; a *wide, extensive loss of the abdominal parietes* from high explosive is rarely recovered from.

## SURGICAL AFFECTIONS OF THE ABDOMINAL WALL

**Inflammatory Diseases.**—Tuberculous abscess may be met with in connection with tuberculosis of the lower ribs or costal cartilages or even of the os innominatum. A primary chancre may be seen on the abdominal wall above the pubis, but until the advent of war was a very rare lesion ; gummata are likewise rare. Actinomycosis is encountered characteristically in the right iliac fossa and is marked

by a board-like infiltration of the abdominal wall and the presence of sinuses and fistulæ.

**Hæmatoma of the Abdominal Wall** is almost always due to the rupture of the rectus abdominis muscle or of one of the larger vessels intimately associated with its blood supply. The tear may be partial or complete, and most frequently involves the infra-umbilical portion (Fig. 248). The cause is rarely a direct injury, but more often due to some sudden muscular action, such as coughing. The onset, however, may be quite insidious, and in such cases the condition may be a complication of an acute infective or debilitating disease, thus adding to the anxieties of a case, *e.g.*, of typhoid fever or influenza. The fecund woman appears more prone to this catastrophe than is her barren sister; the condition is usually met with in the elderly.

The rectus muscle may also be ruptured during the spasms of tetanus or strychnine poisoning, but such are only pathological curiosities.

*Treatment* consists in suture of the torn muscle sheath.



FIG. 248

A rupture of left rectus abdominis muscle below umbilicus.

**New Growths of the Abdominal Wall.**—The skin of the abdominal wall

is naturally liable to the same lesions that may be found elsewhere. A squamous-celled carcinoma of the anterior abdominal wall is common in Kashmir (the well-known *kangri cancer*), and a similar condition is met with in tar workers and those who have been exposed to X-rays. A rodent ulcer is only rarely encountered, as is a fibrosarcoma (Fig. 249).

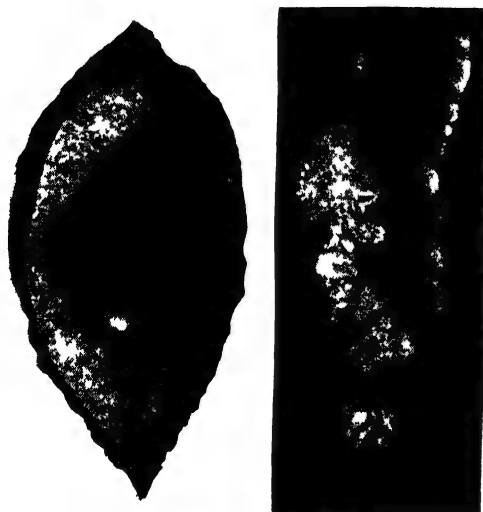


FIG. 249

A fibrosarcoma of the abdominal wall; both surface appearance and cross-section are shown.

**The Desmoid Tumour** (re-current fibroid of Paget, cellular fibroma) is an uncommon tumour of the sheath of the rectus abdominis muscle, affecting women more than men. The neoplasm is almost always single, occurs below the level of the umbilicus in 75 per cent. of cases and never takes origin exactly in the middle line. It arises in the

musculo-aponeurotic structures of the abdominal wall, and when small is completely embedded in the muscle substance; more often it implicates the fascial envelopes, especially the posterior layer of the rectus sheath. Growth is slow, at any rate in the early stages, tending to take place along the plane of least resistance, that is, in the direction of the muscle fibres. At first, therefore, the tumour is oval and flattened, and later becomes bossed or lobulated (Fig. 250).

These growths are fairly cellular fibromata and there is complete absence of a capsule. The peculiar histological features are the inclusion of striped muscle fibres and the sequence of regressive changes which these undergo, resulting in the formation of plasmodial masses resembling foreign body giant cells. Myxomatous changes may lead to a rapid increase in size, but these tumours never become malignant, yet are liable to recur.



FIG. 250

A desmoid tumour.

*Clinically* the growth is painless; the skin moves freely over it, and if the muscles of the belly-wall are relaxed the tumour can be manipulated so as to demonstrate its independence of the intra-abdominal contents. If the patient is requested to contract the muscles of his anterior abdominal wall, the tumour becomes completely immobilised (Bouchacourt's sign).

*Treatment.*—Early and the most ruthless extirpation is the only effective form of treatment.

**Osteogenesis in Laparotomy Scars** is by no means infrequent. Those especially liable to this change are

situated in the middle line involving the linea alba, and more commonly above than below the umbilicus. Patients complain of slight pain in the wound together with some stiffness or thickening of it.

**Dilated Veins of the Abdominal Wall** serve as a point of some diagnostic importance, and the reversal of the normal blood flow has even more significance. The venous blood normally courses from above downwards in the lower two-thirds of the abdominal wall; when clinical examination demonstrates that the flow of blood is from below upwards, obstruction to the inferior vena cava is almost certain; the blood endeavours to reach the heart along the dilated superficial venous collaterals to the superior vena cava.

In cases of compression of the inferior cava by ascites, ovarian cysts and other abdominal swellings, the veins of the abdominal wall become prominent only at a late stage; the coexistence of ascites and the early development of dilated superficial veins is an indication of malignant disease. In patients in whom there is no great abdominal distension and no suggestion of an ascending thrombosis from the

veins of the lower extremities, but in whom there is early varicosity of the veins of the abdominal wall, it may be presumed that the inferior vena cava is being obstructed by some malignant change in an adjacent structure.

## SURGICAL AFFECTIONS OF THE UMBILICUS

*Discoloration of the skin* around the umbilicus (Cullen's sign) occurs in certain diseases, e.g., ruptured ectopic gestation, acute pancreatitis, etc. *The caput medusæ* is a varicose condition of the veins around the umbilicus but is a most infrequent phenomenon; its presence constitutes an arresting advertisement of those habits which have determined



FIG. 251

A drawing illustrating the tracking of pus from the pelvis upwards in front of the peritoneum to point at the umbilicus.

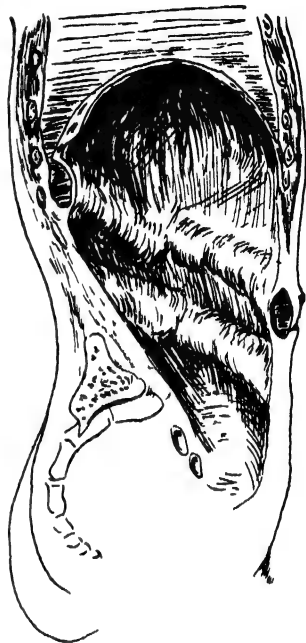


FIG. 252

An abscess from the posterior abdominal wall tracking forward to point at the umbilicus

cirrhosis of the liver. *Acanthosis nigricans* may be seen at the umbilicus as well as in the axilla, mouth, neck, around the external genitalia and under the breasts. It usually indicates an associated intra-abdominal malignancy, as a rule gastric or uterine carcinoma. *Retroperitoneal abscesses* may point at the umbilicus (Figs. 251 and 252).



FIG. 253

An endometrioma of the umbilicus.

**Congenital Malformations** include a congenital umbilical hernia, i.e., the exomphalos (p. 614) and certain malformations of the omphalo-mesenteric duct and the urachus, which may give rise to a sinus, a fistula or a solid tumour. The

last is sometimes seen as a bright red cherry-like structure filling up

the umbilical depression, and this has been given the name of an umbilical polyp or the mucous adenoma of the umbilicus.

**Secondary Carcinoma of the Umbilicus** may develop as an infrequent and late manifestation in connection with diverse primary forms of intra-abdominal new growth, being most frequently secondary to carcinoma of the stomach, gall-bladder, intestine, ovary and uterus. It has also been described in association with carcinoma of the breast.

**Endometrioma of the Umbilicus** is very rarely seen in women during the fourth and fifth decades of life; sometimes pain is experienced in the tumour at the menstrual period, and a brownish blood-stained discharge occurs from the navel on such occasions (Fig. 253).

### DISEASES OF THE OMENTUM

**Torsion of the Omentum** usually complicates in some manner a pre-existent hernia, but apart from this is a very rare condition. The patient, who is generally somewhat obese, is seized with generalised pain in the upper abdomen. This pain, at first not very severe, becomes more marked and is felt in the right side of the abdomen from the costal arch to the iliac fossa; vomiting may or may not occur. The diagnosis of a mild appendicitis



FIG. 254

Torsion of the great omentum.

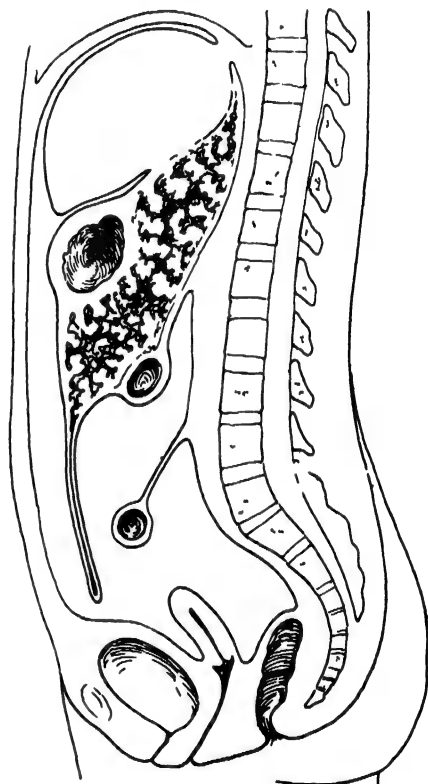


FIG. 255

A fibrosarcomatous growth filling up the lesser sac.

readily suggests itself, but as a rule surgical aid is not usually summoned until about the fourth day, by which time a doughy mass may sometimes be felt (Fig. 254).

*Treatment* consists in excision of the twisted portion of the omentum.

**Growths of the Omentum.**—Primary growths of the omentum are very rare. A sarcoma has been described originating in connection

with the inner lining of the lesser sac of the peritoneum, as shown in Fig. 255. A malignant hæmangioma is also described. Secondary growths of the omentum are, of course, extremely common, and may be associated with primary tumours in any part of the abdomen.

**Cysts of the Omentum** are very infrequent and may be classified as (a) lymphatic, (b) enterogenous, (c) urogenital, (d) dermoid (Fig. 256) and (e) hydatid.

### SURGICAL AFFECTIONS OF THE MESENTERY

**Mesenteric Vascular Occlusion** (Fig. 257) may be either arterial, venous or combined; the arterial is of two types, but embolism is far more frequently encountered than thrombosis, which is usually secondary to embolism. The superior mesenteric territory is more often involved than is the inferior mesenteric. The venous thrombosis is almost always of a secondary character, and may be due to portal obstruction or to peripheral splanchnic sepsis. Fatal pyæmia has on occasion been averted in the latter case by courageous ligation of the superior mesenteric vein, as reported by Julian Taylor.

*Clinical Picture.*—Whatever the nature of the vascular occlusion, the symptoms are identical and conform to one of two clinical types; the first is characterised by a rather more lengthy history, and there may have been several attacks of minor severity before the onset of the catastrophe, whereas the second variety consists of but one single acute attack.

A. The so-called chronic type exhibits mild symptoms of mesenteric arteriosclerosis for a variable period before the ultimate fulminating infarction. There is history of paroxysms of colic possibly accompanied by the passage of sanious stools, while in the intervals between there may be constipation. This prodromal period may last for months or even years before the final arterial occlusion occurs.

B. Acute fulminating mesenteric occlusion. The onset is most dramatic, the anguish intolerable, the shock profound and the

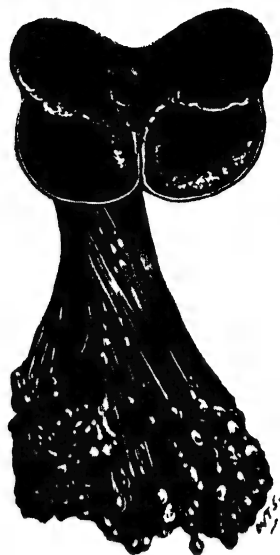


FIG. 256

A dermoid cyst of the great omentum.

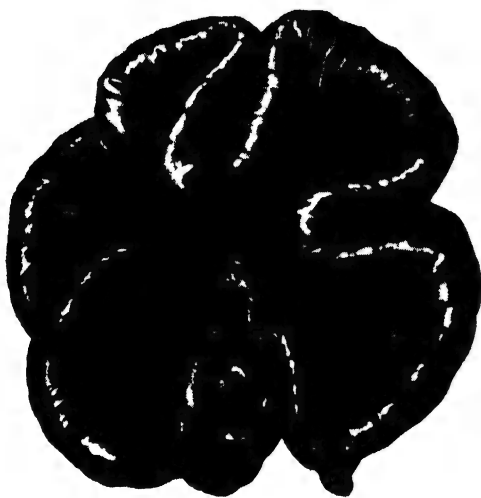


FIG. 257

Coils of small intestine illustrating the appearances seen in mesenteric vascular occlusion.



vomiting severe. The pain is experienced all over the abdomen, at first intermittent rather than continuous, but later, during its greatest intensity, the patient draws up the knees and raising the hands in supplication calls out in his agony. Hæmatemesis is stated to occur in 25 to 30 per cent. of cases, but the writer has not encountered this phenomenon.

Complete intestinal obstruction of the paralytic type is not present until gangrene of the bowel supervenes and several evacuations will take place. Bloody diarrhœa occurs in the early stages, the blood being either bright red or dark and tarry.

Before the onset of peritonitis there is no rigidity, but the abdomen is universally tender, and there is "rebound tenderness." Distension appears early, is usually general and on occasion may be extreme. The temperature is subnormal, the pulse rate is rapid from the start and steadily increases. The vomiting is profuse and in the later stages is typical of paralytic obstruction, the vomitus flowing out of the angle of the mouth in great quantity without the slightest apparent effort on the part of the patient.

*Treatment* consists in resection of the affected coils, provided too great a length is not infarcted.

**The Mesenteric Lymph Glands.** — *Tabes Mesenterica*, tuberculous infection of the mesenteric lymph glands, is more frequently met with in children than in adults and the



FIG. 258

A coil of small intestine showing a tuberculous ulcer on the antimesenteric border and caseous glands in the mesentery secondary to it

infection indubitably takes place from the imbibition of tuberculous milk, the organism being almost always of bovine type. Throughout Great Britain and especially in London there has been a marked reduction in the incidence of tuberculous mesenteric glands during the past twenty-five years.

The tubercle bacillus can apparently make its way through an intact intestinal mucosa, and the bowel may not exhibit any lesion; if ulceration of the small intestine is present, the appropriate glands will, of course, be implicated as well (Fig. 258).

In the early stage of hyperplasia, tuberculous mesenteric lymphadenitis may be associated with attacks of pain, pyrexia, constipation or diarrhœa; there may be loss of weight and of appetite, and appendicitis may be simulated. The pathological changes which characterise glandular tuberculosis may occur in the mesentery;



caseation and abscess formation may develop; peritoneal adhesions may impede the normal functioning of the bowel and may even engender an attack of acute intestinal obstruction. The mesentery plays a most frequent and important rôle in the pathology of obstruction, the *fons et origo* of which is often tuberculosis of its contained lymph glands.

Tuberculosis of the mesenteric glands rarely coexists with any other tuberculous manifestation, and Gauvain can recall no case of abdominal gland infection among thousands of patients suffering from bone and joint tuberculosis.

*Treatment.* — Surgery is reserved for the complications only, and dissection of the enlarged glands is to be deprecated. General constitutional treatment should be advised.

**Tumours of the Mesentery** include lymphosarcoma, fibrosarcoma and myxofibroma or cystic sarcoma (Fig. 259); all are rare.

**Cysts of the Mesentery** have been classified by Russell Howard and Perry as (1) chylous or serous cysts, (2) blood cysts, (3) hydatid

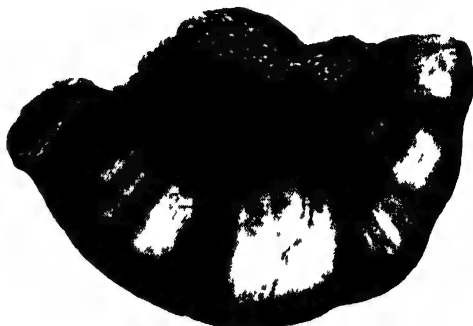


FIG. 259

A lymphosarcoma of the mesentery of the small intestine.



FIG. 260

A retroperitoneal cyst removed by the author.

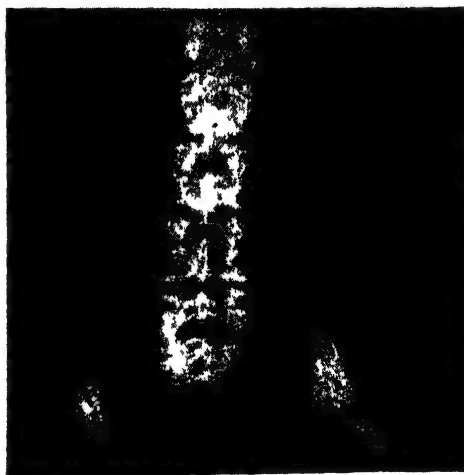


FIG. 261

X-ray appearance of the cyst prior to removal.

cysts and (4) teratomatous cysts. They also are rare, and their treatment is excision.

**Retroperitoneal Tumours** are of more interest to the pathologist than to the surgeon; the majority are malignant and almost every type of sarcoma has been described.

**Retroperitoneal Cysts** are classified by Handfield-Jones as follows: (1) cysts of urogenital origin; (2) cysts of mesocolic origin; (3) those

arising in cell inclusions; (4) lymphatic cysts; (5) blood cysts; (6) parasitic cysts; and (7) those of developmental origin in fully formed organs (Figs. 260 and 261).

Cysts of urogenital origin are thin walled, unilocular and have no visible blood vessels in their walls. The exact diagnosis is made only at operation or after removal and microscopic examination.

GORDON GORDON-TAYLOR.

## THE PERITONEUM

*Surgical Anatomy.*—The student is referred to textbooks of anatomy for a full description of the intricate details of the general arrangement

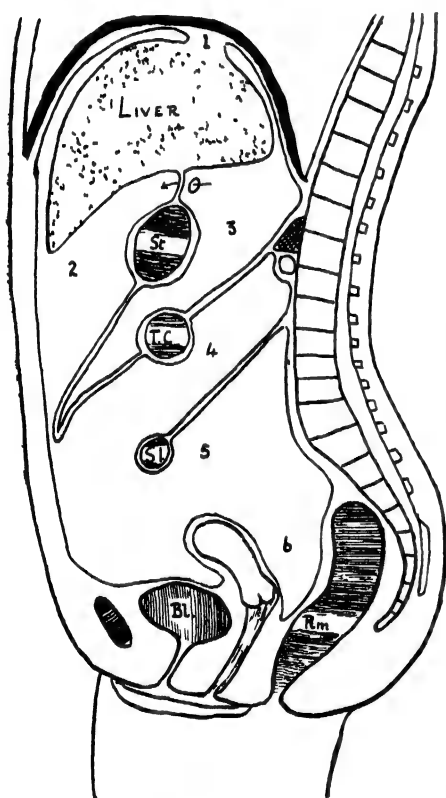


FIG. 262

Diagram illustrating some of the peritoneal compartments.

1, is the area bare of the liver; 2, the anterior subhepatic compartment, 3, the lesser sac, the arrow indicating the foramen of Winslow; 4, the space between the transverse mesocolon and mesentery; 5, the space below the mesentery continuous with 6, pouch of Douglas.

and relations of the peritoneum, and only those essentials which have a direct bearing on surgical problems will be discussed here. The peritoneum is a serous membrane which lines the abdominal cavity, providing smooth surfaces to ensure free and unrestrained movement between the viscera; further it is endowed with the most marvellous capacity for controlling and overcoming infection, and upon its health and integrity depends the well-being of its owner.

It covers some parts of the intestinal tract completely, thereby forming a pedicle which allows a wide range of movement; the **mesentery** supports the small intestine, the **mesocolon** (transverse or sigmoid) the free parts of the large intestine, while the stomach is slung in the folds of the **gastro-hepatic omentum**. Certain solid viscera, viz., the liver and spleen, are almost entirely covered with peritoneum, others only partly so. The greater part of the duodenum, the ascending and descending colon, are clothed only on their anterior surfaces as are the kidneys and the pancreas. These organs are described as being "retroperitoneal." The peritoneal cavity is something of a misnomer, since the cavity is but a potential one under normal conditions.

Two main divisions of this cavity are described, the *greater and lesser sacs*. The latter lies behind the liver, gastrohepatic omentum and

stomach, and communicates with the former by a small opening in the suprapyloric region named the "foramen of Winslow." The greater sac

can be subdivided (arbitrarily, not actually) into two main compartments, one lying above the transverse mesocolon and colon and the other below. Fig. 262 makes this clear as also that subdivisions of the subcolic area are permissible for purely clinical purposes, between the mesocolon and the mesentery and again below the mesentery down to and including the pelvis. When an individual is lying supine and horizontal, the promontory of the sacrum and the 5th lumbar vertebra project forward so far that a "watershed" is formed, and in this position fluid will flow either upwards toward the liver or downward to the pelvis, according to its relation to this watershed. Fig. 263 shows the influence of position on this flow of fluid and illustrates the advantages of "Fowler's position," in which all fluids from any part of the peritoneum tend to flow towards the

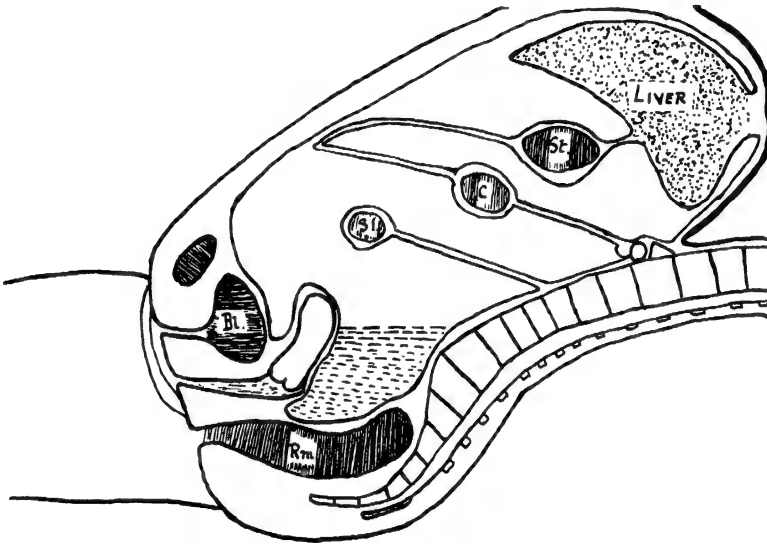


FIG. 263

Diagram illustrating the value of Fowler's position showing a collection of fluid in the pouch of Douglas.

pelvis, the least dangerous position for the collection of inflammatory exudates.

Above the shelf formed by the transverse mesocolon, colon and great omentum, the peritoneal relations are of considerable complexity and several subhepatic and subphrenic spaces are described. These have special reference to subphrenic abscesses and are set out in full in the section relating to them.

**The Great Omentum** is a special fold of peritoneum derived from the two layers which clothe the stomach. These pass downwards from the greater curvature and then turn back to unite with the layers in which is fixed the transverse colon. In this way a four-layered apron of peritoneum hangs down in front of the small intestine and colon. In its lower part all the layers are fused together and a thin, highly vascular membrane is formed. The great omentum is endowed with wonderful powers of defence and has been—most aptly—described as the "scavenger of the abdomen." It seems to be irresistibly drawn to any trouble in the peritoneal cavity and, having reached the scene of action, wraps itself round an inflamed area and envelops it with protective adhesions.

### PERITONITIS

Inflammation of the peritoneum or peritonitis may be acute or chronic, local or generalised. It is almost invariably due to bacterial invasion, which reaches the peritoneum either from the gastro-intestinal canal, the urinary system or the female genital organs, and only rarely by penetrating wounds or via the blood or lymph streams.

Acute localised peritonitis is the inevitable concomitant of most infective intra-abdominal diseases, but the prompt removal of its cause leads to rapid resolution of the infection. The peritoneum possesses the most marvellous powers of defence and recuperation provided it is relieved of continued irritation and reinfection. Acute diffuse or generalised peritonitis has a high mortality even when recognised and treated in its early stages, and if neglected must certainly be fatal. The importance, therefore, of early diagnosis and prompt treatment of acute abdominal emergencies cannot be overestimated. The marked decrease in the incidence of peritonitis is an indication of a greatly improved standard of diagnosis.

Chronic peritonitis is usually tuberculous, though it may result from organisms whose manifestations are more commonly acute, viz., gonococcus, pneumococcus, etc. Chronic local or plastic peritonitis produces adhesions and may be regarded as a protective phenomenon.

### ACUTE PERITONITIS

*Etiology.*—Acute inflammation of the peritoneum is almost invariably due to invasion of bacteria, the causes of which can be conveniently grouped as follows :—

1. Acute infections, *e.g.*, appendicitis, diverticulitis, cholecystitis, salpingitis.
2. Acute perforations, *e.g.* of peptic, stercoral and typhoid ulcers.
3. Injuries of any hollow viscus—penetrating wounds, rupture without external wound (*e.g.*, jejunum).
4. Injury of any solid viscus, *e.g.*, liver, spleen, ectopic gestation, in which the intraperitoneal hæmatoma may become secondarily infected.
5. Ascending infections of the female genital tract, *e.g.*, gonococcal, pneumococcal and streptococcal infections (particularly puerperal sepsis).
6. Torsion, strangulation and gangrene of solid and hollow viscera, *e.g.*, strangulated hernia, torsion of ovarian cyst, etc.
7. Blood-stream infections, *e.g.*, pneumococcal peritonitis during pneumonia.
8. Irritation by sterile fluids such as bile, pancreatic juice and urine.

*Bacteriology.*—The bacteria, which usually inhabit the intestinal canal, must play a predominant part in peritonitis. Under normal conditions they are almost non-pathogenic, but in the presence of inflammation, perforation or strangulation of part of the intestinal tract they take on a greatly increased virulence. This infection is almost invariably a mixed one and the following organisms may be found: (1) aerobes, *B. coli*, *B. pyocyaneus*, *B. proteus* and the pyogenic cocci, and (2) anaerobes such as *B. welchii*.

*Pathology.*—The pathological changes vary considerably according to the severity of the infection and reaction of the patient. The onset may be abrupt in origin as in the perforations of peptic or stercoral ulcers, or gradual in the case of inflammatory lesions such as appendicitis; in this and similar conditions a localised peritonitis occurs gradually on the surface of the inflamed viscus.

The peritoneum becomes hyperæmic and inflamed, losing its shiny lustrous appearance and gaining a rough granular surface. A fluid exudate is poured out; at first clear and serous, it later becomes seropurulent and then finally purulent. It contains fibrin, which is often deposited in large flakes and which assists in localising the infected area by causing coils of intestine, the great omentum and possibly the abdominal wall to become stuck together, in this way forming a protective barrier beyond which the peritoneal cavity is normal. This defence mechanism is constantly demonstrated in acute appendicitis; the inflamed and possibly gangrenous appendix lies in an abscess cavity, the walls of which are formed by the terminal ileum, cæcum, great omentum and possibly the deep surface of Poupart's ligament.

The subsequent course of such an acute localised peritonitis depends upon the efficacy of prompt treatment, the virulence of the infecting organism and the powers of resistance of the patient. Thus, if the inflamed viscus (*e.g.*, the appendix) is removed within the first thirty-six hours, the inflammatory reaction will subside, the fibrin will be absorbed and the peritoneum returns to normal with few, if any, adhesions; resolution may be said to have occurred. Under less favourable conditions, suppuration may take place within the area shut off by fibrinous adhesions and a localised intraperitoneal abscess forms. Finally, if a very virulent infection should attack a seriously ill patient, in whom the defence mechanism has broken down, pus spreads rapidly among the coils of intestine and a generalised spreading peritonitis is established.

*Effect upon the Intestine.*—Normal peristaltic action in the intestinal tract must necessarily tend to delay or even prevent the formation of soft adhesions which localise the lesion; further it must encourage the spread of the infection by massaging the infected exudate further afield. Nature combats these handicaps by placing the affected segments of bowel at rest by suppressing peristaltic action. Helpful as this is, yet it has certain grave disadvantages in that the bowel distends and its contents become stagnant; in this way the virulence of the intestinal flora is greatly increased and their passage through the gut-wall into the peritoneal exudate encouraged. In the neglected or more severe cases of general peritonitis, this cessation of bowel movement becomes an established paralytic ileus (p. 642) and the patient's life is gravely endangered.

*Toxic Absorption.*—The immense mortality rate of untreated acute peritonitis cannot be solely due to the local conditions. An attempt has been made to explain it by the absorption of *B. welchii* toxin from the intestine and the peritoneal exudate (Maybury and Williams), but although this bacillus is often present in large numbers this theory

does not gain acceptance to-day, and the administration of the anti-toxin of *B. welchii* is no longer considered helpful. It is probable that the toxæmia is in the nature of an absorption of proteoses and amino-acids from the paralysed coils of intestine.

### ACUTE LOCALISED PERITONITIS

Acute peritonitis starts as a localised lesion in all abdominal emergencies, except those in which widespread flooding of the cavity occurs, as for example in perforations of peptic and stercoral ulcers or in multiple penetrating wounds of the intestine. Apart from these conditions generalised peritonitis follows mistakes in diagnosis, delayed, ill-judged or otherwise ineffective treatment or a diminished resistance on the part of the patient. It is useful, therefore, to consider first the clinical picture of localised peritonitis before passing to a description of the more dangerous spreading or generalised disease.

*Symptoms.*—These are indistinguishable from those of the causative condition, and it is misleading to describe any specific symptoms for local peritonitis. If acute appendicitis is taken as an example, the characteristic origin and development of the pain, the initial vomiting or nausea and the constipation are present without the peritoneal coat having been involved; but whereas the symptoms alter little when localised peritonitis sets in, the signs change in a definite manner.

*Signs.*—The rise in temperature and pulse rate together with the abdominal tenderness, which result from the causative lesion, tend to increase with the onset of peritonitis, and a new and very important sign makes its appearance, viz., *rigidity*. Although voluntary guarding of the abdominal muscles may be present in the early stages of many intraperitoneal inflammations, true rigidity never occurs until the peritoneal coat is involved. At first, localised areas of muscle become rigid and immobile, while in spreading infections the whole abdominal wall may become board-like in its rigidity. The differentiation between true and false rigidity is one of the most important clinical lessons the student must master.

Two tenderness tests are of value in doubtful cases, both of which Zachary Cope has emphasised: first, the production of pain in the affected region by deep pressure over an unaffected zone, this being seen in acute appendicitis when pressure over the left iliac fossa produces pain in the right side; secondly, tenderness on rebound, which is the production of pain not so much by pressure but by the release of that pressure.

Auscultation may be useful, revealing normal sounds over the unaffected parts and diminished or absent peristaltic sounds in the zone of peritonitis. Palpation probably reveals nothing, since the rigidity prevents deep examination, but at any time a definite tumour mass may be felt. In patients suffering from acute appendicitis a circumscribed swelling is usually to be palpated after sixty to seventy-two hours.

*Diagnosis* is that of the causative disease.

*Prognosis.*—Prompt diagnosis and treatment should lead to resolution and recovery in all patients. Delay will often mean the formation of a local intraperitoneal abscess and, if the patient is unable to localise the infection, an acute general peritonitis will follow.

*Treatment.*—In the early stages the cause of the peritonitis must be removed promptly and efficiently, and in cases of acute localised peritonitis we do not allow any exception from this rule. Any delay may condemn the patient—quite unjustifiably—to the danger of general peritonitis. Gonococcal infections in the pelvis are not included, for we regard them as a diffuse pelvic peritonitis (see below).

#### ACUTE DIFFUSE OR GENERALISED PERITONITIS

The origin, etiology and pathology have been discussed above, but it is instructive to consider certain variations in the general picture. The onset is variable, being dramatically sudden in perforations of the hollow viscera and in acute pancreatitis, and gradually progressive in appendicitis, cholecystitis and infections of the pelvic organs. The course likewise differs, widespread flooding of the cavity and very virulent infections leading quickly to a fatal issue. The more gradual lesions may persist for several weeks before resolution or death occurs.

*Symptoms.*—*A. Early.*—This stage will be introduced by the symptoms of the causative disease, which merge into those of peritonitis. Pain may be of sudden or gradual onset and at first may be localised, but slowly spreads till the whole abdomen is affected. It is of a dull constant aching character which increases in severity as the infection advances. Vomiting occurs at the beginning of the attack and becomes a prominent feature of the later stages.

*B. Late.*—Pain increases in severity, vomiting becomes profuse and constipation is absolute. Even when death approaches, the mind remains clear.

*Signs.*—*A. Early.*—The tongue is furred, breathing shallow and temperature and pulse rates raised. The abdomen is flat or retracted and does not move with respiration. It is tender and very rigid. Auscultation reveals marked diminution or absence of peristaltic sounds.

*B. Late.*—The patient lies half propped up in bed with the hips and knees flexed to relax the tension on the abdominal muscles. The face has the earthy pallor of toxæmia, it seems to sink inwards to a remarkable degree, there is a cold clammy sweat on the forehead and nose and the eyes are withdrawn into the orbits. The expression is one of great anxiety, but the mind remains clear and alert. Such is the “*facies Hippocritica*,” so typical of severe toxæmia. The pulse rate has risen to 150 or more, but the temperature has fallen to 99° F. or even below normal. The retracted abdomen has been replaced by the distension of paralytic ileus, but the muscles are still rigid and generalised tenderness persists. There is absolute constipation and fæulent vomit flows effortlessly from the mouth. There may be retention of urine, which is highly coloured and contains albumen. Auscultation meets with silence and percussion may reveal shifting dullness in the flanks.



The *prognosis* is always grave and with every hour that passes the prospect becomes more hopeless. Early diagnosis of the cause and its prompt treatment will save many patients, but any delay will meet with disaster.

*Differential Diagnosis.*—The picture of an established case of generalised peritonitis cannot be mistaken, but in the early—and important—stage much difficulty may be experienced. The morbid processes likely to cause confusion are as follows :—

1. Thoracic. Pleurisy and pneumonia often give symptoms and signs referred to the upper abdomen, and coronary thrombosis may suggest a perforated peptic ulcer or an acute cholecystitis. A careful attention to the history and mode of onset, together with a *routine* examination of the chest, should exclude these diseases.

2. Abdominal colic, be it intestinal, biliary or renal. The former can cause great confusion, but the others are recognised by the nature, extent and distribution of their pain and tenderness.

3. Intestinal obstruction may lead to peritonitis, but in the early stages is characterised by pain, complete absence of tenderness and rigidity and by greatly exaggerated sounds on auscultation. The combination of the history with the negative findings should always prevent any mistake.

4. Intraperitoneal hæmorrhage, especially when due to trauma, may be most confusing at first, because the violence may have bruised the abdominal muscles sufficiently to cause rigidity and tenderness. The general picture of internal bleeding, however, soon becomes unmistakable.

5. Renal disease. Pyelitis may prove misleading, but the rapidity of onset with so high a temperature ( $104^{\circ}$  to  $105^{\circ}$  F.) and rigors should prevent error. Uræmia sometimes manifests itself as a slowly appearing ileus, but the absence of tenderness and rigidity should be conclusive.

6. Spinal cord and column. Tabes dorsalis can be misleading when abdominal crises are present, and an examination of the pupils and the nervous system alone can prevent mistakes. Spinal caries with a psoas abscess may also give symptoms entirely limited to the abdomen.

7. Torsion of an ovarian cyst, subserous fibroid or aberrant spleen is among the more rare conditions which may need to be distinguished.

In effect the differential diagnosis is that of all acute abdominal disease, and it cannot be too constantly or forcibly impressed upon students that, in every case of an abdominal emergency, the chest, urine and nervous system must invariably be examined.

*Treatment.*—This is directed to the removal or suppression of the cause, peritoneal toilet and drainage.

**Removal of the Cause.**—Attention is directed to the particular viscus involved and this receives appropriate treatment. The lesion cannot always be removed, but it must be closed or sealed off from further communication with the peritoneal cavity ; for example, a gangrenous appendix will be removed, whereas a perforated ulcer will be sealed by suture. Such treatment may be regarded as prophylactic, because if done soon enough it will prevent the occurrence of general peritonitis.



**Peritoneal Toilet.**—The inflammatory exudate should be removed as completely as possible consistent with gentleness of handling and absence of exposure of unaffected areas. Very gentle swabbing and, better still, aspiration will extract all the fluid if care and patience are exercised. The area is then liberally dusted with sulphanilamide.

**Drainage.**—This is a very vexed question and no explanation will meet with general acceptance, so diverse are the views held by different surgeons. It is better to follow certain general principles. In the first place, it is completely impossible to drain the general peritoneal cavity, for within thirty-six hours the tube is surrounded with coils of intestine and omentum and sealed off by adhesions; in the second place, the peritoneum is unique among human tissues in its wonderful powers of defence. If the original lesion has been removed or closed and the irritant exudate extracted, the peritoneum is quite able to look after itself without any external assistance. It is only when the cause has not been removed or if certain infective conditions are left behind that drainage becomes necessary. These conditions are (a) local abscess; (b) retention of cause, *e.g.*, cases of gangrenous appendicitis or cholecystitis, in which there has been no attempt to disturb the localising adhesions and a tube has been inserted without further exploration; (c) inefficient suture of intestine leading to the fear of a faecal fistula, *e.g.*, when the caecal wall is so friable that it will not retain stitches after appendicectomy; (d) certain special lesions such as acute pancreatitis, which demand drainage for five to six weeks; and (e) all operations in which there is unavoidable oozing of blood.

When a drainage tube has been used, it should be kept *in situ* for five days and then removed. There is no point in shortening it daily, and if it should be extruded before the fourth day no effort must be made to reinsert it or a smaller one.

*After-treatment* is directed to the prevention or cure of paralytic ileus (p. 642) and to the elimination of toxins. The patient is placed in Fowler's position and a continuous intravenous drip saline is started immediately on return from the theatre. Provided the surgeon is satisfied that the cause has been removed and that all pockets of pus have been drained, the problem is simply that of the ileus.

*Treatment of the Late Cases* is practically hopeless, but the cause should be sought for and removed by laparotomy unless the patient is moribund. The peritoneum is carefully cleansed, dusted with sulphanilamide and closed with drainage.

#### SPECIALISED TYPES OF ACUTE PERITONITIS

**Pneumococcal Peritonitis** is practically confined to young female children under the age of 12 years. It occurs in two forms, either as a secondary blood-stream infection during lobar pneumonia or middle-ear disease, or as an ascending infection from the vulva via the vagina, uterus and Fallopian tubes. This latter type is an example of the so-called "primary peritonitis of children." It is usually acute and generalised, but it occasionally produces a subacute local lesion.

*Symptoms.*—If the abdominal condition is secondary to pneumonia, the child is already gravely ill and the pain and vomiting appear as yet another dread complication. In the primary type the onset is gradual in a previously well and happy child. Vague abdominal pain ushers in the attack with nausea, loss of appetite and possibly diarrhoea. The patient becomes ill, very fretful and peevish, and within a few days a typical general peritonitis has developed without any suggestion as to its cause.

The *signs* differ in no way from those given above, but the progress is not so rapid. The abdomen is tender and rigid and the child is obviously gravely ill, thin and miserable. In the localised form the signs are concentrated over a definite tender swelling in the lower abdomen. A vaginal discharge is present in nearly every case and the pneumococcus can be isolated from it.

*Treatment.*—If the pneumococcal origin has been proved, it is wise to employ expectant treatment, because although the prognosis is always grave the results are better than after laparotomy. Full doses of sulphapyridine, together with blood transfusions, hold out the best hope of cure. If the nature of the peritonitis is in doubt an exploration must be done, and if the thick, greenish and odourless pus is recognised as pneumococcal the wound is closed without drainage. In every case a swab will be taken for investigation.

In the localised abscess, also, the pus should be evacuated and the wound closed without drainage, general and chemotherapy being relied upon to complete the cure.

**Gonococcal Peritonitis** is also confined to the female sex, but in this case it is usually in women during their period of sexual activity. It does rarely occur in young children as a complication of infective vulvovaginitis.

In women it is invariably the result of coitus with an infected male. The infection tends to remain localised to the pelvic peritoneum and is rarely so acute as in the preceding varieties. It spreads to the peritoneum from the Fallopian tubes and there has generally been a history of vaginal discharge and vulval soreness.

*Symptoms.*—Some days after coitus the woman complains of nausea and vomiting and then of lower abdominal pain immediately above Poupart's ligament on each side. There is constipation, a vaginal discharge with soreness and discomfort of the vulva, and frequent painful micturition. On examination there is no abdominal rigidity, but tenderness is noticeable above both inguinal ligaments and especially in the vaginal fornices. The temperature is moderately raised ( $101^{\circ}$  to  $102^{\circ}$  F.) and the patient often imagines she has caught a chill.

In the less common cases there may be general tenderness and rigidity indicating that the infection has become a diffuse one.

*Diagnosis.*—The presence of a discharge, the localisation of pain to the pelvis and the general condition should suffice to raise the suspicion of gonococcal peritonitis. It is not always possible, however, to differentiate the varied causes of pelvic peritonitis and a pelvic appendicitis may lead to great difficulty. If real doubt exists it is

better to perform an exploratory laparotomy than to overlook a peritonitis of intestinal origin.

*Treatment.*—If the diagnosis has been made with confidence, operation is definitely contraindicated. Treatment of the genital infection by sulphapyridine and later with vaginal pessaries and douches and careful nursing will usually lead to a resolution of the infection.

**CHRONIC PELVIC PERITONITIS** of gonococcal origin follows either imperfect resolution of an acute attack or arises spontaneously. There can be few more tragic diseases than this, often transmitted to an innocent girl at marriage. It leads to dense adhesions in the pelvis, chronic pelvic pain with severe dysmenorrhœa and the danger of intestinal obstruction. Within a short time a happy healthy girl has been converted into a fretful and disillusioned chronic invalid. The treatment of these patients presents a grave problem. Every effort must be made to clear up the local conditions, but in many cases nothing short of the removal of both tubes and uterus will lead to a restoration to some measure of health and happiness.

**Streptococcal Peritonitis** merits special mention owing to its grave prognosis and its association with puerperal infections. It may be seen also as a manifestation of streptococcal septicæmia and it may dominate the picture in an uncomplicated peritonitis of intestinal origin, *e.g.*, acute appendicitis. In these cases, especially in the early stages, the effusion is of a dirty, blood-stained, serous nature; such a finding during a laparotomy should point to the need for prophylactic measures against paralytic ileus, so common in this type of infection.

There is nothing specific in the way of treatment beyond that already laid down, but when secondary to puerperal sepsis, the genital lesion must receive energetic attention. Sulphapyridine should be given freely and streptococcal antitoxic serum may be useful.

### LOCAL INTRAPERITONEAL ABSCESS

Collections of pus may occur in any part of the peritoneal cavity and can be divided into those above the transverse colon and omentum (grouped together as subphrenic abscesses) and those below them, these usually localising either in the pelvis or in one or other iliac fossa.

#### **Subphrenic Abscess.**

*Anatomy.*—Barnard's classification with slight modifications still holds the field and depends on the anatomical arrangement of the potential spaces beneath the diaphragm and liver. These are right and left anterior, right and left posterior intraperitoneal spaces and the extraperitoneal bare area on the superior surface of the liver.

The *right anterior space* lies to the right of the falciform ligament and has both a subdiaphragmatic and a subhepatic division. It contains the gall-bladder, pylorus and the first part of the duodenum.

The *right posterior space* communicates freely with the above. It lies below the diaphragm behind the right lobe of the liver, round the inferior margin of which it spreads forwards and joins the anterior compartment.

*Symptoms.*—If the abdominal condition is secondary to pneumonia, the child is already gravely ill and the pain and vomiting appear as yet another dread complication. In the primary type the onset is gradual in a previously well and happy child. Vague abdominal pain ushers in the attack with nausea, loss of appetite and possibly diarrhoea. The patient becomes ill, very fretful and peevish, and within a few days a typical general peritonitis has developed without any suggestion as to its cause.

The *signs* differ in no way from those given above, but the progress is not so rapid. The abdomen is tender and rigid and the child is obviously gravely ill, thin and miserable. In the localised form the signs are concentrated over a definite tender swelling in the lower abdomen. A vaginal discharge is present in nearly every case and the pneumococcus can be isolated from it.

*Treatment.*—If the pneumococcal origin has been proved, it is wise to employ expectant treatment, because although the prognosis is always grave the results are better than after laparotomy. Full doses of sulphapyridine, together with blood transfusions, hold out the best hope of cure. If the nature of the peritonitis is in doubt an exploration must be done, and if the thick, greenish and odourless pus is recognised as pneumococcal the wound is closed without drainage. In every case a swab will be taken for investigation.

In the localised abscess, also, the pus should be evacuated and the wound closed without drainage, general and chemotherapy being relied upon to complete the cure.

**Gonococcal Peritonitis** is also confined to the female sex, but in this case it is usually in women during their period of sexual activity. It does rarely occur in young children as a complication of infective vulvovaginitis.

In women it is invariably the result of coitus with an infected male. The infection tends to remain localised to the pelvic peritoneum and is rarely so acute as in the preceding varieties. It spreads to the peritoneum from the Fallopian tubes and there has generally been a history of vaginal discharge and vulval soreness.

*Symptoms.*—Some days after coitus the woman complains of nausea and vomiting and then of lower abdominal pain immediately above Poupart's ligament on each side. There is constipation, a vaginal discharge with soreness and discomfort of the vulva, and frequent painful micturition. On examination there is no abdominal rigidity, but tenderness is noticeable above both inguinal ligaments and especially in the vaginal fornices. The temperature is moderately raised ( $101^{\circ}$  to  $102^{\circ}$  F.) and the patient often imagines she has caught a chill.

In the less common cases there may be general tenderness and rigidity indicating that the infection has become a diffuse one.

*Diagnosis.*—The presence of a discharge, the localisation of pain to the pelvis and the general condition should suffice to raise the suspicion of gonococcal peritonitis. It is not always possible, however, to differentiate the varied causes of pelvic peritonitis and a pelvic appendicitis may lead to great difficulty. If real doubt exists it is

better to perform an exploratory laparotomy than to overlook a peritonitis of intestinal origin.

*Treatment.*—If the diagnosis has been made with confidence, operation is definitely contraindicated. Treatment of the genital infection by sulphapyridine and later with vaginal pessaries and douches and careful nursing will usually lead to a resolution of the infection.

**CHRONIC PELVIC PERITONITIS** of gonococcal origin follows either imperfect resolution of an acute attack or arises spontaneously. There can be few more tragic diseases than this, often transmitted to an innocent girl at marriage. It leads to dense adhesions in the pelvis, chronic pelvic pain with severe dysmenorrhœa and the danger of intestinal obstruction. Within a short time a happy healthy girl has been converted into a fretful and disillusioned chronic invalid. The treatment of these patients presents a grave problem. Every effort must be made to clear up the local conditions, but in many cases nothing short of the removal of both tubes and uterus will lead to a restoration to some measure of health and happiness.

**Streptococcal Peritonitis** merits special mention owing to its grave prognosis and its association with puerperal infections. It may be seen also as a manifestation of streptococcal septicæmia and it may dominate the picture in an uncomplicated peritonitis of intestinal origin, *e.g.*, acute appendicitis. In these cases, especially in the early stages, the effusion is of a dirty, blood-stained, serous nature; such a finding during a laparotomy should point to the need for prophylactic measures against paralytic ileus, so common in this type of infection.

There is nothing specific in the way of treatment beyond that already laid down, but when secondary to puerperal sepsis, the genital lesion must receive energetic attention. Sulphapyridine should be given freely and streptococcal antitoxic serum may be useful.

### LOCAL INTRAPERITONEAL ABSCESS

Collections of pus may occur in any part of the peritoneal cavity and can be divided into those above the transverse colon and omentum (grouped together as subphrenic abscesses) and those below them, these usually localising either in the pelvis or in one or other iliac fossa.

#### Subphrenic Abscess.

*Anatomy.*—Barnard's classification with slight modifications still holds the field and depends on the anatomical arrangement of the potential spaces beneath the diaphragm and liver. These are right and left anterior, right and left posterior intraperitoneal spaces and the extraperitoneal bare area on the superior surface of the liver.

The *right anterior space* lies to the right of the falciform ligament and has both a subdiaphragmatic and a subhepatic division. It contains the gall-bladder, pylorus and the first part of the duodenum.

The *right posterior space* communicates freely with the above. It lies below the diaphragm behind the right lobe of the liver, round the inferior margin of which it spreads forwards and joins the anterior compartment.

The *left anterior space* lies to the left of the falciform ligament and in front of the gastrohepatic omentum, the anterior surface of the stomach and the great omentum.

The *left posterior space* is the lesser sac of the peritoneum, which therefore cannot communicate with any of the other spaces except through the foramen of Winslow.

*Etiology.*—Any of the many causes of peritonitis may eventually lead to the formation of a subphrenic abscess. Commonly it follows perforated peptic ulcers, appendicitis, cholecystitis, ruptures of the liver and spleen and occasionally the spontaneous extension of an empyema through the diaphragm.

*Symptoms.*—**Group I.—Post-operative subphrenic abscess** may follow any acute inflammatory lesion within the peritoneum. Either from ineffectual localisation by the patient, lack of removal of the cause or imperfect surgical technique, an infective or irritant focus is left undrained beneath the diaphragm. The clinical story in such a patient relates to the original illness, the operation and a period of from four to seven days during which a steady improvement takes place, both the local conditions and the pulse and temperature settling down. Then this progress is stayed and an insidious deterioration sets in. Pulse and temperature rise, there is upper abdominal pain and the patient shows signs of toxæmia.

**Group II.—Spontaneous subphrenic abscess** results from perforations of peptic ulcers, which leak slowly and become sealed with omental plugs, or such acute conditions as appendicitis, in which the infection is carried to the subphrenic region by the lymphatics or the retro-peritoneal tissues. In these cases there will be an interval of ten or more days before the onset of the abdominal pain and signs of a mild toxæmia.

*Signs.*—If the pus is below the liver in front, a swelling is apparent below the costal margin and in the epigastrium of the affected side, and the diagnosis presents no difficulty; this is far from true when the pus lies above the liver, deeply buried beneath the diaphragm. Nevertheless a careful analysis of the clinical signs should lead to a correct diagnosis. The liver is displaced downwards and the diaphragm upwards, therefore producing a considerable increase in the dullness on that side; a small circular area of gas resonance may be found in this dullness. Above the diaphragm is a small sympathetic pleural effusion and above this a zone of compressed lung, above which again will be a typical strip of increased vocal resonance and ægophony. In addition, there may be an appreciable bulging of the lower costal area on the affected side and both chest and abdomen move poorly with respiration. A leucocytosis of over 20,000 will point to a collection of pus.

*Diagnosis* should rest between pus above or below the diaphragm. This disease is so much less frequently seen in this era of improved diagnosis that its possible presence may be overlooked, but the very obscurity of the clinical picture should suffice to raise the suspicion of subphrenic pus. Percussion and auscultation of the successive zones enumerated above will not always clinch the diagnosis, but



X-ray screening will invariably do so. The elevation of the dome of the diaphragm, which is motionless, the absence of any considerable quantity of fluid in the pleura and the upward but not lateral displacement of the heart all point to a subdiaphragmatic lesion.

*Prognosis* is always grave as this relatively uncommon disease points to a breakdown either of the patient's resistance or of surgical technique.

*Treatment.*—(1) **Prophylactic.** Early diagnosis and prompt treatment of abdominal disease has already led to a great decrease in the incidence of this complication and will do so still further in the future. (2) **Active.** The approach for drainage depends on the situation of the pus. The anterior subhepatic collections form visible swellings below the costal margin and thus drainage is a very simple matter. The incision is made through the oblique muscles just outside the rectus sheath an inch below and parallel to the costal margin.

The subdiaphragmatic abscesses must be drained from behind without traversing the pleural cavity. The 11th or 12th rib is resected through an incision outwards from the edge of the erector spinæ. The diaphragm is incised low down below the limit of the pleura. The finger enters the perinephric fat and works its way upwards, following the abdominal surface of the diaphragm. The abscess is thus opened without traversing either the pleura or the unaffected peritoneum.

The transpleural route should be employed only when other routes are impossible, as its mortality rate is unduly high.

**Pelvic Abscess.**—These are far more common in women than in men, owing to the liability of the female genital organs to infection. Thus in women the causes are: salpingo-oöphoritis, puerperal and abortional sepsis (including the penetrating wounds of the vaginal fornix in criminal cases), torsions and strangulations of ovarian cysts or pedunculated fibroids, and finally secondary infection of the hæmatoma following an ectopic gestation. Common to both sexes, appendicitis and diverticulitis are frequent causes, while infected peritoneal exudates from any area may flow downwards and give rise to an abscess in the pouch of Douglas.

*Clinically*, therefore, these conditions may be post-operative or spontaneous, there being little difference between them except for the operation. There will be a history of the causative disease followed by operation or a period of apparent resolution. Then there is a falling off in the improvement and the patient begins to complain of low abdominal pain, vomiting, frequency and possibly difficulty of micturition and pain or difficulty when the bowel acts or an aperient is given. Examination reveals tenderness above the pubes and one or both Poupart's ligaments, whilst vaginal or rectal examination encounters the bulging tumour, which is tender.

*Treatment.*—There is no necessity for early operative interference provided the pulse rate and the general condition remain satisfactory, for these abscesses frequently burst into the rectum. A careful watch is kept, hot rectal and vaginal douches and short-wave diathermy are given. As soon as the rectal wall is getting thin, an opening

should be made into it through a speculum and a small drainage tube introduced. Similarly, but more rarely, these abscesses point towards the posterior vaginal fornix and may be drained by a posterior colpotomy.

**Iliac Abscesses** in the right and left iliac fossæ result from appendicitis in the former case and diverticulitis in the latter. Nothing special need be said about them except that their prevention requires early diagnosis and treatment, and should they form, simple incision and drainage with or without removal of the cause will be called for, the details of which will be found under the heading of the respective diseases.

### CHRONIC PERITONITIS

**Simple Chronic Peritonitis** is rare apart from tuberculosis, but certain forms are described, which are apparently not due to the Koch's bacillus.

An aseptic chronic variety is reported, though it is exceedingly rare. It is believed to occur in connection with the retention of a sterile foreign body within the peritoneum, especially after operations. Eventually, after a period of ill-health the patient succeeds in extruding the foreign body either via the rectum or the abdominal wall.

Chronic generalised peritonitis is a very rare condition. A thick gelatinous membrane is formed over the small intestine, which leads to dense adhesions. Symptoms of mild chronic intestinal obstruction occur with a state of chronic invalidism. No treatment is indicated unless obstruction supervenes, when the task confronting the surgeon may well prove insuperable.

Chronic localised peritonitis is also rare apart from tuberculosis. Plastic adhesions may form around any variety of intestinal disease, but this does not constitute a primary chronic peritonitis. It is quite evident from the published descriptions that the majority of these cases are in fact examples of the chronic type of regional ileitis (Crohn's disease), which have not been recognised as such by the authors.

### TUBERCULOUS PERITONITIS

Tuberculous peritonitis may occur in either sex at any age, but the first fifteen years of life provide the majority of cases, while after puberty most of the patients are young females. The infection may reach the peritoneum in several ways: (a) via the intestinal canal, the bacilli having been ingested with infected milk; (b) by direct spread from any focus in the neighbourhood, such as genito-urinary infections, spinal caries, psoas abscess, etc.; (c) it may extend to the pelvic peritoneum from the Fallopian tubes in young women; and (d) it may be a blood or lymph-borne infection from the lungs, cervical or mediastinal glands or from any other tuberculous lesion in the body.

**Acute Miliary Tuberculous Peritonitis** is hardly of surgical interest, and is dealt with in textbooks of medicine. It is always part of a terminal infection and no treatment is of any avail.



**Chronic Tuberculous Peritonitis** is unhappily a very common manifestation of the disease, especially among children and young girls. It may take several forms, namely: (1) ascitic, (2) encysted or loculated, (3) adhesive and (4) purulent.

**The Ascitic Type.**—The peritoneum is thickened and studded with tubercles, and adhesions are present between coils of intestine, while the great omentum is thickened and rolled up, so that it forms a transverse band across the abdomen just above the umbilicus. There is a profuse, clear, straw-coloured effusion, which contains tubercle bacilli. There may be active lesions of tuberculosis in other parts of the body.

*Symptoms.*—The child is ill, thin and fretful, but although there may be some abdominal discomfort, the symptoms are usually slight. The effusion is slow in onset and the picture is one of a progressive loss of health and vitality.

*Signs.*—The body is thin and the muscles are wasted, while the abdomen is prominently distended, the skin being tense and shiny and blue veins are to be seen coursing beneath the thin skin. All the classical signs of fluid are present, the shifting dullness in the flanks, a central area of resonance and a fluid thrill. Deep palpation reveals doughy masses formed by adherent coils of intestine, and the transverse band of omentum is clearly defined. There is an evening rise of temperature and the face has the clear lustrous skin with its hectic flush and the eyes have the brilliance which are associated with tuberculosis.

*Diagnosis* should not be difficult owing to the age of the patient, such lesions as the ascites of cirrhosis of the liver and of generalised carcinoma of the peritoneum being seen at a much later period of life.

*Treatment* is essentially institutional and surgical intervention should be avoided. The practice of opening the abdomen and letting out the fluid has little to recommend it and should be adopted only if the distension is so great as to be a serious embarrassment.

**The Encysted or Loculated Type** occurs in children, but more frequently in young women. A definite tumour is formed consisting of coils of intestine matted together, enclosing a loculated cavity containing a serous effusion. Tubercles and a thickened peritoneum are present in the affected area, but the rest of the peritoneum is normal.

The localisation in the peritoneum is an indication of the successful resistance on the part of the patient, and in this type the general health remains good for long periods. Vague abdominal pain may have been present for some time, and on examination a discrete mass can be felt usually in the lower part of the abdomen. If left untreated, the general condition deteriorates and there is a danger of intestinal obstruction, fistula formation between the coils or, more serious still, secondary infection from leakage of bacteria from the intestines. For these reasons, if the local condition does not respond rapidly to sanatorium treatment, this type of tuberculous peritonitis should be operated upon, the tumour mass explored and dealt with in accordance with the findings, the abdomen being closed without drainage.

**The Adhesive Type** is less common than the ascitic. The abdominal contents are adherent to each other so as to form one mass, from which it is wellnigh impossible to disentangle the composite parts. There is little or no effusion. Fistula formation is common in the later stages.

The *clinical picture* is one of gradually increasing weakness, wasting and abdominal pain, on which may be superimposed at any time symptoms of chronic or even acute intestinal obstruction. The abdomen is not distended, moves on respiration and is not tender, but palpation reveals such characteristic findings that the diagnosis is unmistakable. Although difficult to differentiate local swellings, there is a nodular doughy sensation over the whole abdomen, and the rolled-up omentum may be palpable. The impression is that of a generalised soft resistance without any muscular rigidity.

*Treatment* is entirely medical and surgical interference can be justified only in the presence of obstruction, and the outlook then is almost hopeless.

**The Purulent Type** is very rare and is secondary to tuberculous salpingitis in young adolescent females. True tuberculous pus is found in the pelvis and the condition is an acute one. The girl complains of low abdominal pain, rapidly wastes and becomes seriously ill. Treatment in these lesions is always surgical, with the removal of the diseased tube and ovary.

*General Prognosis* varies with the type, the ascitic and encysted forms holding out high hopes of recovery under the full régime of sanatorium treatment. In the adhesive type the outlook is grave and the mortality high.

*General Diagnosis* should never be really difficult. The encysted forms are frequently regarded as ovarian cysts, but in the generalised types the age and appearance of the patient and the clinical signs are such as should raise the suspicion of tuberculous peritonitis before any other disease.

GUMMATOUS PERITONITIS is a rare manifestation of tertiary syphilis, and still more rarely it may be seen as a congenital lesion. The peritoneum is thickened and there is ascites. The treatment follows general antisymphilitic lines.

R. M. HANDFIELD-JONES.

## CHAPTER XXVII

### HERNIA

#### SURGICAL ANATOMY

**D**EFINITION.—According to one authority the word “hernia” is derived from the Greek “Ernos,” meaning a bud or sprout, a perfect description of the appearance of a hernial sac.

The Oxford Dictionary, however, gives a Latin derivation and translates it as a rupture. The definition given is: “A tumour formed by the displacement and resulting protrusion of part of an organ through an aperture, natural or accidental, in the walls of its containing cavity.”

This definition is very exact, but it implies a knowledge that the cavity possesses first a lining membrane and second a wall so designed that it will resist the varying changes of pressure with it. It is such disturbances of pressure within the peritoneal cavity which force a portion of its contents into either a preformed sac (natural or congenital) or an acquired sac (accidental).

The clinical picture, diagnosis and treatment of all forms of peritoneal hernia are so consistently interwoven with a knowledge of the anatomy and mechanics of abdominal musculature that a brief account of these must be given.

#### THE WALLS OF THE ABDOMINAL CAVITY

These are bony and muscular. The osseous components consist of two portions: first, a central strut formed by the last dorsal, lumbar and sacral vertebræ, together with the 12th rib and pelvic girdle, which give rise to the majority of the abdominal muscles, and secondly, the 6th to 11th ribs, which form a movable lattice framework, giving insertion to most of these muscles. This central strut and the pelvic girdle therefore form a rigid basis upon which the movable thorax with its inserted muscles can exert their pull.

The peritoneal cavity can be regarded as having five normal boundaries (Fig. 264), viz., superior, inferior, posterior, lateral and anterior.

*The Superior Wall* is formed by the diaphragm, the two crura of which take part in forming the posterior abdominal wall.

*The Inferior Wall* consists of the pelvic floor which is hammock-shaped, being composed not only of the levatores ani but of fibromuscular tissues of great strength. This floor is liable to be weakened

by abnormalities in or damage to those structures which penetrate it, viz., the rectum in both sexes and the vagina in the female.

*The Posterior Wall* stretching from the 12th rib above to the 2nd sacral vertebra below gains great strength from the central osseous strut, which is covered in the upper part by the crura of the diaphragm. On either side lie the psoas muscles, beyond which the posterior wall extends to the lateral border of the quadratus lumborum muscles. These latter, though weak, are firmly supported behind by the sacrospinalis. Owing to the forward and downward sweep of the pelvic girdle, the iliac fossæ may be regarded as a continuation of the posterior abdominal wall. It should be noted that the psoas muscle increases the height of the pelvic brim and forms a gutter on either side of it.

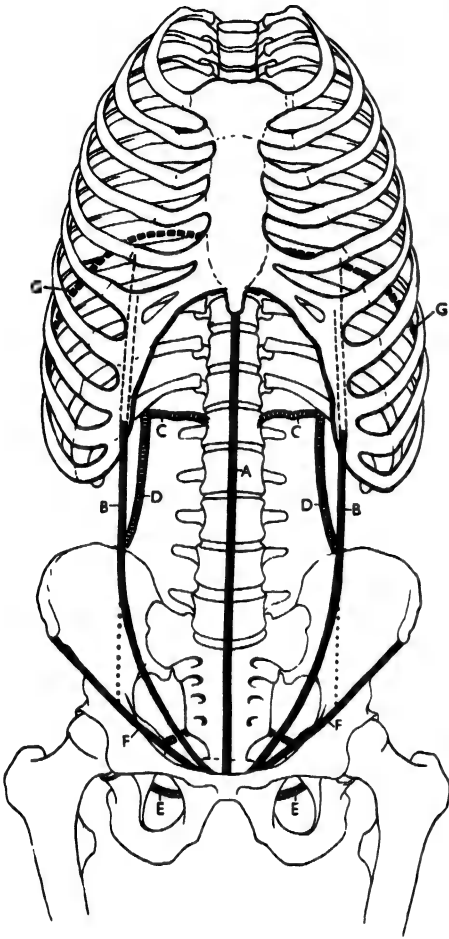


FIG. 264

The boundaries of the peritoneal cavity and their false and true ligaments.

A, linea alba; B, linea semilunaris (and its continuation above) marks the lateral border of the rectus muscle, and below (by the dotted line) the arbitrary junction of ventral and lateral walls; C, arcuate ligaments; D, lateral border of quadratus lumborum (false lumbar ligament); E, white line (junction of lateral and pelvic walls); F, Poupart's ligament; G, diaphragm.

*The Lateral Wall* consists of an upper major portion formed by the transversalis muscle and fascia and the two obliques; the smaller pelvic part is formed by that section of the pelvis which is covered by the obturator muscles and which lies above the white line (Fig. 265).

*The Anterior Wall* stretches from the thorax above to the pubis below, extending laterally as far as the tip of the 9th rib above and the midpoint of the inguinal ligament below. Thence it is continued into the pelvis as far as the lower border of the obturator foramen. Its main muscles are the recti and a part of the fibromuscular components of the lateral walls (Fig. 266).

### MECHANISM OF ABDOMINAL MOVEMENTS

The abdominal musculature is arranged to act synergically with the muscles of respiration. The strain is borne mainly between two structures, the long median linea alba in front and the lumbar vertebræ behind through the medium of the attachments of the lumbar fascia.

The coalescence of the three lateral abdominal muscles forms an artificial ligament at the lateral border of the quadratus lumborum, called the "false lumbar ligament." Between this and the linea alba there is a second ligament, viz., the linea semilunaris, at which point the lateral muscles split to enclose the rectus muscle.

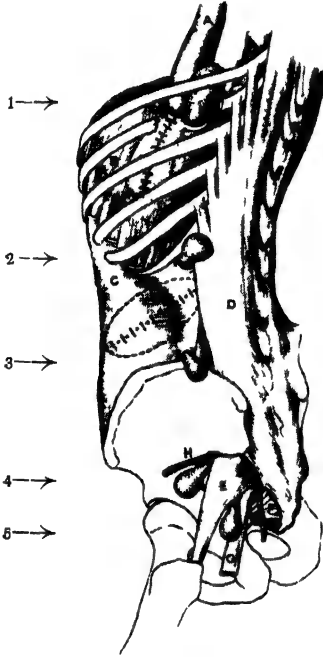


FIG. 265

Herniæ of the lateral boundary of the peritoneal cavity (numbered from above downwards).

1, central; 2, subcostal; 3, supra-crystal; 4, above the pyri-formis; and 5, below the pyri-formis.

A, œsophagus; B, diaphragm; C, transversalis; D, sacro-spinalis; E, pyri-formis; F, coccygeus; G, sciatic nerve; H, gluteal artery.

The direct pull of the lateral muscles upon the vertebra is important. The intermediate sheet of the lumbar fascia is strong, whereas the anterior layer is thin except at its upper border where it becomes thickened to form the arcuate ligament. The firm intermediate sheet reinforced by the posterior lamella gives rise to the transversalis muscle,

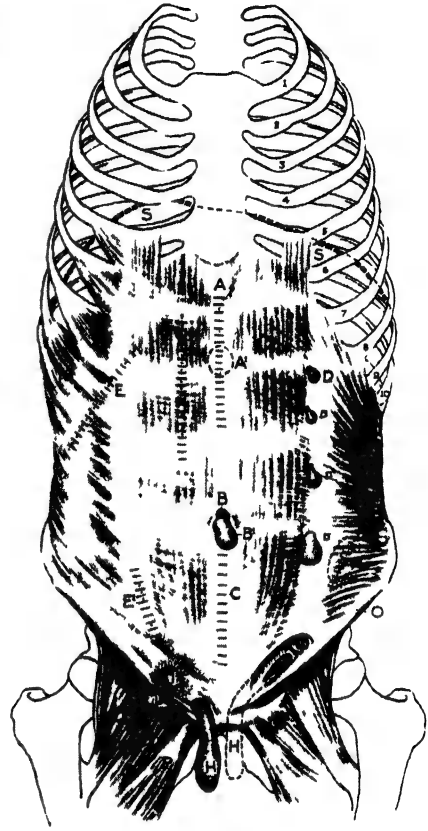


FIG. 266

The ventral abdominal wall and its associated herniæ.

A, epigastric hernia; A<sup>1</sup>, incisional hernia through the linea alba; B, para-umbilical hernia; B<sup>1</sup>, umbilical hernia; C, sub-umbilical incisional hernia; D, hernia through the linea semilunaris; E, Kocher's gall-bladder incision, the effect of which falls upon the rectus muscle. E<sup>1</sup>, Battles' incision likewise affects the rectus; G, hernia through a colostomy opening; H, indirect inguinal scrotal hernia; I, direct inguinal hernia; J, femoral hernia; K, obturator hernia; M, external oblique; N, internal oblique; O, inguinal ligament; P, adductor longus; Q, psoas; R, pectineus; S, the line of the diaphragm.

Note how the diaphragmatic line is reached by the external oblique and rectus muscles.

thereby enabling it to interdigitate with the diaphragm. The internal oblique using the former as a basis interdigitates with the intercostal muscles. The external oblique has a free posterior margin, but its inferior border forms the inguinal (Poupart's) ligament. This passes from the anterior superior iliac spine to the pubic spine, and is then continued backwards and outwards along the ileopectineal line to form the lacunar (Gimbernat's) ligament. The transversalis and internal oblique muscles arise not only from the iliac crest but also from the outer half of the inguinal ligament. Then by means of the conjoint tendon they gain a weak attachment to the inner half of this ligament and a firm insertion into the symphysis pubis and the iliopectineal line medial to and blending with Cooper's ligament. It is obvious that in contraction the main pull of these muscles is upon the linea alba, linea semilunaris, inner half of the inguinal ligament and the lumbar vertebræ. This pull is on a curved plane transversely and falls first upon the linea semilunaris and thence to the linea alba. At and above the umbilicus tendinous intersections of the rectus help to bind these two lineæ together and thereby resist the transverse pull. Below the umbilicus the pyramidalis muscle aids the recti to prevent their divergence.

*The Value of the Interdigitations.*—The construction of the abdominal wall ensures that movements of the abdomen are intimately connected with those of the thorax. It is impossible to raise pressure in one cavity without altering it in the other. The diaphragm gives a degree of independence to the two cavities in quiet but not in forcible movements such as coughing and straining, in which the whole thorax and abdomen are acting against the resistance of the pelvic girdle and floor.

The diaphragm and transversalis muscles can be considered as an entity which effects a squeezing action upon the abdominal contents. During their action there will be a tendency for the lower thorax to cave in, but the lower six ribs resist this collapse, and this is further opposed by the internal oblique interdigitating with the intercostals and so acting as a tensor between thorax and pelvis. This bracing action is reinforced by the interdigitation of the external oblique and serratus magnus, and of the rectus abdominis with the pectoralis major. In this way the thorax is firmly held and the maximum squeezing force brought to bear on the abdominal contents.

In the erect and sitting postures this force is expended upon the iliac fossæ and pelvic cavity. As this latter is filled with various organs including the ileum a cushioning effect occurs with the result that the main brunt of this squeezing force is thrown against the para-psoas gutters and in consequence against the inner half of both inguinal ligaments at a site where both inguinal and femoral canals are placed. When the knees are drawn well up on to the abdomen this region is protected and forcible straining will then fall upon the pouch of Douglas in the male and the posterior vaginal fornix in the female. Provided the pelvic floor is intact, this pressure causes venous congestion and eversion of the anal canal in both sexes.

## ANATOMY OF THE HERNIAL SAC

With the exception of a sudden giving way of a recent surgical wound, all herniæ are preceded by a diverticulum of peritoneum called "the sac." In insidious yieldings of surgical scars, a false sac may be formed from the peritoneal edges at the margin of the rupture.

*The Aperture* is defined as the margin of the opening through which the sac has passed. At this point, therefore, the latter becomes continuous with the general peritoneal cavity, and in consequence this part of the sac is known as its *neck*. The narrower the aperture and the more rigid its margins the more dangerous is the hernia, as strangulation of the contents is likely to occur.

Congenital apertures occur at the umbilicus, inguinal canals and diaphragm, and it is probable that persistence of the sac has prevented adequate closure of the aperture. Acquired apertures result from violence or necessitous surgery; in war wounds an example is provided by injuries to the diaphragm, while in civil surgery non-union of tissues in surgical incisions, especially at the site of drainage tubes, is a common cause of acquired herniæ.

*Contents of the Sac.*—With the sole exception of the pancreas, no abdominal organ is exempt from the possibility of entering a hernial sac. The commonest structure to migrate is the omentum and next a portion of ileum. According to the variety of hernia—the cæcum, transverse colon, pelvic colon and bladder follow in order of frequency. As the sac increases in size, so do the volume and character of its contents, and it is not uncommon to see omentum and both small and large intestine represented in the same sac.

## CAUSATION OF HERNIA

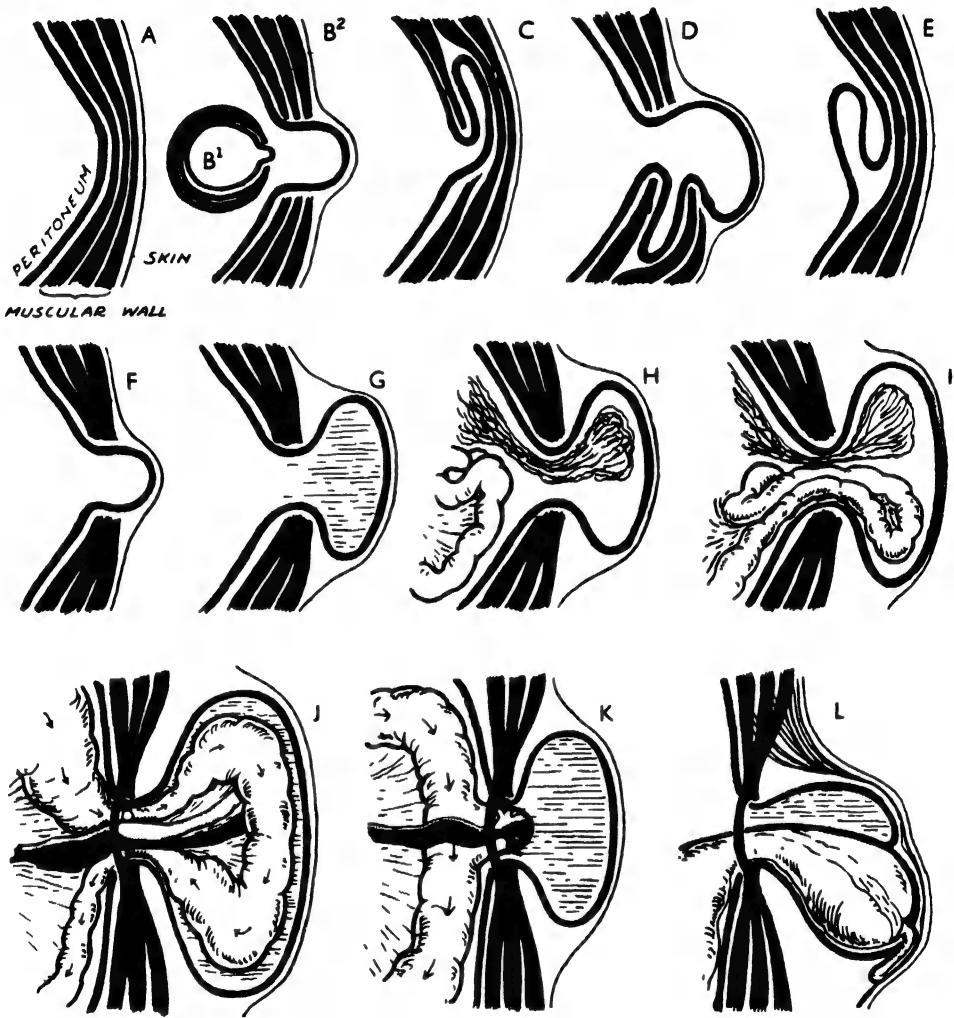
The exact causation remains a matter of dispute, some authorities maintaining that all herniæ are congenital, a bud of peritoneum not being withdrawn or obliterated but remaining *in situ*, providing a potential space for further changes (Fig. 267). It cannot be disputed, however, that some herniæ are acquired even at those sites which are considered to be most frequent for congenital sacs.

The existence of a sac does not necessarily imply the presence of contents. The rupture first "comes down" as a result of sudden or persistent increase in intracœlonic pressure, the commonest of which is straining against resistance during normal or abnormal work or during the performance of a normal function in face of difficulty, particularly defæcation and micturition.

**Congenital Hernia.**—In addition to those occurring in the inguinal and umbilical regions, some authorities consider all femoral herniæ to be congenital. Many undoubtedly are, but others appear to be acquired. Developmental defects in the diaphragm provide other sites for congenital lesions, which are less frequent than is generally supposed.

Exaggeration of natural folds in the peritoneum of the posterior abdominal wall, particularly in relation to the superior mesenteric artery and vein, is a well-recognised cause of retroperitoneal hernia.

**Acquired Hernia** is of three types—spontaneous, traumatic and infective.



The processes involved in hernial sac formation.

The diagrams illustrate the varieties of hernial sacs, the changes which occur in their contents, and the manner in which sepsis may produce incisional herniæ.

A, normal relation of peritoneum to the layers of the abdominal wall. B<sup>1</sup>, the sac is a bud of the lining membrane. B<sup>2</sup>, common type of hernia and its aperture. C, interstitial hernia. D, hernia with a diverticulum which is interstitial. E, retro-peritoneal hernia. F, the sac may be empty. G, fluid—hydrocele of a hernial sac. In peritonitis pus may be present. H, omentum only—epiplocele or omentocoele. I, omentum and intestine. Omentocoele and enterocoele. Note that the omentum lies in front and superficial to the intestine. J, a loop of intestine is strangulated. Note the changes in the intestine and the vessels, and the fluid present in the sac. K, a portion of the lumen is involved. Richter's hernia. L, hernia *en glissade*. Note the sac is above and in front.

Spontaneous hernia is due to traction, pulsion or a combination of the two. The majority are obviously due to pulsion. Traction herniæ are commonly due to extraperitoneal lipomata. Fat has the remarkable



property of oozing through small apertures and pulling neighbouring structures with it, and small lipomata often initiate the process of sac formation. Similarly the great omentum creeps through the neck of a small sac, widening and enlarging it.

Traumatic hernia is seen in surgical incisions, and is due either to the whole of the incision giving way or to one or more portions of it failing to unite.

Inflammation is a rare cause of hernia except in its association with surgical incisions. An abscess of the abdominal wall occasionally so destroys the tissues that an aperture is formed through which herniation may occur.

*Medico-legal Aspect.*—An employee can obtain compensation when it is proved that his work was the cause of his hernia. It is sometimes difficult to prove or disprove that an occupation was the cause or even a provocative agent of the hernia. In investigating such cases a most detailed history of the occurrence must be taken. In practice the workman is almost always given the benefit of any doubt.

### COMPLICATIONS OF HERNIA

The complications to which any hernia is subject are :—

- |                        |                   |
|------------------------|-------------------|
| 1. Irreducibility.     | 4. Strangulation. |
| 2. Hydrocele of sac.   | 5. Incarceration. |
| 3. Torsion of omentum. | 6. Infection.     |

**Irreducibility** is due to many causes. A plug of omentum may become impacted in a narrow neck and consequently œdematous. It is then too large and rigid to retrace its steps. Adhesions may form either between the sac and its contents or between the contents themselves. A truss by ill-placed pressure may cause constriction of the neck of the sac. Other causes are excessive deposition of fat in the omentum, fæcal impaction and strangulation.

**Hydrocele of the Sac.**—If the neck is unduly narrow and omentum plugs it but does not penetrate far into the sac, a fluid exudate from the œdematous omentum may cause a hydrocele of the sac.

**Torsion of the Omentum** may also occur, the signs and symptoms of which are indistinguishable from strangulation.

**Strangulation** occurs when the contents passing through the neck are of such volume that the vessels supplying them are compressed. This may happen upon the first occasion in which abdominal contents are propelled into the sac. In older patients it is usually due to additional contents being thrust into the sac by unusual violence of effort, such as lifting heavy weights, coughing, sneezing and straining at stool.

The *pathology* of strangulation is fully discussed in Chap. XXX on p. 632 in connection with acute intestinal obstruction; similarly the signs, symptoms, diagnosis and treatment of strangulated hernia are described in the same section of this book.

**Incarceration** is more commonly seen in a left inguinal hernia, occupied by a portion of the sigmoid colon. There is no interruption

with the blood supply, but fæcal material becomes stagnant within it and fluid is absorbed. As more fæces enter their bulk is so great that not only does the rupture become irreducible but chronic intestinal obstruction from fæcal impaction is likely to follow.

**Infection of the Sac** is an unusual phenomenon. It is seen when the appendix lying within the sac becomes acutely inflamed; other causes are Richter's hernia and Meckel's diverticulitis within a sac (the so-called Littre's hernia).

### EXAMINATION OF THE PATIENT

The examination falls into two parts, local and general, the former to determine the type of hernia present, the latter to assess the general constitutional condition of the patient and to collect data upon which an opinion as to treatment may be based. First, however, a careful history will have been taken.

*Symptoms.*—A patient with an external hernia without complications will complain of pain and swelling. Pain is most marked upon the first occurrence of the hernia and becomes progressively less as time goes on, as the contents descend more frequently and as the neck becomes sufficiently stretched to tolerate the passage of contents into the sac. Swelling is persistent or intermittent; if the latter it may be absent while the patient is at rest and appear only upon standing or straining.

Internal hernia is unlikely to give symptoms until complications set in.

*Local Examination.*—**A. Of the Hernia.**—The patient should first be lying upon a couch. Each hernial aperture is inspected and palpated, particular attention being directed to the suspected site.

If reducible, the contents of the sac will disappear within the abdomen either spontaneously on lying down or by manipulation. Omentum is silent when reduced, but the intestine gurgles and squeelches palpably and sometimes audibly in a very characteristic way. If the patient is asked to cough or strain, thereby raising the intra-abdominal pressure, the swelling reappears. It is then surrounded by the fingers of one hand and the patient again asked to cough, when an expansile impulse tends to separate the examining fingers and to push them a little further from the abdominal wall. As the force expends itself the fingers resume their original position. This is the well-known sign—"the impulse on coughing."

If a hernia is partially reducible it is usually the intestine which returns either completely or in part, the omentum remaining within the sac.

Attention is turned to the aperture and to the muscles in the immediate neighbourhood.

**B. The Abdominal Musculature.**—The patient is now asked to stand erect facing the surgeon who is seated. This part of the examination must be conducted with the patient first in relaxation and then in activity. The routine consists in observing and examining each potential hernial site, general build of the trunk, degree of physical fitness,

obesity and posture. Each set of muscles is actively contracted in turn, and in this way each group of structures will be thrown into relief or in the presence of much fat their outlines should be palpable. This examination should proceed in the following order—linea alba, rectus abdominis muscles, linea semilunaris, inguinal canals, femoral regions, lateral and posterior abdominal walls. This technique throws the hernia, if present, into bold relief; it also gives correct information as to the contents of the scrotum and the presence or absence of varicose veins.

General examination is directed towards the assessment of the patient's chances of deriving benefit from an operation. Diagnosis has already been achieved, what advice is the surgeon to give? Attention is directed first to the cardiovascular system with regard to the state of the heart and blood pressure; to the respiratory system to exclude all conditions causing a persistent cough; to the alimentary system with regard to constipation, distension and hæmorrhoids; to the urinary system to recognise any form of chronic urinary obstruction, such as phimosis, enlargement of the prostate or urethral stricture. Lastly, it is necessary to exclude any infective conditions in the vicinity of the hernia which might lead to sepsis occurring in the operation wound.

#### GENERAL PRINCIPLES OF TREATMENT

The possibilities in treatment may be summarised as follows :—

Reducible hernia—(a) truss or belt and pad, (b) injection, (c) operation.

Irreducible hernia which is not strangulated—operation.

Irreducible hernia which is strangulated—(a) taxis, and if this fails, (b) operation.

As a general statement it may be said that all patients with an irreducible hernia are in danger of intestinal obstruction. Secondly, even if the risk is high before the onset of strangulation, it is better to operate than to wait until that risk has been made desperate by the presence of intestinal obstruction. Thirdly, with the exception of certain cases of umbilical hernia in children, no rupture can be considered as permanently cured by the wearing of a properly fitting appliance. Fourthly, as a patient ages the more does his abdominal wall stretch and his intra-abdominal pressure increase. Fifthly, pulmonary, intestinal or urinary complications make the wearing of a truss less satisfactory and the occurrence of strangulation more likely.

**Truss.**—A truss can be fitted only to a reducible hernia. Its essentials are that it should be of reasonable price, hard-wearing, comfortable, relatively waterproof and control completely the hernial aperture, so that contents cannot enter the sac. A great many excellent trusses are on the market, and because one type does not suit a patient it does not necessarily follow that another will be unable to do so. In certain patients it is wise to incorporate the truss with an abdominal belt, the use of which is necessary in median, paramedian and lumbar herniæ.

A bilateral truss should always be ordered if there is a weakness of the opposite side and this should be a routine in patients doing heavy work, since a unilateral truss may encourage a hernia of the opposite side in heavy manual labourers.

A truss having been ordered, the surgeon must satisfy himself that it is a perfect fit not only in rest but in the presence of active movements. Each patient must be instructed to wear the truss upon all occasions, except for sleep at night, and in elderly people a light truss is even then advisable. A special rubber-covered truss can be obtained for bathing.

**Injection Treatment.**—The injection of sclerosing fluids has been practised in recent years, and many thousands of cases are reported from America and elsewhere in which results comparable with surgery have been claimed, with the added advantage that treatment is ambulatory. Cases for injection must, however, be carefully selected. It is essential that the rupture should be completely and easily reducible and kept so reduced by a truss during the entire period of treatment, *i.e.*, night and day for at least one month. The object is to produce an aseptic fibroblastic reaction around the emptied sac by injections of a sclerosing solution every third day. Eventually a mass of fibrous tissue is said to close the sac and obliterate the canal by adherence of its fibrous layers, thus curing the hernia. Complications are not frequent but include painful scarring, swelling of the testicle, impotence and gangrene; further, any subsequent operation is rendered infinitely more difficult. It cannot be said that this method has met with favour in this country, although a number of distinguished surgeons have given it an unbiased and fair trial.

**Taxis** consists in manual reduction of an obstructed or strangulated hernia. It is folly to attempt this procedure without proper preparation for immediate operation should the effort fail.

To be successful taxis must be painless and the muscles relaxed. Whether the patient is young or old, an adequate dose of morphia and atropine is given to diminish peristalsis and spasm, relieve pain and relax the muscles of the aperture. Until the morphia has reached its full effect, an ice-bag should be placed over the sac.

In babies and young children the legs should be slung to a gallows splint and the end of the bed tilted, a procedure which rarely fails to reduce the hernia. In adults a large pillow is placed under the knees and the end of the bed is raised on high blocks.

Taxis must be applied early and it is dangerous to use force greater than firm pressure. The sac is squeezed gently between the fingers of both hands, and the contents of the bowel, if possible, reduced through its lumen. The contents of the sac are then reduced, not from the fundus but by degrees from the neck. The direction of reduction necessarily follows the anatomical course of each individual hernia. The dangers of taxis are obvious, *viz.*, rupture of the bowel at the apex or neck, reduction *en masse* (Fig. 268) and interstitial reduction. If it is successfully accomplished, the patient should be kept in bed under careful observation for three days.

**Surgical Treatment.**—The methods available are : (1) herniotomy, *i.e.* removal of the sac ; (2) herniorrhaphy, *i.e.*, removal of the sac and suture of the aperture ; (3) hernioplasty, in which autogenous living suture material (fascia lata) is used to repair the aperture.

The results of operation are less satisfactory than they should be, a high percentage of recurrences following imperfect surgery. It should be remembered that a hernia occurs because either a natural valve has become incompetent (*cf.* indirect inguinal hernia) or a tendinous structure has given way (as in direct inguinal hernia); further, it is not possible for a surgeon to recreate an efficient muscular valve. The general principles, therefore, underlying all operations upon hernia will be the provision of as rigid a support as possible in place of the deficient valve or damaged aponeurosis. Where there is no loss of tissue and apposition without tension is possible, silk suture should suffice. If apposition cannot be obtained without tension, the gap must be filled in with a living fascial suture.

In babies a simple herniotomy is usually satisfactory, the internal oblique and transversalis muscles recovering their normal sphincter action. For all other patients herniorrhaphy or hernioplasty will be necessary to obtain sound repairs. No matter what age the patient, adequate preparation to avoid post-operative complications and to facilitate sound healing will amply repay both patient and surgeon.



FIG. 268

Dissection of the abdominal wall looked at from the peritoneal aspect. The rod lies in the internal ring. The sac, which has been reduced *en masse*, lies in the retroperitoneal tissues immediately below it.

### INGUINAL HERNIA

**Surgical Anatomy.**—The formation of the inguinal canal is intimately associated with the descent of the testicle. This process is described on p. 792. As the testicle passes through the layers of the abdominal wall, a valvular or semisphincteric arrangement is produced by the characteristic arching of the conjoint tendon (Fig. 269).

The relations of the inguinal canal are :—

*Anterior Wall*, aponeurosis of external oblique, origin of internal oblique in the outer half of the canal.

*Posterior Wall*, transversalis fascia, insertion of conjoint tendon in inner half of canal.

*Floor* is the gutter formed by the inguinal and lacunar ligaments.

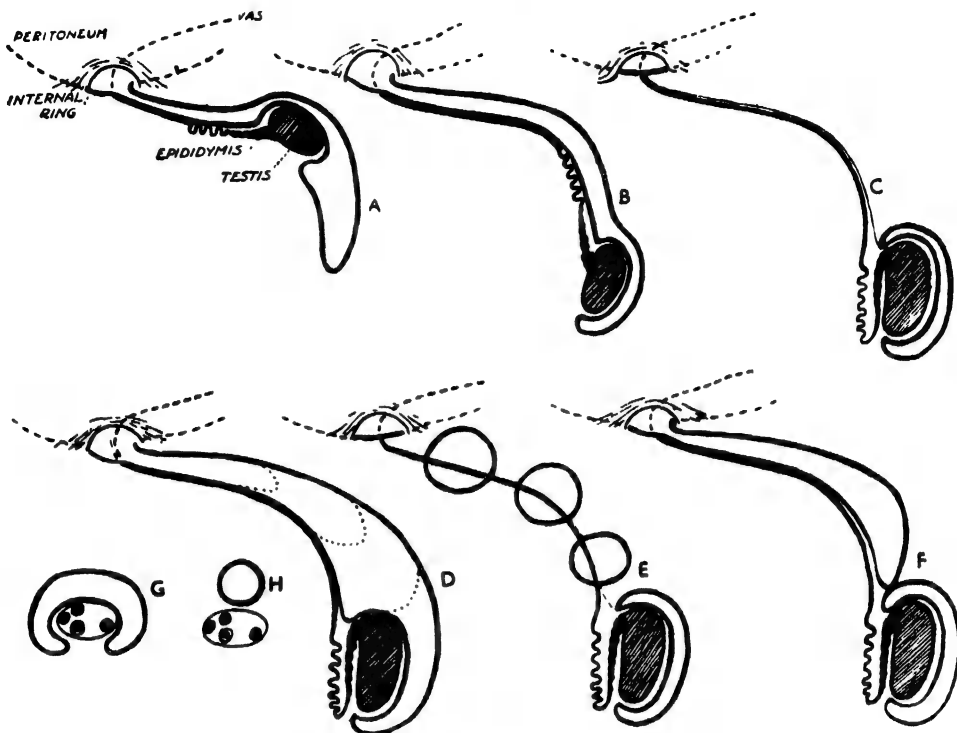


FIG. 269

The relation of the peritoneum to the descent of the testis. The potential sites of hernia and hydrocele formation.

A, the preceding peritoneum during testicular descent. B, patent processus vaginalis. C, complete or normal closure with the epididymis applied to the testis. D, potential sites of hernial sac formation. E, potential sites of hydrocele formation. F, common type of infantile hernia. G, a cross-section of the cord in a congenital sac. Note the sac nearly surrounds the cord. In the female the round ligament constantly shows this relationship. H, the relation of the acquired sac to the cord.

### INDIRECT INGUINAL HERNIA

The sac lies within the normal coverings of the spermatic cord and invariably lies in front of it (Fig. 270). In the female the round ligament takes the place of the cord. A congenital sac may be either complete, in which both processus and tunica vaginalis remain patent in their whole length, and incomplete when the processus is patent, but the tunica has been properly shut off. The neck is situated at the internal abdominal ring and has the deep or inferior epigastric artery as a contact medial relation. Small sacs do not always emerge from the external abdominal ring and this type of hernia is sometimes known as a "bubonocoele."

Many authorities maintain that all indirect inguinal herniæ are

congenital in origin, abdominal contents entering a preformed sac, no matter at what age the rupture first appears. This is probably true of the great majority and may possibly be so of them all. This type of hernia is common in both sexes, more so in the male, is frequently bilateral, but when unilateral is slightly more common in the right side. It is seen at any age, but the majority first appear in childhood and young adult life.

*Clinical Signs.*—The signs and symptoms are those of hernia in general. When small, it will be confined to the inguinal canal and not protrude beyond the external ring, in which case its nature and impulse on coughing may be best appreciated by passing a finger upwards into the canal by invaginating the scrotal skin in front of the examining finger. Such a hernia will disappear in recumbency and reappear upon standing, coughing and straining. Large herniæ come through the external ring and



FIG. 270

Part of the abdominal wall showing the inguinal canal, the femoral vessels and the spermatic cord. The sac of an inguinal hernia is seen projecting from the external ring lying in front of the spermatic cord.



FIG. 271

Large indirect inguinal hernia filling right side of scrotum and causing retraction of penis.

descend into the scrotum (Fig. 271). In this stage they are occasionally confused with a hydrocele, but this mistake should never be made. The upper part of the swelling should be surrounded by the fingers of the hand at the entrance to the scrotum. If the thumb in front meets the fingers behind with only skin and spermatic cord interposed, the condition cannot be a hernia descending from the abdomen.

Uncomplicated inguinal hernia give few, if any, symptoms. In the earliest stages patients may complain of pain, but later merely discomfort and a certain degree of mental apprehension as to future possibilities. A strangulated hernia suddenly becomes the seat of acute pain and tenderness and the general symptoms and signs of acute

intestinal obstruction supervene.

### DIRECT INGUINAL HERNIA

This is a rupture through the posterior wall of the inguinal canal being preceded by a rent in the transversalis fascia. The sac emerges between the inguinal ligament below and the arching fibres of the conjoint tendon, which it displaces upwards. It appears above the spermatic cord and displaces it downwards.

In this hernia the neck is usually wider than the fundus and is rather a bulging of the peritoneum than a true hernia. It carries with it extraperitoneal structures, of which the bladder is likely to be a medial relation; only when it is large can the sac be really described as having abdominal contents.

*Clinical Signs.*—This hernia usually occurs abruptly and with considerable pain. It is truly acquired and occurs most commonly in middle-aged, stout, plethoric working men. Rarely it is seen in young people who have a long back and correspondingly long abdominal wall, good but thin muscles and a congenital widening of the space between the lower margin of the conjoint tendon and the inguinal ligament. It is not uncommonly bilateral.

It will be recognised as a diffuse rounded bulge over the inner half of one inguinal ligament. When large it will project forward, but no matter how big it is it never enters the scrotum.

It may be confused with an indirect inguinal hernia, in old long-standing cases of which the internal and external rings may have become superimposed by the drag of a large rupture. In such cases the relationship of the deep epigastric artery gives the diagnostic clue; in direct hernia this vessel is lateral to the neck, whereas in indirect ruptures it lies medially.

### INTERSTITIAL INGUINAL HERNIA

In this rare form of hernia the sac lies between the various layers of the abdominal wall. Three varieties are described:—

1. Extraparietal hernia, in which the sac lies between the skin and external oblique aponeurosis;
2. Interparietal, in which the hernia passes upwards and outwards between the external and internal oblique muscles; and
3. Intraparietal or properitoneal between the peritoneum and transversalis fascia.

The last variety is misleading and dangerous, since there is no external swelling to betray its presence, and strangulation within it is likely to pass unrecognised. In the other two varieties, an external swelling is visible and palpable. In all three imperfect descent of the testis is usually coexistent.

### HERNIA EN GLISSADE

This curious type of rupture most commonly occurs in the inguinal canal, but is occasionally seen in femoral herniæ. The organs usually involved are the cæcum and appendix on the right, the sigmoid colon



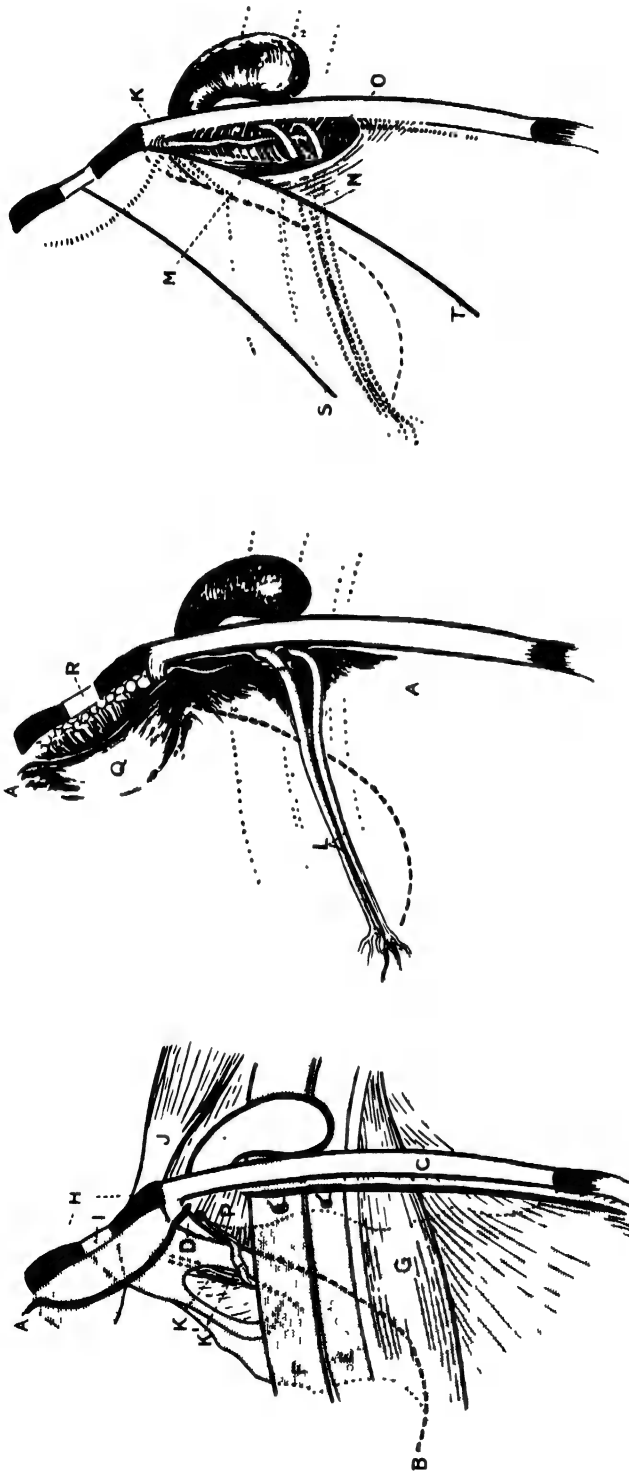


FIG. 272

The anatomy of a femoral hernia.

The view taken is a diagrammatic explanation of the viewpoint of a surgeon approaching a femoral hernial sac on the right side, the surgeon standing opposite the anterior superior spine and looking down upon the structures he is exposing in an operation through the supra-inguinal route.

- A, peritoneum.
- B, ileocepectineal line.
- C, inguinal ligament.
- D, lacunar ligament.
- E, femoral artery.
- F, femoral vein.
- G, psoas and iliacus.

- H, pubic spine.
- I, symphysis.
- J, adductor longus.
- K, obturator artery.
- KI, abnormal obturator artery.
- L, epigastric vein and artery.
- M, trans. and conjoint tendon.

- N, internal oblique.
- O, trans. fascia.
- P, pectineus.
- Q, bladder.
- R, prevesical fat.
- S, line of linea alba.
- T, linea semilunaris.

on the left and the bladder on either side. But these do not enter into the sac in the ordinary way, but lie behind it, appearing to slide down the canal with the peritoneum reflected off their anterior surfaces to form the neck and posterior wall of the sac. As a result the condition clinically appears to be an ordinary indirect inguinal hernia, but in fact the sac is empty and the "contents" extraperitoneal. This type of hernia is rarely recognised before operation. It occurs in middle-aged people, is usually irreducible but rarely strangulated. Treatment is difficult, a truss failing to control the rupture and at operation it is not easy to obtain a satisfactory closure even with a living suture of fascia lata.

### FEMORAL HERNIA

*Surgical Anatomy.*—This hernia occurs in the gutter between the medial margin of the psoas and the pectineus muscle. It emerges by passing beneath the inner part of the inguinal ligament (Fig. 272). The sac traverses the femoral canal and therefore has the following relations. On its medial aspect is the lateral border of the pubic spine and iliopectineal line, to which is attached the lacunar (Gimbernat's) ligament, the edge of which is directed upwards and outwards, being obliquely situated rather like a semilunar valve. Lateral to the sac is the soft but large femoral vein lying within the vascular sheath. In its passage the sac lies upon the pectineus fascia and eventually meets the septum crurale (fascia propria of Cooper). In spite of statements to the contrary it does not customarily emerge through the cribriform fascia but turns laterally between the superficial layer of the



FIG. 273

A left femoral hernia.

deep fascia and the tendon of adductor longus, thereby coming to lie superficially to the former. Any increase in size, therefore, tends to direct the sac upwards and outwards in the direction of the anterior superior spine (Fig. 273). In the thigh the fundus is lying quite superficial and is crossed anteriorly by the superficial circumflex iliac and superficial epigastric arteries.

The neck occupies the crural ring where the space available is very restricted; consequently the neck always remains small and the danger of strangulation by the sharp edge of the lacunar ligament is ever present. In this situation the neck is in an intimate relationship to that branch of deep epigastric artery which anastomoses with the obturator. This anastomotic vessel occasionally takes the place of the latter vessel,

being known as the "abnormal obturator artery." This is in some danger during operation for relief of a strangulated hernia.

Femoral hernia is not so common as the inguinal, but nevertheless is a frequent cause of disability. It affects women more often than men and is frequently bilateral.

*Clinical Signs.*—These herniæ rarely reach a large size, and usually remain quite small. Patients may complain of pain and are probably aware of the presence of a swelling. Owing to the narrowness of the neck and the rigidity of its walls, most femoral sacs have no abdominal contents, in spite of which a definite tumour can be palpated. During its passage through the femoral canal to its superficial position in the thigh, the fundus pushes in front of it a quantity of extraperitoneal fat and superficial fat. These tissues are compressed and becoming partly brosed adhere to the apex of the sac; in this way a tumour is produced even when the sac is empty. Such herniæ, therefore, will be irreducible and there can be no impulse on coughing.

In larger herniæ omentum and intestine enter the sac. Reduction will be obtained by applying pressure first in a downward, backward and inward direction and then upwards and inwards.

Treatment is always operative for two reasons: firstly, the difficulty of fitting any truss with comfort; and secondly, the grave danger of strangulation.

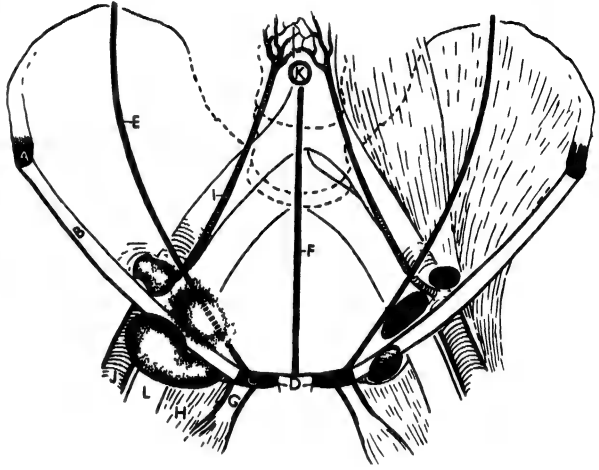


FIG. 274

The surface markings of the inguinal regions together with the areas which can be palpated in a patient.

On the right the hernial orifices are shown and on the left the directions that the hernial sacs tend to take.

The dense black areas or lines are palpable.

A, anterior superior spine; B, inguinal ligament; C, pubic spine; D, symphysis; E, linea semilunaris; F, linea alba; G, adductor longus tendon; H, pectineus; J, femoral artery; K, umbilicus; L, femoral vein.

The epigastric artery (1) arises from the femoral artery which is situated at the mid-point between the symphysis pubis and the anterior superior spine. The internal ring, therefore, must be lateral to this and lies at the mid-point of the inguinal ligament. In the angle between the deep epigastric artery and the linea semilunaris lies the direct hernia above the inguinal ligament; the femoral hernia lies in the angle between the adductor longus tendon and the inguinal ligament and is, therefore, below the latter.

## DIFFERENTIAL DIAGNOSIS OF INGUINAL AND FEMORAL HERNIÆ

The commonest clinical fault is a failure to distinguish between these two herniæ. This mistake need never be made if patients are examined properly. The method may be summarised thus:—

"First define the anterior superior iliac spine and the pubic spine, then palpate in its whole length the inguinal ligament, remembering that it is convex downwards. Keeping the fingers of one hand in firm contact with it, define its exact relationship to the NECK of the sac. Disregard the fundus and concentrate all your attention upon the neck and the inguinal ligament" (Fig. 274).

In inguinal hernia the neck will be above the ligament, and in femoral hernia it will be below.

### RICHTER'S HERNIA

This special type of rupture is described here as the great majority are seen in femoral herniæ. This dangerous condition consists in the impaction of a part of the antimesenteric zone of a coil of small intestine within the narrow neck of a hernial sac (Fig. 275).



FIG. 275

A Richter's hernia of the small intestine in a femoral hernia. The line of constriction is well shown and it will be seen that it does not embrace the whole of the gut-wall.

The effect upon the trapped area of intestinal wall is identical with the condition obtaining in a strangulated coil, except that only a small part of the gut-wall will become gangrenous. It will be understood that no symptoms will occur until impaction has taken place, and that every Richter's hernia therefore is strangulated.

*Clinical Signs.*—As the lumen of the bowel is not wholly involved a full picture of acute intestinal obstruction is not always seen. Vomiting is sometimes profuse and diarrhœa is not uncommon. The small size of the sac and its occurrence in fat patients may make the diagnosis difficult.

*Treatment* is immediate operation.

### INCISIONAL HERNIA

The causes of incisional hernia are: (a) misplaced incision, (b) insecure suturing, (c) destruction of nerve supply to abdominal muscles, (d) hæmatoma formation, (e) sepsis and (f) ill-chosen size and location of and prolonged retention of drainage tubes (Fig. 276). Increase in intra-abdominal pressure due to cough, intestinal distension or a combination of these must throw a great strain upon any incision. A drainage tube necessarily tends to increase the risk of herniation; further, the less valvular in form the incision is, the more likely is hernia formation.

In fat patients accurate apposition of fascia to fascia without the interposition of fatty nodules is difficult, and in consequence insecure union is probable. The abdominal wall is roughly convex and the first strain of a bursting force will be borne by those structures at the apex of the convexity, that is upon the muscle layer subjacent to the deep

fascia. Should the fascia give way it is usual for the muscles to slide laterally and for the peritoneum to protrude or rupture, thus permitting an escape of abdominal contents. Suprafascial hæmatomata, therefore, are unlikely to cause hernia formation, unless infected, and then only if infection spreads beneath the fascia. Subfascial hæmatomata, on the other hand, predispose to cutting out of sutures and separation of the fascial edges with consequent herniation immediately an increase of intra-abdominal pressure occurs.

In the presence of sepsis the proteolytic action of the pus digests tissues in the neighbourhood of the wound, converting this into granulation tissue, and as a result the wound is liable to stretch or burst during convalescence.

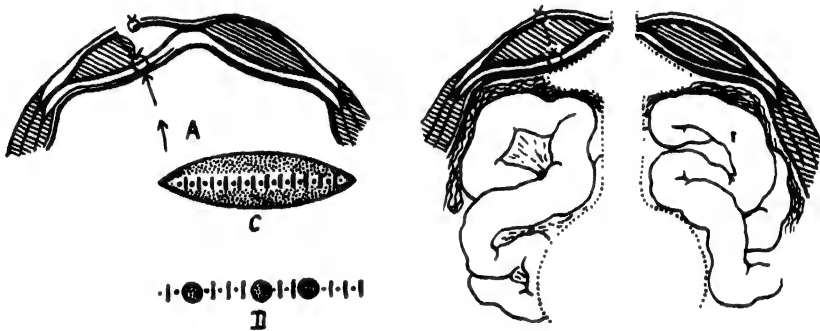


FIG. 276

## Incisional herniæ.

A, with abdominal distension the sutures of the anterior layer of the rectus sheath may give way. B, with pus in any quantity the exit is smaller than the pool, as a consequence of which an hour-glass collection is formed; the upper half consisting of pus held up beneath the anterior abdominal wall. Digestion of the sutures may occur. C, incisional hernia extending through the whole wound. D, multiple or single openings in the incision due to localised weakening.

When a drainage tube is inserted into a localised peritoneal abscess, it passes from a dependent pool of pus to the surface via a narrow neck in the abdominal parietes. This collection of pus is soon roofed in by an adhesive mass of omentum and small intestine, above which a small loculus is apt to form (Fig. 276, B). This second submural pool may spread along the under surface of the incision and digest a wide area of surrounding tissues as well as those structures in the immediate vicinity of the tube itself. Should this tube be too large or remain *in situ* too long, the adjacent tissues are converted into a fibrous track; directly the tube is removed omentum will plug the orifice and, as is its custom, creep through the opening, thus forming the beginning of an incisional hernia.

Destruction of nerves of the abdominal wall leads to paralysis and wasting of the segments affected. This is particularly prone to occur in Kocher's incision in an approach to the gall-bladder, in which the 8th, 9th and even the 10th intercostal nerves may be injured. This

would lead to a transrectal hernia from destruction of the nerve supply to the right rectus muscle above the umbilicus. A similar paresis may occur below the umbilicus in incisions made in the line of the linea semilunaris, where an inexperienced surgeon may divide nerves passing to the rectus muscle.

An incisional hernia may occur through the whole length of an incision or through one or more small orifices in an otherwise intact scar (Fig. 276, C and D). It may be immediate or remote; when the former it is termed a ruptured abdominal wound, when remote it occurs as a gradual yielding weeks, months or years later. In the early stages a hernia through the whole length of the incision beneath an intact skin mimics a divarication, while through a single small opening it behaves as a spontaneous hernia of the acquired type such as a para-umbilical hernia.

*Treatment* depends to some extent upon the site, but, generally speaking, the technique is based upon Mayo's operation for para-umbilical hernia.

### UMBILICAL HERNIA

**Congenital Umbilical Hernia** or exomphalos is a developmental defect in the formation of the anterior abdominal wall occurring in new-born babies (Chap. XXIX).

**Infantile Umbilical Hernia** is one of the commonest conditions seen in surgical out-patients of a children's hospital. It occurs during the first months of life and is due to a gradual yielding of the umbilical cicatrix. The hernia is covered by normal skin and is quite soft except when the child cries or strains, when it becomes larger and more tense. It is easily reducible and should be retained in place by a soft pneumatic rubber pad and belt; operation may be required later.

**Supra-umbilical Hernia** is sometimes incorrectly described as an acquired umbilical hernia. Its orifice, however, is immediately above the true umbilical ring, but as it increases in size it projects downwards and forwards and so involves the skin of the umbilical cicatrix. It occurs frequently in fat, middle-aged women in whom it may grow to great size. It will contain omentum in the early stages, but later the transverse colon and coils of small intestine will be drawn into it.

### OTHER HERNIÆ OF THE ABDOMINAL WALL

**Epigastric Hernia** is the term applied to herniation through the linea alba above the umbilicus. It may be false or true. A false epigastric hernia is a protrusion of a nodule of extraperitoneal fat through a small aperture in the linea alba, without any abnormality of the underlying peritoneum. It is by no means uncommon, particularly in men. It becomes tense on exertion, is painful and tender, and when the patient lies down becomes softer and is found to have a pedicle to which the mushroom head is attached. At the base of this pedicle on careful palpation an annular opening in the linea alba can be felt.

As this false hernia increases in size a diverticulum of peritoneum may be dragged after it; a true epigastric hernia has then appeared.

In neither case does this type of hernia ever become of any size, and it is very unusual for the latter ever to contain anything but a small plug of omentum. Owing to the dragging pain the small sac or lipoma should be removed and the gap closed.

**Divarication of the Recti.**—This is apparently due to absence of the linea alba, so that there is a space between the medial margins of the recti muscles. More commonly it is seen above the umbilicus, but may extend as far as the pubis, in which case it will be accompanied by exomphalos or an infantile umbilical hernia. In its most severe form other congenital defects, such as imperfectly descended testes, ectopia vesicæ or congenital heart disease, are likely to be present.

Although this is obviously a congenital lesion, a similar acquired condition is seen in both sexes, especially women who are either very obese or unduly thin and multiparous. On putting the recti into contraction the peritoneum between their medial margins bulges forward as an oval-shaped swelling. Sometimes the condition is so marked that the whole of the abdominal contents, with the exception of those in the retroperitoneum, lie within the sac.

Many incisional herniæ both above and below the umbilicus will behave in the same way as a divarication. In most patients a plastic operation will be called for to repair the defect.

**Hernia through the Linea Semilunaris** is an uncommon condition seen in young, spare men engaged in arduous occupations. Characteristically, there are several small nodules in the linea semilunaris opposite the entrance of the vascular bundles—they are segmental, therefore, in distribution and consist mainly of extraperitoneal fat and rarely give rise to a swelling of any size. They are analogous to false umbilical hernia.

**Lumbar Hernia.**—The common variety is that following a nephrectomy incision. The divided muscles fail to unite securely and their edges retract towards the last rib and iliac crest respectively. The resulting gap is consequently a wide one. The swelling is moderately large and bulges outwards on exertion.

Occasionally a spontaneous hernia arises in Petit's triangle through the false lumbar ligament. The aperture is bounded by the iliac crest below, medially by the latissimus dorsi and laterally by the free edge of the external oblique. A subcostal hernia is also said to occur, appearing immediately below the last rib and between the latissimus and external oblique. Both these herniæ are rare and are due to stretching and eventual rupture of the transversalis muscle at its origin from the false lumbar ligament.

## HERNIA THROUGH THE PELVIC WALLS

**Sciatic Hernia** is formed by the yielding of the fascia covering the sciatic notch. The sac follows either (1) the gluteal vessels and emerges above the pyriformis or (2) the sciatic nerve and appears below this muscle. Whatever their course these herniæ come to lie beneath the gluteus maximus muscle. It is an extremely rare condition, but when present usually gives rise to acute intestinal obstruction, the nature

of which is discovered only at operation. It may, however, give rise to attacks of apparent sciatica and in exceptional cases may obtain so great a size as to form a visible swelling in the buttock.

**Obturator Hernia** is another rare lesion. The sac emerges below and medial to the obturator vessels and nerve by passing through the inner margin of the obturator foramen. It comes to lie beneath the

pectineus muscle which separates it from the femoral artery and vein (Fig. 277). This hernia can be suspected only when there are intermittent attacks of pain referred along the course of the obturator nerve to the knee-joint. Usually it makes itself evident by a sudden attack of intestinal obstruction and its exact nature is discovered at operation.

**Perineal Hernia** is unlikely to occur except in association with prolapse. The pelvic floor is very strong; it consists of the levatores ani reinforced by dense fibromuscular material containing a quantity of unstripped muscle. Damage to the pelvic floor, the commonest cause of which is childbirth, usually manifests itself in the form of uterine prolapse in varying degrees of severity.

A true perineal hernia has been described and is said to occur through the pouch of Douglas and emerge between the anus and perineal body. A case is recorded in which the hernia reached as far as the knees. It should be borne in mind that in marked prolapse of the rectum in the male and of the uterus in the female, a sac with a potential

hernia will descend in the region of the pouch of Douglas together with the prolapsed viscera.



FIG. 277

The pelvic musculature and associated hernial sites. The pelvic cavity as seen from within, showing the relations of the herniae which may occur below the pelvic brim.

The true perineal hernia passes between the two levator ani muscles. The obturator hernia is above and in front of the levator ani attachments and therefore belongs to the ventral wall; the herniae between the obturator internus and the attachment of the levator ani and above and below the pyriformis muscle, are hernias of the lateral abdominal wall.

Notice how the sacrum forms a part of the posterior abdominal wall.

A, pyriformis; B, coccygeus; C, obturator internus; D, levator ani; E, ileopectineal line; F, inguinal ligament; G, quadratus; H, white line; I, anus; J, internal urethra.

### DIAPHRAGMATIC HERNIA

Diaphragmatic hernia is of great interest and, owing to improvement in diagnostic methods, is found to exist more frequently than was previously supposed. It consists in the herniation of abdominal contents into the chest through an aperture in the left leaf of the diaphragm. It may be either congenital or acquired in origin.



Congenital diaphragmatic hernia may be classified into two main groups according to the length of the œsophagus. In some cases this latter structure is congenitally short and as a result surgical interference is not possible.

The congenital herniæ are of five main types: (1) that due to absence of both crura, giving rise to the condition called "*diaphragmatica transversa*," the organs passing into the posterior mediastinum; (2) that in which the left crus only is missing, the costovertebral hernia; (3) that situated at the œsophageal opening which is congenitally

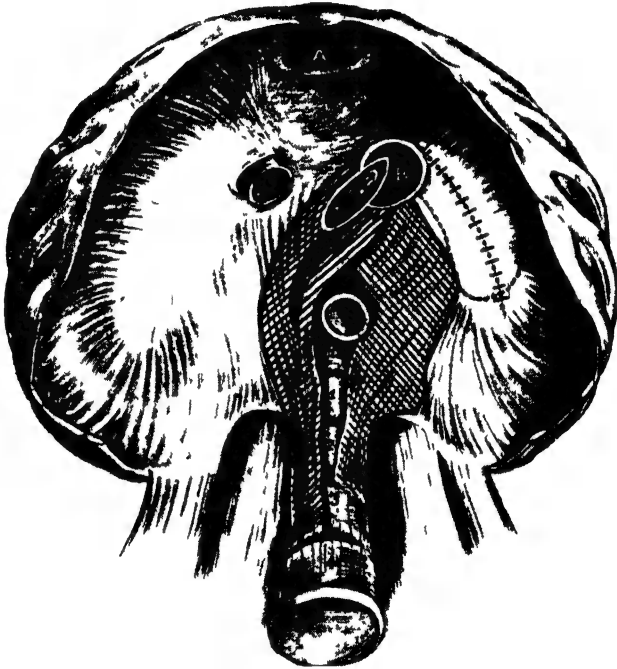


FIG. 278

Herniæ through the superior boundary. Diaphragmatic herniæ—seen from below (intra-abdominal).

A, retrosternal.

B, para-œsophageal.

widened so that a potential sac lies anterolateral to it, the para-œsophageal opening; this condition is commonly associated with a short œsophagus: (4) that occurring at the junction of superior and anterior abdominal walls, the retrosternal type; and (5) that due to absence of the left tendon of the diaphragm and described as a hernia through the left dome (Fig. 278).

Acquired diaphragmatic hernia is due to actual rupture of a part of the left leaf, either by violent exertion or by a penetrating wound traversing the pleuro-peritoneal cavities.

Neither congenital nor acquired herniæ are preceded by a sac of peritoneum, and the extent of abdominal contents entering the thorax depends upon the size of the aperture and the duration of the condition. The stomach in whole or in part is an invariable content, but almost all

the abdominal contents have been found in herniæ with large aperture. The heart is displaced towards the right side and the lung upwards towards its apex.

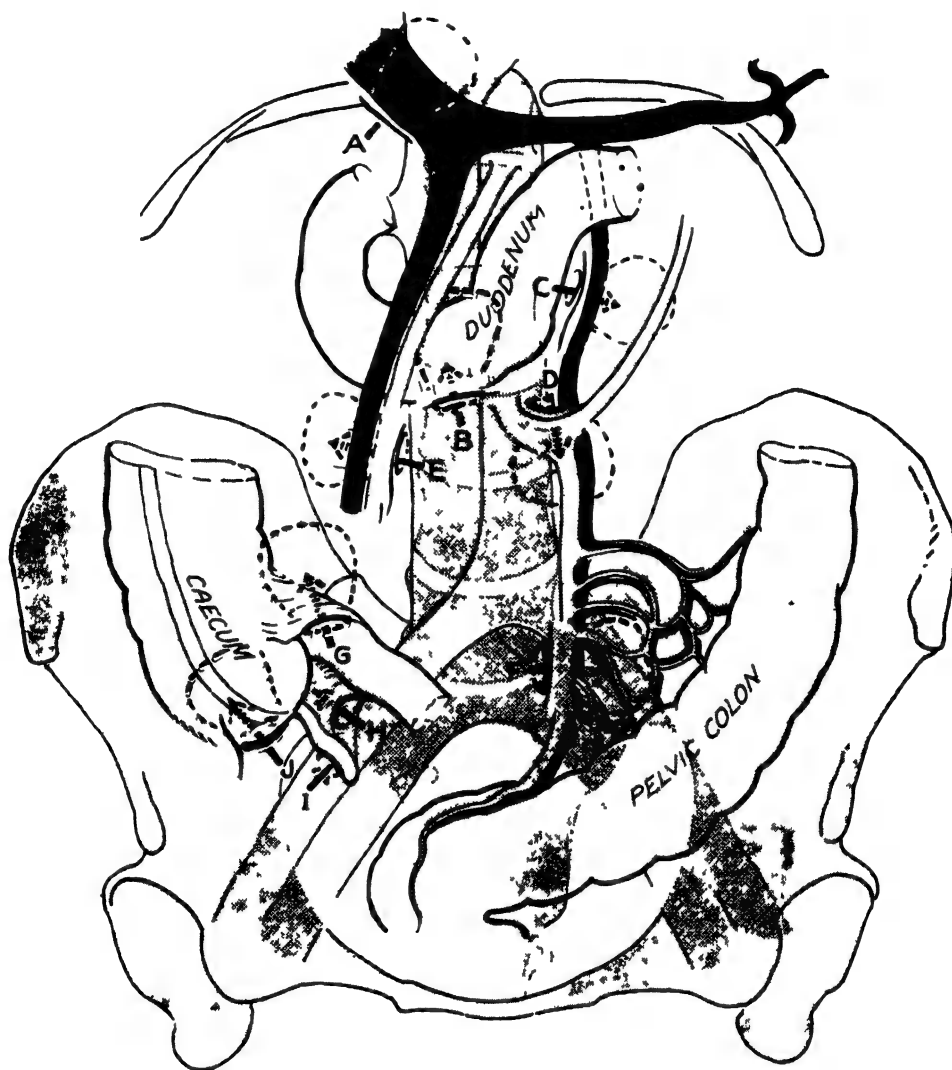


FIG. 279

Retroperitoneal herniæ. The potential sites of herniæ formation and the structures related to the neck of the sac.

A, hernia into the foramen of Winslow (portal vein) Paraduodenal herniæ. B, retroduodenal hernia, C, behind the inferior mesenteric vein; D, behind the left colic artery; E, behind the mesentery of the small intestine (superior mesenteric artery and vein). F, intersigmoid (left iliac artery and vein and sigmoid branches). Paracæcal. G, ante-ileal; H, retro-ileal; I, retro appendicular, J, retrocæcal.

*Clinical Signs.*—Many patients have no symptoms and they remain unaware of their abnormality. The others complain either of a vague, atypical dyspepsia or dysphagia with perplexing signs in the chest, or violent pain and collapse should strangulation occur.

*Treatment.*—Unless a congenitally short œsophagus is present, the contents should be reduced and repair of the diaphragmatic aperture performed.

### RETROPERITONEAL HERNIÆ

This type of rupture is uncommon, being either congenital or acquired. This latter is the more frequent and is due to rupture or insecure suturing of the mesentery in operations such as gastro-jejunostomy or resection of portion of the large or small intestine.

The congenital hernia is of three main types, paraduodenal, paracæcal and intersigmoid, each being due to the exaggeration of normal peritoneal folds (Fig. 279).

**Paraduodenal** are of four varieties :—

1. Retroduodenal, the commonest, is associated with a mobile third part of the modenum. The sac passes behind it in an upward and outward direction toward the liver, whilst its mouth looks towards the left iliac fossa.
2. Behind the inferior mesenteric vein. The mouth of the sac looks towards the right kidney, and its contents pass behind the vein towards the spleen.
3. Behind the superior mesenteric artery and vein. The sac passes towards the right kidney, and its mouth points to the left.
4. Behind the left colic artery. The sac goes towards left iliac fossa, and its mouth points towards the liver.

**Paracæcal** are four in number, their names describing their exact position, viz., retrocæcal, retro-appendicular, ante-ileal and retro-ileal.

**Intersigmoid.**—The contents pass behind the sigmoid branch of the inferior mesenteric artery and the pelvic colon into the left iliac fossa. The mouth, therefore, looks towards the right and is guarded by the iliac vessels behind and sigmoid anterior in front.

*Clinical signs* of all these herniæ are those of acute intestinal obstruction and diagnosis is made only at operation.

RUPERT VAUGHAN HUDSON.  
R. M. HANDFIELD-JONES.

## CHAPTER XXVIII

### THE STOMACH AND DUODENUM

**A**NATOMY.—The Stomach lies in the upper part of the abdominal cavity, below the liver and diaphragm. It is completely invested with peritoneum, being slung at its upper margin by the gastrohepatic omentum to the liver. Its lower surface has attached to it the great omentum, and so, to all intents is free. Its proximal end is firmly fixed by its continuity with the œsophagus, and its distal end is within  $2\frac{1}{2}$  to 3 in. of the fixed second part of the duodenum. It is apparent, therefore, that the stomach possesses a wide range of movement. In life the shape and position vary greatly according to posture, respiration, stage of digestion and distension or disease of neighbouring viscera. The shape as pictured in textbooks of anatomy is that of the dead stomach, and X-rays have demonstrated the changes which occur during digestion. The stomach has an anterior and a posterior surface, an upper and a lower border or curvature, an expanded left-hand extremity called the fundus, and a narrow tubular right end where it joins the duodenum, viz., the pylorus.

The anterior surface is in relation to the under surface of the left lobe of the liver, the left half of the diaphragm and the anterior abdominal wall. The posterior surface forms the anterior wall of the lesser sac of the peritoneum and through it, is in relation with the diaphragm, the spleen, the pancreas, the left suprarenal capsule, the upper pole of the left kidney and the transverse colon and mesocolon. The superior border or lesser curvature extends in a gradual curve from the right-hand margin of the œsophagus to reach the duodenum. Along its length is the attachment of the gastrohepatic omentum, by which it is slung in the upper abdomen. In its folds runs the coronary artery, a branch of the celiac axis, which reaches the œsophageal opening in the diaphragm and then turns down along the lesser curvature, sometimes as one vessel and at other times as two parallel branches, and anastomoses with the gastric branch of the hepatic artery. Towards the pylorus there is a notch in the lesser curvature named the incisura angularis, which marks the division of the body of the stomach from the pyloric antrum. The greater curvature arises from the left-hand end of the œsophagus, arches upwards and to the left to keep contact with the left dome of the diaphragm, and so forms the great cul-de-sac of the stomach, named the fundus. It then runs downwards, forwards and to the right, and finally curves upwards again to reach the pylorus. It is the free border of the stomach, and takes part in the greatest excursions of movement in distension of the organ. It has attached to its fundal part the gastrosplenic omentum, and to the remainder the great or gastrocolic omentum, in which run the right and left gastro-epiploic vessels. The œsophagus opens into the stomach by the cardiac orifice, which lies at the upper end of the lesser curvature and well behind and below the upper limit of the fundus. The pyloric opening leads to the duodenum, is guarded by the pyloric sphincter, and is marked on its peritoneal surface by the pyloric vein. The incisura angularis marks the point at which the stomach is arbitrarily divided into cardiac and pyloric portions. The former includes

the fundus and body of the stomach, while the latter is the small, narrow and cylindrical end proximal to the sphincter.

*The blood supply* is from the cœliac axis artery. The coronary artery and the gastric branch of the hepatic supply the lesser curvature. The right gastro-epiploic is a branch of the gastroduodenal artery which comes from the hepatic, while the left gastro-epiploic is a branch of the splenic artery. These two arteries traverse the greater curvature and supply both the stomach and great omentum. The fundus is supplied by the vasa brevia branches of the splenic artery. The veins follow their respective arteries, and all end in the portal vein.

*The lymph drainage* is best described by dividing the stomach into three areas by prolonging the left margin of the œsophagus downwards to meet the greater curvature (Fig. 280). This gives a fundal area and a main gastric area, which is again divided into an upper and a lower zone by drawing a line parallel to the greater curvature in such a way that the cardiac area is divided into an upper two-thirds and a lower one-third, and the pyloric area into equal upper and lower zones. The fundal area is drained by vessels going to the glands in the hilum of the spleen. The vessels of the upper cardiac zone drain direct into the main glands around the cœliac axis artery, while those of the lower zone run at right angles to the greater curvature, where they enter main trunks running in the omental attachment to end in the glands in the subpyloric region and along the pyloric end of the greater curvature. The efferents of all these glands end in the cœliac group.

*The nerves* are the right and left vagus and sympathetic branches from the solar plexus. The left vagus supplies the anterior surface, and the right the posterior.

**The Duodenum** is the first and most fixed part of the small intestine, stretching from the pylorus to the duodenojejunal junction at the left side of the body of the second lumbar vertebra. It is C-shaped, and into its concavity, which opens to the left, is fitted the head of the pancreas.

The first part is 2 in. long, and is invested completely with peritoneum in its first inch and on its anterior surface only in its second inch. In front is the under surface of the right lobe of the liver, above the attachment of the lesser omentum, and behind a small recess of the lesser sac, the common bile duct, the portal vein and the gastroduodenal artery. To its right is the gall-bladder in contact with which it turns downwards to become the second part.

The second part is 4 in. long and stretches from the gall-bladder to the right side of the third lumbar vertebra. Behind, it is in contact with the hilum of the right kidney and the right renal vein, the commencement of the right ureter and the termination of the common bile duct. Anteriorly it is crossed by the attachment of the transverse mesocolon, and so lies partly above and partly below the colic shelf. On its inner aspect it has the head of the pancreas.

The third part is  $3\frac{1}{2}$  in. long, and passes horizontally across the midline

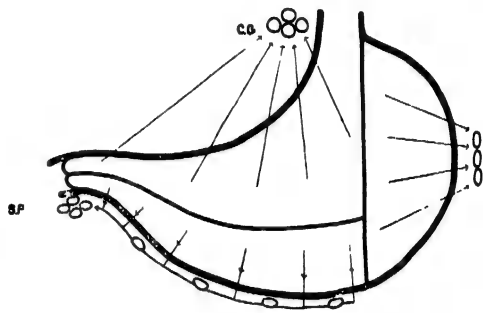


FIG. 280

Diagram showing the lymphatic drainage of the stomach.

C.G., the cœliac glands; S.P.G., glands in the hilum of the spleen; S.P.G., subpyloric glands.

to reach the left side of the 3rd lumbar vertebra. It passes in front of the right ureter, the inferior vena cava and the aorta. Its anterior surface is covered with peritoneum and is crossed by the root of the mesentery and by the superior mesenteric artery and vein.

The fourth part is  $1\frac{1}{2}$  in. long and runs upwards to the side of the 2nd lumbar vertebra, where it turns abruptly forwards to become the duodenojejunal flexure.

The blood supply is from the superior pancreaticoduodenal branch of the gastroduodenal artery, and from the inferior pancreaticoduodenal branch of the superior mesenteric artery. The veins pass to the portal vein, and the lymphatics drain into the pyloric and pancreaticoduodenal glands, the efferents of which end either in the coeliac or superior mesenteric group of juxta-aortic glands.

Four duodenal fossæ of the peritoneum are described as existing around the duodenojejunal flexure, and are said to be one of the causes of internal herniæ. They are named superior, inferior, paraduodenal and retroduodenal fossæ.

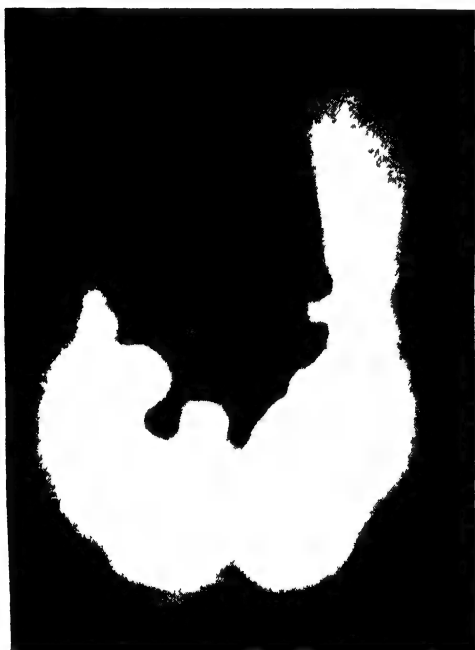


FIG. 281

An X-ray of a barium meal showing a lesser curvature ulcer.

## METHODS OF EXAMINATION

**A. Clinical.**—An exact case history will be sufficient to allow a correct diagnosis in many cases of gastroduodenal disease, and in few parts of the body is care and skill in this direction so profitable. A physical examination of the abdomen yields little information, unless areas of tenderness or a tumour be present.

**B. Test Meals.**—The fractional test meal is done with an Einhorn's tube, the gastric contents are removed, a meal of oatmeal gruel given, and then 15 c.c. withdrawn every quarter of an hour. In this way a determination can be made of the amounts of mineral chlorides, total acidity and free hydrochloric acid, and of the presence of starch, sugar, blood, pus or bile.

For diagnostic purposes test meals are far less reliable than barium meal radiography, but in early cases of malignant disease, for example, the combination of the two methods may be of service.

**C. Barium Meal Radiography.**—In the hands of a skilled radiologist the examination of the stomach and duodenum by the barium meal technique will yield over 90 per cent. of correct diagnosis. Screening will always give more information than photographs, and it should be understood that the exposure of a series of films does not constitute an efficient radiographic examination. The various findings will be described under the appropriate disease (Fig. 281).

**D. Gastroscope** has passed the experimental stage, and Hermon Taylor's flexible gastroscope marks a great advance. Although this method of examination must always have a limited field of usefulness, it is likely to lead to an earlier diagnosis of carcinoma than is possible by other means.

### CONGENITAL ANOMALIES

These are very rare, consisting of stenosis in the body of the stomach, at the pylorus, or in the duodenum at the ampulla of Vater. The stomach may be misplaced, *e.g.*, in the thorax in a congenital diaphragmatic hernia. These conditions are pathological curiosities only.

### INJURIES

Injuries of the stomach and duodenum are not common and may be classified as: (1) contusions; (2) rupture, which is spontaneous or traumatic; and (3) penetrating wounds.

**Contusions of the Stomach** are produced by crushing or "run over" accidents, in which no grave symptoms are present, but there is epigastric pain and vomiting of blood or blood-stained mucus. Provided no other injury coexists, a short rest is sufficient treatment.

**Rupture of the Stomach.**—The spontaneous variety, which is very rare, follows over-distension with food, acute dilatation or severe vomiting. The rupture is high up on the lesser curvature near the oesophagus, and the result is fatal. Traumatic rupture is rare, because the stomach is so well guarded by the costal margin, and its injury without any accompanying abdominal lesion is rarer still. It may result from a violent blow in the epigastrium, from crushes, "run over" accidents, the careless passage of oesophageal bougies or from over-distension with fluid during lavage or with air or gas. Cases are reported of intratracheal catheters being passed into the oesophagus by mistake and then attached to a mechanical anæsthetic apparatus with resultant rupture of the stomach. The area affected is usually the anterior surface near the greater curvature, and if the accident occurs when the stomach is full, peritoneal soiling may be extensive.

**Penetrating Wounds of the Stomach** are caused by stabs from sharp instruments, knives, daggers, bayonets, etc., or by rifle bullets or shell splinters. A few cases are recorded of perforation from within in people who swallow swords. In the majority of cases other organs are injured as well.

**Symptoms and Signs.**—These will depend on the size of the tear, the degree of gastric distension, the amount of food contained at the time and the co-existence of other visceral injury. In all cases some leakage into the peritoneum will occur, with some bleeding into the stomach. Peritonitis of varying severity and extent, and hæmatemesis are to be expected. In some cases so fine a perforation has occurred in an empty stomach that the rent has been closed by omental adhesions, or a small localised perigastric abscess may have formed, but usually the condition is more serious. At first there is a stage of acute onset with marked shock, an initial attack of vomiting, increasing abdominal

pain, and later the vomiting becomes re-established and severe. The vomit usually contains blood, and this will be the only symptom pointing directly to the stomach. The condition may be complicated by rupture of other hollow viscera or of a solid viscus resulting in intraperitoneal bleeding. Indeed, no typical picture can be given as the factors producing the clinical condition vary so widely.

*Treatment.*—Every wound which penetrates the abdominal wall MUST be explored, no matter how trivial the symptoms appear, and in all crushes and "run over" accidents a doubtful diagnosis should be synonymous with an exploratory laparotomy. The wound in the stomach is sutured in two layers with catgut, and the operator then turns his attention to the elimination of any other injury. After the usual peritoneal toilet the abdominal incision is closed with or without drainage as each individual case demands.

**Rupture of the Duodenum** is rare, except at the duodenojejunal flexure, where injuries are not uncommon. These are described under "Rupture of Hollow Viscera" (p. 529). When rupture of the duodenum does occur it may be partial or complete, intraperitoneal or retroperitoneal. It may be damaged in penetrating wounds in conjunction with other visceral organs. The symptoms and treatment follow the lines of those of gastric injuries.

### FOREIGN BODIES IN THE STOMACH AND DUODENUM

The swallowing of foreign bodies is largely confined to children, hysterical young women and insane adults. At one stage in their early life children are always putting things in their mouth, and they may swallow a variety of small toys, beads and nuts and bolts from their cots or pens. The hysterical girl is the type that eats her own hair and produces the "hair ball." Insane adults swallow the most surprising miscellany of objects, many of which are too large to be ingested with equanimity by the sane, *e.g.*, forks, knives and spoons. The author has operated on one female lunatic on three occasions to remove a full-sized dinner fork from her stomach, and on four occasions on another who swallows large sewing needles in pairs on a single loop of thread.

**Hair Balls** are produced by hair collecting in the stomach, where, being formed into a firm mass, it may become moulded to the shape of the organ with a projection upwards into the œsophagus and another forward into the pylorus. They are seen in neurotic women who nibble at the ends of their own hair. There is a specimen in the St Mary's Hospital Museum of two hair balls removed by Clayton Greene in 1912. A recent communication in the *Lancet* reported that this same patient had been operated on for the seventh time for hair balls (Fig. 282).

*Symptoms* depend on the type of foreign body present. A great many will pass through the pylorus, after which they will probably succeed in traversing the length of the intestinal tract and be passed per rectum. In those that fail to pass the pylorus, slight epigastric pain and nausea may be the first sign that anything is amiss, unless the fact that a foreign body has been swallowed is known. In these



cases no symptoms occur for several days until ulceration of the mucous membrane occurs

*Treatment.*—If the patient is seen shortly after the swallowing, a radiograph is taken. If the size and shape make it certain that there is no hope of the foreign body passing, an operation is performed as soon as possible; but if the contours suggest that it will pass the pylorus, soft milky foods, *e.g.*, porridge, bread and milk, should be given and a second film taken in twenty-four hours. In those cases in which no progress is made, not more than seven days should be allowed to elapse before the foreign body is extracted and the stomach sewn up with a double row of sutures. Hair balls will remain unsuspected for many months or years, until a mild dyspepsia calls attention to the abdomen.

Foreign bodies in the duodenum are very rarely seen clinically, as those which succeed in passing the pylorus are not likely to get held up in the duodenum. Long needles, however, may fail to negotiate the curve of the duodenum and penetrate its walls.

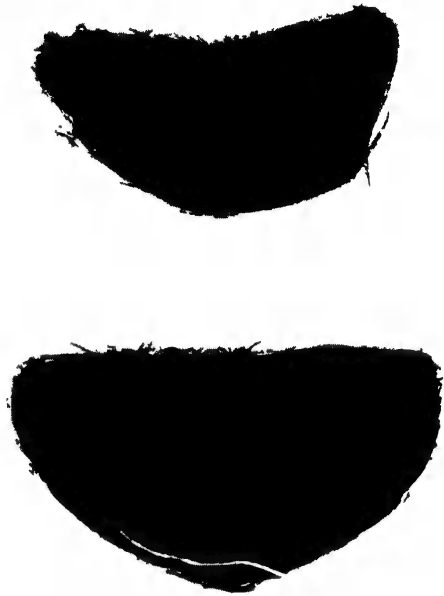


FIG. 282

Two hair balls removed from the stomach of a female patient.

### GASTRIC AND DUODENAL FISTULÆ

These fistulæ may be either external, in which the opening is on to the skin of the abdomen, or internal, when the communication is with another hollow viscus. Modern methods of diagnosis and treatment have made these conditions extremely rare.

**External Gastric Fistulæ** are now seen only as pathological curiosities. The most famous example is that of Alexis St Martin, whose fistula resulted from a bullet wound. An untreated carcinoma of the stomach might invade and break through the abdominal wall.

**Internal Gastric Fistulæ** may follow injury, but are usually due to disease. The communication is with the duodenum, jejunum, colon or gall-bladder, and the disease may be primarily in the stomach or in these other viscera. Ulcer and carcinoma of the stomach may invade the intestine, carcinoma of the colon may erode the stomach and chronic ulcerative cholecystitis with gall-stones may lead to a communication being formed with the pyloric end of the stomach. The symptoms of a gastrocolic fistula are persistent diarrhœa occurring shortly after a meal, the stools containing undigested food,

and less commonly the vomiting of fæcal material. These will be superimposed on the symptoms of the causative disease. The diagnosis will rarely be in doubt. A fistula between the gall-bladder and the stomach may lead to the vomiting of a calculus. The treatment is directed towards the cause, and may entail resections of a portion of both the stomach and the colon. The operation of gastro-enterostomy provides the classic example of a deliberately produced internal gastric fistula.

**External Duodenal Fistulæ** occasionally follow injury, particularly operative procedures. Operations on the biliary system (*e.g.*, trans-duodenal or retroduodenal choledochotomy) will rarely be followed by a fistula, but those on the right kidney provide the majority. The second part of the duodenum lies over the right renal hilum and its vessels, and is peculiarly liable to injury by careless manipulation or in the application of clamps to the renal pedicle, particularly if severe bleeding is occurring at the time. Fistula may follow a renal operation after an interval, as a result of sepsis in the renal space without efficient drainage. An abscess may form and erode the duodenum, and if the wound reopens then a duodenal fistula will result. Many of these fistulæ will heal spontaneously after a time, but if they fail to do so, an operation for their closure must be attempted, although it will prove a hazardous undertaking.

**Internal Duodenal Fistulæ** occur only with the gall-bladder as a result of chronic ulcerative cholecystitis with gall-stones; these latter will eventually be passed into the duodenum and be evacuated per rectum, provided they are not sufficiently large to become impacted in the lower part of the ileum. The symptoms are those of the causative condition, and modern methods should render this type of fistula a matter of historical interest only. After the stone has passed, the symptoms will probably subside, and it may be considered safer to leave the patient without attempting any operation.

## THE STOMACH

### INFANTILE HYPERTROPHIC STENOSIS OF THE PYLORUS

*Etiology.*—This condition occurs in infants during the first six weeks of life, being more common in first-born males, who are usually breast fed and among the more prosperous members of the community. The cause is imperfectly understood, and it is possible that there are two types, one in which there is a true overgrowth of the pyloric muscle, and another in which the symptoms are produced by spasm only. It is suggested that the change in the pylorus may be due to: (1) incorrect feeding leading to pyloric spasm, which eventually produces an actual hypertrophy; (2) hyperadrenalism both intra-uterine and after birth; (3) mass reflex action initiated by phimosis; or (4) some defect in the neuromuscular control of the sphincter.

*Pathology.*—The sphincter muscle is greatly hypertrophied without any fibrosis, as a result of which the pylorus is converted into an

enlarged firm cylinder about 1 in. in diameter and glistening white in colour. The mucous membrane is thrown into folds, and this further obstructs the lumen. The stomach is dilated and hypertrophied, but the duodenum is normal and into it the thickened pylorus projects with a groove around it, in a manner suggestive of the cervix uteri in the vagina (Fig. 283).

*Symptoms and Signs.*—The first symptoms appear at any time between the second and sixth week. Until then the baby has been healthy, with no unusual features in connection with appetite, feeding, weight and general condition, except that it may have been given feeds excessive in amount. The symptoms are vomiting, wasting and constipation. The vomiting rapidly becomes persistent and is projectile in type, the vomit containing neither bile nor blood. The loss of weight is very rapid and constipation is absolute. An examination of the abdomen will reveal visible peristalsis and a palpable tumour.

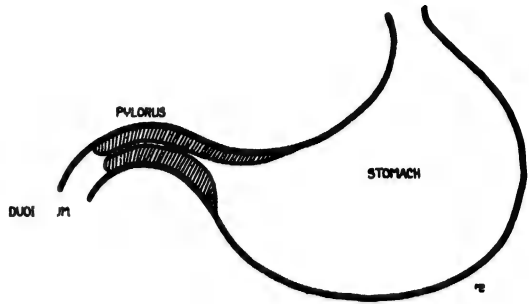


FIG. 283

A diagrammatic sketch of congenital pyloric stenosis.

*Treatment.*—A trial should always be given at the outset to gastric lavage and careful feeding. It is said that cases which recover with such treatment are spasmodic and not true hypertrophy. Nevertheless many babies with typical symptoms have been saved in this way. Small feeds given often at regular intervals are combined with careful lavage before each feed, which is introduced through the catheter before its withdrawal. If this fails to arrest the vomiting, and if the child continues to lose weight, no time should be lost in advising operation. The important point is that the decision to operate should not be left until the infant is too emaciated to survive the laparotomy.

*The Rammstedt Operation* is the only one practised to-day. It should be done under local anaesthesia and consists in dividing the thickened pylorus along its anterosuperior surface down to the mucous membrane, which will pout through the wound. Care must be taken to divide the fibres in the part which projects into the duodenum and to avoid injuring the mucosa of the duodenum or stomach. The final separation of the muscle fibres should be by blunt dissection with the handle of the scalpel. No attempt is made to cover in the pouting mucosa and the abdomen is closed. Gentleness and speed are highly important, and the mortality will depend on the surgeon's skill and the state of the child before operation.

### ACUTE DILATATION OF THE STOMACH

*Etiology.*—This is a definite clinical entity, and its importance must be appreciated by every practitioner. It may occur as a fatal

ending to chronic dilatation or other gastric disease, but is of the gravest significance as a post-operative complication, because it is rapidly fatal if not recognised. Of the many theories brought forward to account for it, only two are worthy of consideration. First, after operation the intestines sag down into the pelvis and drag on the mesentery, and as a result the superior mesenteric vessels are drawn tight over the third part of the duodenum and constrict it against the vertebral bodies; second, the condition is a paralytic one comparable to paralytic ileus, and is possibly due to pulling on the sympathetic plexuses around the celiac axis artery. The operations concerned are usually those on the stomach, gall-bladder, spleen, pancreas or kidney, but it may occur after almost any operation under general anaesthesia.

*Pathology.*—The stomach is enormously dilated, filling the whole abdomen. The distension never stops at the pylorus, but in many cases ends abruptly at the point where the superior mesenteric vessels cross the duodenum, while in others the upper part of the jejunum will participate.

*Symptoms.*—These are vomiting and abdominal distension. The ordinary post-anaesthetic vomiting having subsided within the first twenty-four hours, any further vomiting should be regarded with suspicion. It may be indicative of several complications, all of which are serious. In acute dilatation large quantities of greenish watery fluid are repeatedly vomited, the amount of the fluid being one of the most striking features. The distension is definite from the beginning and later becomes enormous. There is one feature of paramount importance in this distension; in that of the intestine, either colic or enteric, the epigastric triangle bounded by the costal margins and the transverse colon is never seriously encroached upon. In gastric dilatation it is in this triangle that the distension first appears and always remains most obvious, the transition from the flat chest-wall to the distended abdomen being markedly abrupt. The patient's general condition rapidly deteriorates from dehydration due to the profuse vomits. The pulse becomes thin, thready and rapid, and collapse soon sets in, death occurring within twenty-four hours. In some cases the dilatation is subacute and the vomiting less frequent and less profuse, the distension less marked and the general condition better maintained, but they will pass into the acute type unless treated.

*Treatment* consists in gastric lavage and the prone position. In the early stages if the slightest doubt exists, a Ryle's tube is passed into the stomach, preferably through the nose. The contents are completely removed by suction and warm sodium bicarbonate solution is run in. This process of siphonage and washing is continued until the washings are clear. The tube is left *in situ* and the stomach emptied every half-hour.

Should the case be left undiagnosed until large vomits are occurring, a large stomach tube must be passed as rapidly as possible. An attempt to swallow a small tube has been known to lead to aspiration of the vomitus into the trachea, death resulting. The stomach is emptied and washed as before and the tube removed. The patient is then placed flat on his face and the foot of the bed raised on blocks. If

another attack of vomiting occurs, this procedure is repeated. Success depends upon early diagnosis and prompt lavage. Secondary operations, such as gastrostomy and gastrojejunostomy, are useless.

### CHRONIC DILATATION OF THE STOMACH

This is not a clinical entity but merely a symptom of gastric and other disease, and comes under the two headings, viz., obstructive and atonic. The obstructive type can be further subdivided into intrinsic and extrinsic :

- |              |   |   |   |  |
|--------------|---|---|---|--|
| 1. Intrinsic | . | . | { | (a) Chronic ulcer of stomach and duodenum.<br>(b) Pyloric stenosis, infantile and acquired.<br>(c) Gastric polypus, blocking the pylorus.<br>(d) Fibrous stricture of the pylorus.<br>(e) Gastropotosis.<br>(f) Chronic duodenal ileus.<br>(g) Malignant disease of the stomach. |
| 2. Extrinsic | . | . | { | (a) Malignant disease of liver, gall-bladder, pancreas, colon and kidney.<br>(b) Perigastric adhesions.<br>(c) Mobile right kidney.<br>(d) Pancreatic cysts.<br>(e) Aneurysms.   |

The clinical features are considered under the sections dealing with the disease in question.

### GASTRIC TETANY

Tetany is due to parathyroid deficiency, but it does occur in other conditions, one of which is pyloric obstruction. Occasionally in these cases the typical spasms will be seen affecting chiefly the upper extremities. The hand goes into the position of *main d'accoucheur* and muscular twitchings occur all over the body. The stomach should be washed out and the cause of the pyloric stenosis dealt with.

### THE INFLAMMATORY DISEASES OF THE STOMACH

These may be classified as follows :—

Acute gastritis	.	.	.	{	Catarrhal. Phlegmonous. Suppurative.
-----------------	---	---	---	---	--

Chronic gastritis.

Linitis plastica or fibromatosis of the stomach.

Tuberculous and syphilitic disease.

Acute and chronic gastritis are described in textbooks of medicine, and tuberculous and syphilitic disease are so rare that no description of them is needed here.

**Linitis Plastica or Fibromatosis of the Stomach.**—This rare condition, also called “leather-bottle stomach,” consists of a widespread fibrosis in the submucous coat of the stomach, which may be wholly or partly affected. As a result the walls may be an inch or more thick, the

cavity diminished and elasticity lost. Undoubtedly most cases are due to carcinoma, but there are some in which no growth can be found, and which are due to infection from a chronic ulcer or to a toxic condition not identified. The disease is insidious in onset with vague epigastric discomfort and vomiting. A palpable tumour is to be felt and a diagnosis should be made by X-ray.

*Treatment* is gastrectomy.

### CARCINOMA OF THE STOMACH

*Etiology.*—Carcinoma of the stomach is the commonest of all cancers in men, accounting for about 22.5 per cent. of the total, and is the third commonest in women, being exceeded only by those of the uterus and breast. It affects men slightly more frequently than women, occurring between the ages of 35 and 70 years, although cases are on record before 20. Its causation is unknown, but its relationship to chronic gastric ulcer is discussed on p. 606.

*Pathology.*—Naked-eye appearance. Four types are seen :

1. The submucous scirrhus type.
2. The fungating cauliflower growth.
3. The ulcer, which is either (a) the ulcerating carcinoma or (b) the ulcer-carcinoma.
4. Leather-bottle stomach.



FIG. 284

A large fungating growth of the stomach.

**The Submucous Growth** is seen either at the pylorus or on the lesser curvature as a thick ivory-white mass in and beneath

the mucous membrane. At the pylorus it will spread round the canal and obstruct the orifice. It may cause the pylorus to project into the duodenum, but the infiltration stops short at that point and never affects the duodenal wall. From the main mass the growth spreads along the submucous plane, gradually becoming thinner until it can no longer be distinguished. Superficially it is ulcerated and infected. In this group the amount of fibrosis varies, some cases causing pyloric obstruction with little tumour formation, while others give rise to a large cellular growth.

**The Fungating Cauliflower Growth** appears as a large soft growth projecting into the lumen of the stomach, and infiltrating the stomach wall to a comparatively small extent (Fig. 284). It may grow to a

very large size before giving symptoms. It is seen usually in the body of the stomach.

**The Ulcerating Carcinoma** consists of an excavating ulcer with hard everted edges infiltrating the muscle and peritoneum (Fig. 285). The carcinoma arising in a chronic ulcer has the usual appearance of that ulcer and the growth arises at one side, usually towards the pylorus.

**The Leather-bottle Stomach**, when due to carcinoma, is widely and diffusely infiltrated with a scirrhus type of growth which spreads throughout the organ in the submucous layer. The stomach becomes contracted and very thickened.

*Microscopic Detail.*—These growths are either an adenocarcinoma of the columnar-celled type or a carcinoma simplex. The adenocarcinomata mimic very closely the normal gastric mucosa. The amount of fibrosis varies considerably, some being rapidly growing cellular tumours and others densely scirrhus. Some undergo mucoid degeneration and are known as colloid carcinomata. Oesophageal growths may spread into the cardiac end of the stomach and are squamous-celled tumours.

*Methods of Spread.*—Local infiltration occurs in the submucous, muscular and peritoneal coats. When the peritoneum is invaded adhesions form, and along these adhesions tumour cells spread to neighbouring

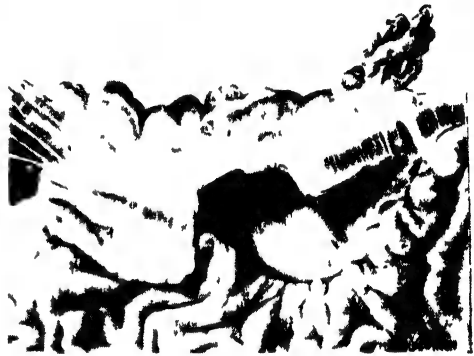


FIG. 285

An ulcerating carcinoma of the stomach.

viscera, *e.g.*, pancreas, liver, spleen and colon. Lymphatic embolism and permeation lead to involvement of the coeliac and subpyloric glands and later of those in the portal fissure. In rare cases the growth spreads up along the thoracic duct, and an enlarged gland appears in the left supraclavicular triangle. Venous embolism leads to deposits in the liver. The growth is apt to spread diffusely on the peritoneum, and structures in the pelvis may be involved by little fragments of growth dropping off the stomach and becoming engrafted; bilateral Krukenberg tumours of the ovaries are the most notable examples of this process. It is worthy of special notice that infiltration always stops short of the duodenum.

*Symptoms.*—It is impossible to present a composite picture of the clinical findings in carcinoma of the stomach, because patients arrive for advice with such a variety of symptoms. The picture will depend upon the type of growth and its position in the stomach. In the later stages all the symptoms may be present, but at least in the early stages it is convenient to classify patients into certain groups. These must not be considered as complete entities, but as affording an indication of the manner in which these patients first seek advice.

**Group I—The Dyspeptic Group.**—The onset is vague and indefinite



in a patient of 40 years and over who has previously been free of indigestion. At first some epigastric discomfort is combined with a failing appetite, and after some time the discomfort will become real pain, which tends to show no typical time relation to food, and further, is not relieved by food. Later, anæmia and wasting become marked features. A palpable tumour is of very late appearance. In those cases in which the carcinoma has resulted from a chronic ulcer, the typical ulcer history will have been present for a long time.

**Group II—The General Malaise group** consists of those patients who come first for advice complaining of loss of weight, loss of energy and being so easily tired. They may deny any indigestion or dismiss it as trivial, but they will probably admit that their appetite is not as good as it had previously been. They will be found to be anæmic.

**Group III—The Pyloric Stenosis Group** is well illustrated by a case of the author's, a lady of 60 years in perfect health who went to stay with friends in the country. One afternoon, without warning, she felt sick and a copious vomit resulted. This was repeated at regular intervals for a few days, when a barium meal revealed a pyloric stenosis.

**Group IV—The Silent Group** comprises a few cases who seek advice for other conditions, and in whom there is nothing to point to the stomach, the growth being discovered in a routine examination.

**Group V—The Rare Group** in which the symptoms are those of œsophageal obstruction, the growth being immediately in the region of the cardiac orifice.

Other special symptoms, usually late in occurrence, are bleeding, ascites and jaundice. Bleeding may be profuse but is rarely so; it may be a slow leak which shows in the vomit as darkish material designated the "coffee ground" vomit, but usually it can be recognised only as occult blood in the stools. Ascites and jaundice are evidence of liver and peritoneal involvement.

In general the picture is characterised by the vague and indefinite onset, and some patients continue to have an indeterminate story until the growth is inoperable. It must be appreciated that a palpable tumour usually means an inoperable growth. The lesson to be learnt is that every patient of 40 years and over who begins to complain of vague indigestion, loss of appetite and of energy, and who has a mild degree of secondary anæmia is in need of a thorough investigation. It is at this stage that a carcinoma is not only operable, but curable. The following findings may emerge from a routine overhaul: (a) a test meal may reveal diminution or absence of hydrochloric acid; (b) occult blood in the stools; (c) blood and fragments of growth in the vomit; (d) a blood count reveals a well-marked secondary anæmia; (e) a barium meal radiograph shows a filling defect, poor motility and delayed emptying time; and (f) gastroscopy. Of these the X-ray examination is of the greatest importance, but increased experience of gastroscopy will lead to an earlier diagnosis than is possible at present.

*Prognosis.*—These growths do not necessarily progress quickly, but their indefiniteness makes diagnosis difficult. Early diagnosis gives a high proportion of cures. The growths at the pylorus give the best results.



*Treatment.*—Growths of the pyloric region can be completely removed by a partial gastrectomy of the Polya-Balfour type. Growths of the body and fundus need a complete gastrectomy, while those near the oesophageal opening are invariably inoperable and will call for a palliative gastrostomy. Growths obstructing the pylorus which are inoperable will benefit temporarily from a gastro-enterostomy.

OTHER GROWTHS OF THE STOMACH are very rare. Sarcoma is seen in young people, myosarcoma in the middle aged, and examples of adenomata, fibromata and myomata are reported.

## PEPTIC ULCERS

Peptic ulceration is defined as ulceration of any part of the gastro-intestinal tract, the mucous membrane of which is in contact with gastric secretions. Under this heading, therefore, will be described :

1. The etiology and pathology of peptic ulceration in general ;
2. Gastric ulcers and their complications ;
3. Duodenal ulcers and their complications ;
4. Gastrojejunal or anastomotic ulcers ;
5. Jejunal ulcers.

### ETIOLOGY OF PEPTIC ULCERATION

In spite of a vast amount of research, the causation of peptic ulcers remains an unsolved problem. Certain facts are known, and some clinical and pathological observations yield suggestions of contributory factors. The known facts include :

(a) Ulcers occur in those parts of the gastro-intestinal canal bathed in gastric juice, *i.e.*, the stomach, the first part of the duodenum and the jejunum after gastrojejunostomy.

(b) The chronic progressive ulcer occurs chiefly in that part of the stomach known as the gastric canal or pathway, and in the duodenum. After gastrojejunostomy the anastomosis and part of the jejunum near at hand become physiologically part of the gastric pathway (see under Pathology, p. 599).

(c) Typical ulcers have been produced in the small intestine by diverting the bile and pancreatic juices to a lower segment of the intestine (Mayo Clinic).

(d) Normal gastric and duodenal mucous membrane is proof against autodigestion (*i.e.*, by their own secretions).

(e) Peptic ulcers are due to autodigestion of localised areas which have lost their normal protection.

(f) Hyperchlorhydria alone is unable to cause ulcer, for many cases of known acid excess remain ulcer-free, while only 23 per cent. of ulcer cases have hyperchlorhydria. It does, however, undoubtedly retard healing.

(g) The hypertonic stomach is one which empties too rapidly and produces its secretions in excess. This type is known to predispose to

duodenal ulcer ; and as it is common in men, this may explain the greater incidence of duodenal ulcer in men.

The factors which may be contributory are :

(h) Focal sepsis in the teeth, tonsils, nasal sinuses, appendix or gall-bladder may all coexist with ulcer. Rosenow's experimental work suggests an etiological relationship between ulcer and infection.

(i) Alcohol in all forms and cigarette smoking, particularly of the cheap Virginian brands, may have some causative significance. They undoubtedly delay healing.

(j) Pyloric spasm or stenosis is also suggested as contributory.

(k) Blocking of the arterioles of the stomach wall has been produced experimentally, and ulcers have resulted. This provides a most attractive theory that an embolus may block a vessel and cause devitalisation of an area of gastric mucosa, which loses its protection and becomes digested. But these ulcers are all acute and heal very rapidly, and all attempts to prevent their healing fail.

(l) A psychological background is present in many cases, especially duodenal, and the association of worry and overwork is a potent factor in etiology.

It is evident that the determining factor must be the cause of the local devitalisation of mucous membrane, which allows autodigestion to occur. It is this problem which has defied solution. Many causes are suggested, *e.g.*, sepsis, thrombosis and embolism of the gastric vessels, anæmia, high blood pressure or a toxin of unknown origin, and finally an unidentified virus is believed to be a possible factor

## THE PATHOLOGY OF PEPTIC ULCERATION

Three types are described :

1. The acute erosion ;
2. The acute ulcer ; and
3. The chronic progressive or indurated ulcer

1. **The Acute Erosion** is found in any part of the stomach, may be multiple or single, and is so small that it is hardly recognisable in life, even when the stomach is widely opened. At post-mortem the stomach wall may have to be held up to a strong light before the erosion is seen. When visible it appears as a small area in which there is digestion of mucous membrane, but muscle is not attacked. There is complete absence of any swelling, œdema or induration around it.

2. **The Acute Ulcer** also occurs in any part of the stomach and may be single or multiple. It rarely exceeds half an inch in diameter, is oval in shape and has a cleanly punched-out edge. The muscle coat is invariably affected in those specimens seen after death or operative removal. There is a slight hæmorrhagic effusion into the mucous membrane surrounding the ulcer, with some œdema but no induration. It is said that acute ulcers perforate, but this is uncommon : in such cases the ulcer cavity is funnel-shaped, with a broad base on the mucosal surface and a small perforation through

the peritoneum. As most specimens are post-mortem ones, it is not easy to give an account of their living pathology. They heal readily with little or no scarring. Erosions and acute ulcers are more commonly diagnosed in women, but there are grounds for the belief that a large number of people have small acute ulcers which form and heal in so short a time that they are not recognised.

3. **The Chronic Progressive or Indurated Ulcer** occurs chiefly in the gastric canal or pathway. The musculature of the stomach is so arranged that a tube can be formed along the lesser curvature, allowing fluids to pass, when the stomach is empty, direct from œsophagus to duodenum. The fundus and cardia form a digestive chamber, while the gastric canal transmits the products of digestion to the duodenum. The chronic ulcer, therefore, is found on the lesser curvature, on the



FIG. 286

A low-power photomicrograph of a chronic progressive peptic ulcer.

1, the overhanging proximal edge; 2, the sloping distal edge; 3, the muscularis mucosæ; 4, the muscle coat; and 5, the point at which muscularis mucosæ and muscle coat unite.

gastric walls adjacent to it, in the pyloric antrum and the duodenum as far down as the ampulla of Vater. After a gastrojejunostomy, the anastomosis and the segment of jejunum concerned come to be included in the gastric pathway.

The term chronic is somewhat misleading in that it applies only to the time factor and the after-effects of the ulceration. The process is not a chronic one in the sense that a tuberculous or syphilitic lesion is chronic, for at each recurrence the ulceration is definitely acute; but every attack leaves behind it a heritage of fibrosis and induration. From the clinical aspect the term will suffice, but it is better to bring it into line with pathological truth by adopting the name "chronic progressive," or "chronic indurated," ulcer.

Its appearance is typical, as shown in the accompanying section (Fig. 286). The ulcer is oval in shape, with its long axis at right angles to the lesser curvature. The proximal (œsophageal) rim is overhanging and undermined, and the distal (pyloric) edge is a gradual slope up from the floor. Almost every ulcer shows evidence of healing occurring

simultaneously with ulceration, the former taking place along the sloping distal edge and the latter progressing under the overhanging proximal rim. Four histological characteristics of a chronic progressive ulcer are described. They are :

- (a) Complete destruction of the muscle coat in the centre of the ulcer ;
- (b) Dense fibrosis in the base ;
- (c) Fusion of the muscularis mucosæ with the muscular coat at the margin of the ulcer ; and
- (d) Presence of endarteritis obliterans in the vessels around.

The old teaching that the chronic ulcer was "terraced" was based on post-mortem specimens, and is occasionally seen in the early stage of an acute exacerbation of the ulceration ; apart from this, terracing does not occur.

The complications of peptic ulceration are accounted for by its healing, progression and induration. The periods of remission of symptoms correspond to the healing of the ulcer ; penetration, perforation and hæmorrhage result from progression ; and stenosis and hour-glass stomach from fibrosis.

### THE UNCOMPLICATED GASTRIC ULCER

These ulcers are slightly more common in men than women, the chronic progressive ulcers being definitely so in the proportion of 4·5 : 1. But the acute ulcers being more common in women, the difference nearly balances the incidence in the sexes.

**Acute Erosions and Acute Ulcers** are commonly seen in young women of the chlorotic type, but they attack both sexes at any age, particularly between 20 and 45 years. They heal so rapidly that it is probable they occur in a great number of people without being diagnosed, giving short-lived attacks of epigastric disturbance, popularly termed indigestion. Usually they are recognised because of hæmatemesis, which may be severe but rarely fatal. Occasionally an acute perforation may occur.

Their *treatment* is medical.

### CHRONIC PROGRESSIVE GASTRIC ULCER

This is seen more commonly in men between the ages of 30 and 55 years, but it is not confined within these limits, *e.g.*, the author has recently operated on a boy of 18, and on three men of 71, 72 and 75 respectively. The age incidence in women is somewhat earlier. They occur in the pyloric antrum, on the lesser curvature and on the posterior wall in that order of frequency.

*Symptoms.*—The clinical picture is so characteristic that a high proportion can be diagnosed on the history alone. It consists of periodic attacks of pain after food. The intervals between the attacks may last as long as a year, during which the patient is perfectly well and unconscious of any gastric trouble. As time goes on the intervals tend to get shorter and the attacks last longer. This periodicity is

of the utmost importance in the diagnosis of peptic ulceration, and is due to the healing of the ulcer in the intervals of activity. Each attack lasts about three to five weeks. During the attacks the chief symptom is **pain**, which in each individual patient bears a constant relation to food. It varies between half an hour to two hours after a meal and is dependent on the position of the ulcer, the nearer the ulcer is to the pylorus the longer the delay in onset. The severity of the pain varies considerably and is not constant in the same individual. It is described as being in the epigastrium on one or other side of the midline, and may be referred to the back in the region of the inferior angle of either scapula. **Vomiting** is not a common symptom, but when present has the effect of relieving the pain immediately. **Constipation** is usually present, and a definite **loss of weight** accompanies each attack. Patients quickly learn that certain foods increase the pain and confine themselves to fish, milk and eggs without ever having seen a doctor. **Hæmorrhage** in the form of a recognisable hæmatemesis is not a symptom but rather a complication of gastric ulceration, although small quantities of occult blood in the stools will be found in all cases. After a prolonged history most ulcers lose their intervals of remission, and this implies that they have become adherent to an adjacent viscus, or rarely have undergone a carcinomatous change.

*Treatment.*—The treatment of the uncomplicated gastric ulcer is medical. Efficient medical treatment entails rest in bed for four to six weeks and aims at the following objectives :—

1. Avoidance of the stimulation of gastric secretion.
2. Neutralisation of such acid as is secreted.
3. Prevention of pyloric spasm.
4. Protection of the healing mucous membrane from trauma.
5. Provision of sufficient nutrition.

These objects are attained by giving small feeds at frequent intervals of milk, cream, arrowroot or Benger's, and adding later custard, junkets and mashed potato; by giving regular doses of alkalis, olive oil and belladonna; by avoiding all hard foods such as toast, rusks, etc., and by total abstention from all forms of alcohol and tobacco. Amongst the hospital class rest presents a grave problem in that sufficient accommodation is lacking, and the time required introduces the financial factor in an acute form. Finally, a strict routine of modified treatment has to be followed for at least a year. Full details will be found in textbooks of medicine.

The indications for operation are the failure of adequate medical treatment and the presence of any complication (see p. 602).

The surgical treatment of gastric ulcer has undergone many changes in the past twenty years. The poor results following gastro-enterostomy have led to more radical measures, and at the present time it is generally agreed that a partial gastrectomy holds out the best promise of lasting cure. It is undoubtedly a severe procedure, but even the most conservative opinion is now convinced that it is the best treatment available.

### THE COMPLICATIONS OF GASTRIC ULCERATION

These are in order of their frequency: (1) perigastric adhesions, (2) penetration, (3) perforation, (4) hæmorrhage, (5) pyloric stenosis, (6) hour-glass stomach, and (7) malignant change.

**Perigastric Adhesions** are seen sooner or later in all chronic progressive ulcers, and are due to a localised peritonitis. They are the first stage in the process that will end in penetration.

**Penetration.**—The ulcer, having become adherent to the pancreas, liver, anterior abdominal wall or other adjacent structure into which the ulceration finally spreads, leads to impairment of the motility, mobility and function of the stomach and makes any chance of healing remote. Pyloric and lesser curve ulcers frequently penetrate the pancreas, which forms the floor. The symptoms are those of the gastric ulcer which has lost its periods of remission, the typical dyspepsia becoming continuous.

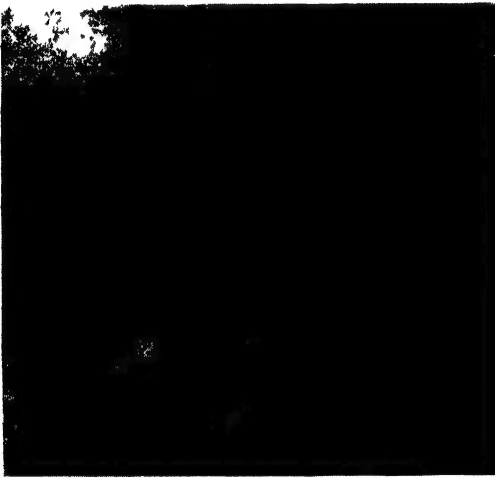


FIG. 287

An acute perforated gastric ulcer.

**Acute Perforation.**—When the ulceration is very acute the peritoneum is eroded before any localising peritonitis can occur. The size of the perforation varies from a pinhole to 1 in. in diameter, but rarely exceeds  $\frac{1}{2}$  in. (Fig. 287). There is some induration around the opening, and a considerable area of the stomach wall surrounding it is œdematous and inflamed. The vast majority

will be found on the anterior surface of the pyloric region, but an occasional example will be seen on the lesser curvature or on the posterior wall. Two simultaneous perforations have been recorded but are very rare. Perforation occurs in both sexes and at any age.

**Symptoms.**—The previous history will usually be typical of periodic attacks of ulcer pain; but if the ulcer is an acute one there will be a very short history, and some patients will deny any previous indigestion. The clinical picture is divided into three stages:

1. **PERITONEAL IRRITATION OR "PERITONISM."**—There is a sudden onset of agonising pain in the epigastrium, spreading rapidly over the upper half of the abdomen and later affecting the lower half, possibly more markedly on one side than on the other. There is an initial attack of vomiting. Within a few minutes the patient becomes collapsed, the face is white and drawn, the forehead covered with a cold, clammy sweat and the temperature falls below normal. The pulse rate remains steady or more probably becomes slow, but never gets rapid in this stage. Usually patients lie quite still on their backs or sides, but occasionally will be found sitting up with bent knees and hips and their

arms clasped across the abdomen. It is characteristic that they answer questions rather resentfully, and many use the same words: "Oh! can't you do something to take away this pain?" On examination the abdominal muscles are absolutely rigid, the abdominal wall does not move with respiration and there is generalised tenderness which is most marked in the epigastrium. Breathing is in short, sharp snatches.

2. **THE STAGE OF APPARENT RECOVERY.**—After a period of two to four hours the pain becomes less severe, and the physical signs less obvious. In some cases the pain and all the signs disappear, and the patient may be asleep when the doctor arrives to see him. This stage is likely to prove most misleading, and its existence must never be forgotten. The previous history and that of the sudden acute attack with collapse should suffice to arouse suspicion, and a careful examination will reveal some localised tenderness on deep palpation, and by this time the pulse rate is rising.

3. **PERITONITIS.**—The pain returns and slowly increases in intensity. The patient's general condition deteriorates quickly, the face is shrunk, drawn and pale, and the pulse rate and temperature are rising steadily. Examination reveals tenderness and rigidity, particularly above the umbilicus, and later paralytic ileus sets in and abdominal distension and persistent vomiting become marked features of the case.

*Prognosis.*—The recovery rate depends on the time which is allowed to elapse between perforation and suture, on the size of the perforation and the degree of soiling of the peritoneum. Not only does the mortality depend on these factors, but so does the smoothness of the convalescence. The aim of the medical attendant is to arrange for operative treatment at the earliest opportunity.

*Treatment.*—This consists in a midline or right paramedian incision above the umbilicus, the identification of the ulcer, its closure by two rows of Lembert's sutures and the careful cleansing of the peritoneal cavity. The necessity for drainage depends entirely upon the degree of peritoneal soiling, and not on the time since perforation. If the opening is large and the stomach full at the time, extensive soiling occurs at once, but a pinhole opening may need no drainage after twelve hours. In the presence of grave soiling a tube is placed in Douglas' pouch through a stab incision above the pubes and possibly a drain to the ulcer area. An occasional ulcer will be encountered whose walls are so indurated and so surrounded by oedema that closure is impossible. It will be treated by putting a tube down to the opening and either stitching omentum over or packing gauze around it to localise the leakage. A gastrojejunostomy should never be performed, unless the closure of the perforation has occluded the pylorus, and this occurs in less than 0.5 per cent. of cases.

A great many ulcers heal after perforation, but it must be recognised that operation is only a preliminary—however essential—to medical treatment. The permanent cures would be increased in number if this were more generally carried out.

**Subacute Perforation.**—The "leaking" ulcer is one in which the



perforation is small, and only a small quantity of gastric contents escapes. The symptoms are less severe than in the acute cases and they clear up within a few hours. In this case the ulcer has become sealed off by a plug of omentum or by adhesions, and there are no further symptoms, but after some hours further leakage and another attack of pain may occur. There is no general peritoneal involvement, and the signs are limited to the epigastrium. In some cases a local abscess may follow. If the condition recurs, the abdomen should be opened and the ulcer closed.

**Hæmorrhage.**—Hæmorrhage from a gastric ulcer may be either :

1. very slight, requiring special tests for its detection ;
2. moderate, with small recurrent amounts insufficient to demand treatment for the actual bleeding ; or
3. severe, being either rapidly fatal or presenting a condition of grave emergency.



FIG. 288

A chronic progressive and penetrating ulcer showing the pancreas in its base together with the splenic artery which has been eroded and has ruptured.

The first type is present in most active ulcers and has no special significance ; the second indicates that treatment is urgently needed for the ulcer rather than for the hæmorrhage, and in the third group the severity of the bleeding outweighs all other considerations. Severe hæmatemesis occurs in many conditions other than ulcer, *e.g.*, in certain grave anæmias, in cirrhosis of the liver and in toxic and septic states. As a complication of peptic ulceration it is seen in two different types of

patient, the anæmic young women with one or many acute erosions or ulcers, and the patient, usually male, with a chronic progressive ulcer. In these people the ulceration exposes a vessel, thins and softens its wall so that a small aneurysm forms and finally bursts (Fig. 288). If the vessel affected is the splenic artery or another of comparable size, a fatal result is a matter of minutes only ; but in the smaller arteries and veins bleeding continues until the fall of blood pressure allows a clot to form and seal the opening. A further hæmorrhage may occur when the blood pressure rises to normal again, and this second hæmorrhage may prove fatal.

*Symptoms* are a severe hæmatemesis and collapse.

*Treatment* is either expectant or operative ; in either case our outlook has changed in the past five years. When due to acute erosion in young women, bleeding is rarely fatal and is permanently cured by



medical treatment, but in chronic ulcer it is an indication that operation will be required in the near future.

**IMMEDIATE TREATMENT** consists in absolute rest in bed, preferably in a darkened room. The foot of the bed is raised on blocks, a radiant neat cradle is placed over the patient and an hypodermic injection of morphia ( $\frac{1}{2}$  gr.) and atropine ( $\frac{1}{100}$  gr.) given. No feeding by mouth is allowed for twenty-four hours, but small pieces of ice may be sucked. Blood transfusion in these patients is still a matter of controversy, but it can do nothing but good if given by the constant drip method. A rapid full volume transfusion is absolutely contraindicated.

**SUBSEQUENT TREATMENT.**—Until recently a severely restricted fluid diet was considered essential. Meulengracht's technique, however, has entirely revolutionised treatment. He advises giving an abundant diet of puree or sieved foods of great variety and high calorific value, which is started within twenty-four hours and continued for at least a fortnight. Witt's modification to suit the people of this country has received almost universal support.

**OPERATIVE TREATMENT.**—Immediate operation recommended by Finsterer of Vienna is not accepted in this country. Operation must follow for every chronic ulcer after successful medical care has tided a patient over the emergency; it can then be performed at leisure and without undue anxiety. Should a second hæmorrhage occur during treatment, a surgeon must be called without delay.

Experience has shown that a partial gastrectomy after severe hæmorrhage with a hæmoglobin count as low as 35 to 40 per cent. is not so hazardous as might be imagined. The decision to operate being made, a constant drip blood transfusion is started immediately and continued throughout the operation and for twenty-four hours afterwards. The results of any less radical procedure are not satisfactory.

**Pyloric Stenosis.**—This may be due to peptic ulcer or carcinoma. When associated with ulcer it is due to cicatricial contracture following healing, and only rarely to spasm due to an active recurrence in an already partly contracted pylorus. The stomach becomes hypertrophied and dilated to enormous size. The symptoms are those of an old long-standing ulcer with or without recent dyspepsia, vomiting of large quantities of partially digested and evil-smelling food, rapid loss of weight and constipation. The distended stomach can easily be mapped out by percussion, visible peristalsis will probably be present and an X-ray photograph shows an enormous stomach and a prolonged delay in emptying.

**Treatment.**—If the ulcer has completely healed, a posterior gastro-jejuno-stomy will achieve a permanent cure, but if active ulceration is present, or if there is any suggestion of a carcinomatous change, a partial gastrectomy should be performed.

**Hour-glass Stomach.**—The fibrosis in this case is in the body of the stomach and results in the formation of two pouches with a narrow connecting passage (Fig. 289). Over 80 per cent. occur in women and follow a saddle-shaped ulcer of the lesser curvature which spreads down on both surfaces. When these patients come for advice

the ulceration is soundly healed in a certain number. The symptoms are those of the old ulcer and vomiting, but the signs are obscure. The X-ray photograph, however, is conclusive (Fig. 290).

There are several operations recommended, viz. :

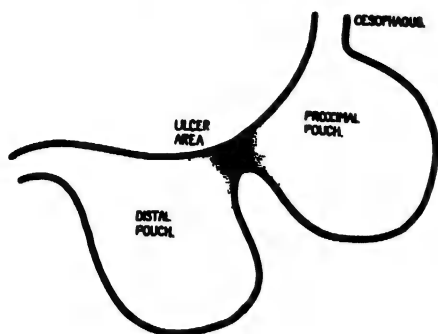


FIG. 289

A diagrammatic sketch of an hour-glass stomach.

1. Gastrogastrostomy, which is an anastomosis between the pouches.
2. Gastrojejunostomy into the proximal pouch.
3. Gastrojejunostomy into both pouches.
4. Sleeve-resection.
5. Partial gastrectomy.

The ideal treatment is a partial gastrectomy. If the ulcer is un-

doubtedly healed, a gastrogastrostomy with a wide stoma will suffice.

**Carcinomatous Change in a Gastric Ulcer.**—A radical change has come over pathological and surgical opinion on this subject since 1920, up to which time it was accepted that carcinoma commonly arose in chronic progressive ulcers. A great deal of work, notably by English pathologists, has shown that this view is exaggerated; nevertheless, a very large number of ulcers are now being removed by partial gastrectomy and examined histologically, and it is firmly established that carcinoma does arise in a chronic gastric ulcer, though not as frequently as was previously believed. In England 5 per cent. is sometimes stated as a reasonable figure; in truth 0.5 per cent. is probably nearer the mark.

### THE UNCOMPLICATED DUODENAL ULCER

The etiology and pathology of peptic ulceration in general has already been discussed, but duodenal ulcers present a few minor differences which will be described here. Both acute and chronic ulcers are found.

**Acute Duodenal Ulcer.**—The frequency with which these occur



FIG. 290

An X-ray of a barium meal showing an hour-glass stomach. The proximal and distal pouches are well shown, as is a very large lesser curvature ulcer.

is impossible to estimate. They rarely lead to a fatal result and never call for operative treatment, so that there is little material for examination. Such post-mortem statistics as are available suggest that the acute ulcer is less common than that in the stomach, but there is reason to believe that the truth lies in the opposite direction. It has been customary to dismiss as "hyperchlorhydria" those cases in which symptoms are suggestive of, but less severe than, chronic progressive duodenal ulceration, and it is precisely this type of patient which probably has an acute ulcer incapable of exact diagnosis. Acute duodenal ulcers are multiple in 50 per cent. of cases and coexist with acute gastric ulcers in 25 per cent. They are met with most commonly in men between 20 and 35 years of age, but may occur in quite young children. There is considerable clinical evidence that acute sepsis and duodenal ulcers have an etiological relationship, and their occurrence as a complication of burns is an example of this. In appearance the acute duodenal ulcer is exactly similar to that in the stomach.

*Symptoms* differ from the typical picture of the chronic duodenal ulcer only in the duration of the attacks and of the intervals of freedom, and this difference is so small that it is not always possible on clinical grounds to be sure that an ulcer is still definitely acute. They occur in young men at times of hard mental work without physical exercise, or in men working at high pressure under considerable nervous or emotional stress. A day's relaxation playing golf may cut short an attack and a week's holiday may affect a permanent cure. The pain lasts but a day or two, is relieved by food, alkalis and rest and is followed by a short interval of freedom. If these attacks should be allowed to continue, the ulcer inevitably becomes chronic.

#### CHRONIC PROGRESSIVE DUODENAL ULCER

These are more common than chronic gastric ulcers, more frequent in men (8 : 1) between the ages of 20 and 45 years. They affect all classes of the community, but more especially the educated professional class, whose work is both arduous and responsible. The first part of the duodenum is the site of 95 per cent., the remainder being in that section of the second part proximal to the ampulla of Vater. The majority occur in the anterior wall and are rarely multiple except in the case of "contact ulcers" (about 10 per cent.), one of which has probably been the cause of the other on the opposite wall. The appearance is very similar to that of the chronic gastric ulcers and the histology is identical. Fibrosis is always present, but tends to be less extensive.

*Symptoms.*—The uncomplicated chronic duodenal ulcer gives so typical a picture that in the great majority a diagnosis can be confidently made on clinical grounds alone. When seen for the first time, most patients give a long history beginning with a story of acute ulceration, *i.e.*, short attacks during a period of overwork or strain. After six months or a year the chronic condition is established, and the attacks last for three or four weeks and the free intervals for any period up to, or even exceeding, one year. This periodicity is even

more exact and more characteristic than in the chronic gastric ulcer. Some patients notice that the attacks recur in certain seasons of the year, and attribute them to sudden changes in temperature.

During the attack, pain is the chief and often the only symptom. It varies from being severe to a dull burning ache or to a curious feeling of discomfort combined with depression. It is felt in the epigastrium, sometimes on the right side, and may be referred to the back or to the right iliac fossa. It is described as coming on either three hours after a meal or some time before the next, and is often designated "hunger pain." Owing to the shorter intervals between lunch and tea and between tea and dinner the pain may be felt chiefly before luncheon and some hours after dinner, and it is characteristic that patients are wakened up by it about 2 A.M. The pain is immediately removed by food, and many patients quickly learn to have a glass of milk and biscuits beside them on going to sleep, and to have something in the middle of the morning. Vomiting is seldom seen unless there is obstruction. The appetite is not diminished, but rather is it increased, and for this reason there is no loss of weight. Constipation may be present and many patients complain of an unpleasant taste in the mouth. During the free intervals there is complete absence of all symptoms and patients comment on their fitness. Hæmorrhage in the form of occult blood in the stools is present in every duodenal ulcer, but a large hæmorrhage appearing as a hæmatemesis or as a large melæna stool is not common, and should be regarded as a complication and not as a symptom.

*Examination.*—Palpation will reveal localised tenderness over the position of the first part of the duodenum, and there may be sectional rigidity of the upper right rectus muscle. The fractional test meal shows increase in free HCl and total acidity in over 75 per cent. of cases, and is of more useful diagnostic significance than in gastric ulcer. A barium meal will usually clinch the diagnosis, a persistent deformity of the duodenal cap being direct evidence.

*Treatment.*—Medical treatment results in a rapid and permanent cure of the acute ulcers, and while the chronic ulcer remains free of complications it will hold out a fair prospect of cure, but relapses are more frequent than in gastric ulcer. The indications for operation are :

1. Relapse after efficient medical treatment.
2. Any complication.
3. If the patient has had a history of three years and over.

Operative treatment includes two alternatives, either a posterior gastrojejunostomy or some form of partial gastrectomy. The results of the former are so poor and fraught with so many serious after-effects that few surgeons practise it to-day. A partial gastrectomy would appear to be a very drastic procedure for a duodenal lesion, but it must be remembered that any operation is probably useless unless it removes such a portion of the acid-secreting mucous membrane as will permanently relieve the patient of any risk of hyperchlorhydria. It cannot be denied that the treatment of duodenal ulcer is still giving

rise to doubt and anxiety among those physicians and surgeons who think deeply of the underlying problems. Suffice it to say that at the time of writing no operation should be advised until there is sound evidence that the ulcer is no longer an uncomplicated one.

### THE COMPLICATIONS OF DUODENAL ULCER

These are :

1. Penetration.
2. Perforation.
3. Hæmorrhage.
4. Duodenopyloric stenosis.

**Penetration** occurs less frequently than in gastric ulcer, but ulcers on the posterior and inferior walls of the first part and on the internal wall of the second part erode the pancreas. Neglected ulcers of the anterior and superior walls may penetrate the liver. In such cases the characteristic intervals of freedom disappear from the history and hunger pain becomes permanently established. Simple gastrojejunostomy will fail to cure this condition, and more radical treatment is needed. It is fortunate that more accurate diagnosis and earlier operation have reduced the incidence of this complication very considerably (Fig. 291).

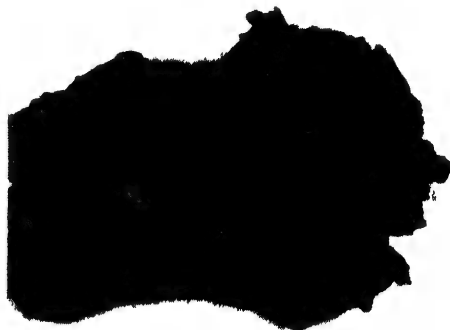


FIG. 291

An unusually large duodenal ulcer penetrating the pancreas.

**Perforation.**—The duodenal ulcers which perforate are always chronic ones in a stage of acute recrudescence, and they are situated on the anterior surface close to the pylorus. The clinical picture is almost identical with that of the perforated gastric ulcer, the same stages of peritonism, apparent recovery and peritonitis being present. The previous history of indigestion will be different, and the tenderness and rigidity more strictly limited to the right side of the abdomen. Treatment consists in immediate laparotomy, identification and suture of the ulcer, a proper peritoneal toilet and closure of the wound with or without drainage, as each individual case demands. A gastrojejunostomy should *not* be performed.

**Hæmorrhage.**—Bleeding is usually a late complication. It is true that some patients maintain that they have had no previous symptoms, although they will probably have had a vague discomfort to which they have paid no attention, but as a general rule there is a typical long-standing history. Bleeding varies from a slight ooze, which can be detected only by tests for occult blood in the stools, to a profuse hæmorrhage endangering life. The large amounts are usually passed on into the bowel and appear as melæna, but occasionally there will be a hæmatemesis. The following case illustrates the clinical picture.

A senior official in a big office, having charge of rooms on three floors of the building, was accustomed to run up the stairs three times each morning as a form of exercise. One morning he reached the top stair on the first occasion out of breath and feeling shaky, on the second occasion he could only walk and arrived at the top feeling faint, and on the third occasion he sank into a chair on the first floor exhausted. He returned to his home, where a few hours later he passed a stool containing a profuse amount of blood.

Bleeding in a duodenal ulcer is of the most serious significance, large arteries in contact with the thin-walled duodenum being easily accessible to the ulcer. The immediate emergency is met by the same treatment as for hæmorrhage from a gastric ulcer, *i.e.*, absolute rest and medical measures. Duodenal bleeding is an indication that an operation for the ulcer has become a matter of some urgency. The patient's general condition and resistance should be improved as quickly as possible to render him fit to withstand operation. A partial gastrectomy should be performed. If a second hæmorrhage should occur, an immediate operation and a blood transfusion are carried out.

**Duodenopyloric Stenosis.**—The fibrosis following an ulcer of the duodenum is usually so closely adjacent to the pylorus that pyloric stenosis follows, but in some cases the contraction is purely duodenal. The symptoms and X-ray findings are similar to those cases following gastric ulcer. The treatment is a simple gastrojejunostomy.

### GASTROJEJUNAL AND JEJUNAL ULCERS

**Etiology.**—Gastrojejunostomy for peptic ulceration of the stomach and duodenum is sometimes followed by ulceration on the line of the anastomosis or in the jejunum within 2 in. of that line. It is never seen if the operation has been performed for carcinoma of the stomach, and occurs very rarely in the suture line of a partial gastrectomy. It was said to occur in 2 per cent. of all cases, but at the present time that figure is unquestionably too high. It is more common in men, presumably because duodenal ulcer is predominantly a male disease. The etiological factors underlying peptic ulceration in general are at work in these conditions, but the operation introduces additional factors which may be of significance. Unabsorbable suture material was often found at secondary operations, and is thought to cause irritation of the mucous membrane. Walton has suggested that it is easy to fail to obtain coaptation of the mucous membrane at the angle of the anastomosis which is last sutured, and this leaves a gap in the mucosa which is accessible to attack by the gastric juices.

**Pathology.**—The gastrojejunal or anastomotic ulcer involves a varying extent of the junction, and is about  $\frac{1}{4}$  to  $\frac{1}{2}$  in. wide. It is deeply excavated, and its histology is typical of peptic ulceration. The induration of the base is most marked and spreads into the transverse mesocolon, as a result of which the transverse colon is drawn down towards the anastomosis. If ulceration continues, a fistula may be formed between the stomach and colon. Jejunal ulcers are identical in naked-eye and microscopic appearances with

the gastric and duodenal ones. They are usually close to the anastomosis and in the efferent loop.

*Symptoms and Signs.*—A period of six months to two years usually elapses after operation. The picture suggests in some features duodenal and in others gastric ulceration. The pain comes on two or three hours after food. Vomiting is claimed by Walton to be a frequent and characteristic symptom, but this is denied by others. If present, it relieves the pain. The position of the pain is typical, being low down near the umbilicus beneath the left rectus muscle, and is referred downwards and outwards to the left iliac fossa.

On examination there will be marked tenderness just above and to the left of the umbilicus, that sector of the left rectus muscle may be rigid, and a tumour palpable. A barium meal will show that the stomach empties rapidly, and in skilled hands the ulcer may be demonstrated.

*Treatment.*—This is essentially operative, medical treatment being reserved for those patients whose general condition does not permit of an extensive operation. The procedure will entail a resection of the anastomosis and a partial gastrectomy. If a gastrocolic fistula is present the colon must be dissected free and sutured.

*Complications.*—These ulcers may penetrate into the colon or perforate acutely into the peritoneal cavity. The symptoms of the perforation are typical, but the tenderness and rigidity are below the shelf of the transverse mesocolon, and so leave the upper segments of the recti muscles less affected than in perforation of gastric and duodenal ulcers. Treatment consists in exposure and suture of the opening. The symptoms of a gastrocolic fistula are described on p. 589.

### OTHER DISEASES OF THE DUODENUM

**Duodenal Diverticula** are very rare, affecting either the second part around the ampulla or the third part. They give rise either to vague dyspeptic symptoms or to pressure on neighbouring structures, *e.g.*, the common bile duct. They are diagnosed only by X-rays (Fig. 292), and their treatment consists in removal, which may prove a difficult and hazardous operation.

**Chronic Duodenal Ileus.**—Wilkie drew the attention of British surgeons to a small group of cases in which the duodenum is greatly distended and in which attacks of vague epigastric discomfort and vomiting formed the clinical picture. In this country it is still regarded as uncommon, but the *Lancet* has commented on the apparently high incidence in France. The author has personal knowledge of eleven cases, in each of which the findings closely correspond to Wilkie's description. The case history of one patient illustrates the clinical picture.

A lady of 45 years, highly intelligent and far removed from the abdominal neurotic, had suffered for many years with periodic attacks of epigastric discomfort and persistent vomiting lasting for ten to fourteen days. She was perfectly well during the intervals, but during the attacks she was definitely ill and lost weight rapidly owing to the vomiting. She had had a gastro-



jejunostomy performed, and this had made her worse ; it had been undone and the stomach and jejunum sewn up. Later still she had her gall-bladder removed. All these procedures left her with the same recurrent attacks, and she was being diagnosed as a neurotic. The diagnosis was made by X-rays during an attack.

The distension of the duodenum usually ends at the point where the superior mesenteric vessels cross the third part, and the dilatation may affect the stomach, but it has been described as including the upper part of the jejunum. In the

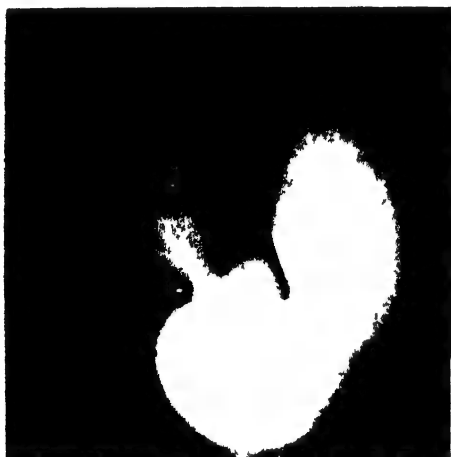


FIG. 292

Duodenal diverticulum.

author's cases it was so marked that the duodenum ballooned up towards the abdominal incision, but it stopped short at the superior mesenteric vessels, and the stomach was not noticeably affected. The possibility of this condition should be considered more frequently in cases of unexplained attacks of dyspepsia and vomiting. A barium meal examination is more likely to give positive information if carried out during an attack. The treatment consists in a duodenojejunostomy, *i.e.*, an anastomosis between the dilated third part of the duodenum and the jejunum as

near as is convenient to the duodenojejunal junction. It is characteristic of this condition that a gastrojejunostomy fails to relieve it.

**Growths of the Duodenum.**—A primary carcinoma of the duodenum is very rare. Duodenal ulcers never become malignant and pyloric carcinoma fails to invade the duodenum. There is described a columnar-celled carcinoma of the second part, but this probably originates in the ampulla of Vater. It causes obstruction either to the duodenum or to the common bile duct. One or two cases have been described recently of ring strictures of the third part being due to scirrhus growths. The author has recently removed successfully a ring carcinoma of the third part of the duodenum, but it is unusual that these patients are diagnosed at a sufficiently early stage for so happy a result.

R. M. HANDFIELD-JONES.



## CHAPTER XXIX

### THE SMALL AND LARGE INTESTINE

**T**HE surgical anatomy of the stomach and duodenum is described in Chap. XXVIII, and of the rectum in Chap. XXXI. This section deals with the small intestine, cæcum and colon.

*Development.*—From the embryonic mid-gut is derived that part of the intestinal canal between the ampulla of Vater and the splenic flexure of the colon, the remaining segment of the large intestine arising from the hind-gut. During foetal life the development of the complex intestinal canal from a single loop with its median mesentery takes place in the amniotic sac. As its growth proceeds, the intestine is withdrawn into the abdomen, and by a process of rotation the normal position of the small and large intestine is obtained. Certain rare malformations in the abdomen are due to this rotation having failed to occur completely. The ileum in foetal life is connected to the yolk-sac by the vitelline or omphalomesenteric duct, which should eventually disappear. Its persistence gives rise to the many variations of Meckel's diverticulum.

*Surgical Anatomy.*—The small intestine extends from the duodenojejunal junction to the ileocæcal valve, and varies between 22 and 24 ft. in length. Its coils are suspended by the mesentery and are freely movable. The large intestine begins with the mobile cæcum, to which is attached the appendix, and then has alternately fixed and mobile sections. The ascending and descending colon lie behind the peritoneum of the posterior abdominal wall, while the transverse and sigmoid colon have each a well-marked mesocolon. The large bowel is easily distinguished from the small gut by its sacculation, appendices epiploicæ and three longitudinal muscle bundles. The jejunum and the ileum have certain differences, which should make their differentiation reasonably easy; in the former the mesenteric arteries have only one arcade of anastomosis, and the fat does not reach the border of the intestine, so that peritoneal "windows" may be seen, whereas in the latter there are three, four or even five vascular arcades, and the mesenteric fat not only reaches but encroaches on the border of the bowel.

*Vascular Supply and Lymphatic Drainage.*—The superior mesenteric artery, being the artery of the midgut, supplies the whole small intestine from the ampulla of Vater onwards, the cæcum, appendix and colon as far as the neighbourhood of the splenic flexure. The jejunum and ileum are supplied by the vasa intestinae tenuis; the ileocolic artery dividing into ileocæcal, appendicular and right colic branches gives blood to the last 6 in. of the ileum, cæcum, appendix and ascending colon as far as the hepatic flexure, whilst the middle colic artery supplies the transverse colon and splenic flexure. Corresponding veins drain blood into the main superior mesenteric vein. The inferior mesenteric artery, the vessel of the hind-gut, supplies the remainder of the colon through its left colic, sigmoid and superior hæmorrhoidal branches, the venous blood returning to the inferior mesenteric vein. The lymphatic drainage of the small intestine is by the lacteal vessels into the receptaculum chyli via the glands of the mesentery.

The arrangement of glands draining the colon is always constant. Along the concavity or mesocolic border are the paracolic glands, both small and numerous : grouped around the main branches of the two mesenteric vessels are sets of intermediate colic glands, and finally at the origin of the main vessels from the aorta is placed the main or central colic group of glands.

*Methods of Examination.*—The clinical examination of the abdomen combined with the accurate taking of case histories is of the utmost importance, and no investigation is complete until a rectal, and in the female a vaginal, examination has been made. The fæces are examined for the presence of blood, obvious and occult, pus, mucus, blood, bile pigments, organisms and parasites.

Barium meals, barium enemata and radiographic examinations yield important results, and finally the sigmoid colon can be inspected visually by means of the sigmoidoscope.

### CONGENITAL ANOMALIES

**Absence and Atresia.**—Absence of any part of the intestinal canal is confined to the condition known as Imperforate Anus (Chap. XXXI. p. 647). Congenital narrowing may occur at the pylorus, in the duodenum, small and large intestine, either in the form of septa or short lengths of narrowed and indistensible bowel.



FIG. 293

A Meckel's diverticulum.

**Exomphalos.** — Very rarely new-born babes are found to have an incomplete fusion of the abdominal wall in the neighbourhood of the umbilicus, at the site of which is seen a circular gap, to the edges of which is attached a thin transparent membrane leading to the umbilical cord. A bell-shaped sac is thus formed in which can be seen coils of intestine not yet withdrawn into the abdominal cavity.

The *treatment* is immediate removal of the sac and closure of the gap in the abdominal wall.

**Meckel's Diverticulum.** — The persistence of the omphalo-mesenteric duct may lead to a variety of congenital anomalies, the commonest of which is known as Meckel's diverticulum. This consists of a blind tubular process arising from the antimesenteric border of the ileum about 39 in. from the ileocaecal valve in the adult (Fig. 293). It has no mesentery, and its blood supply is derived from the gut wall. It is usually about 2 or 3 in. long, and its apex may or may not be attached to the umbilicus by a fibrous cord.

If the duct persists in its whole length, a fistula will open on the abdominal wall at the umbilicus ; this, however, is exceedingly rare.

The presence of Meckel's diverticulum is not necessarily of any clinical significance, but it may be the seat of acute inflammation, it may form the apex of an enteric intussusception or may be directly or indirectly the cause of acute intestinal obstruction. Should it occupy the sac of the hernia, the special name of Littré's hernia is applied to it.

**Congenital Idiopathic Dilatation of the Colon** (Hirschsprung's Disease).—This rare condition occurs in children, more frequently in boys than girls. Its cause is not precisely known, but it is now believed to be due to spasm of muscles at the rectosigmoid junction resulting from an imperfect functioning of the sympathetic nervous system. As the accompanying X-ray photograph (Fig. 294) shows, the colon, particularly the sigmoid and descending colon, is enormously distended, and the power of voluntary defæcation is lost. Aperients have no effect and the gut can be emptied only by enemata, and in the more advanced examples even they may fail to achieve any result. The abdomen becomes greatly distended but remains soft, and the passage of a flatus tube allows large quantities of foul-smelling gas to escape with temporary subsidence of the distension. The majority of children die before the age of 12 years from toxæmia or peritonitis, but a certain number grow up into adult life with comparatively little deterioration in general health.



FIG. 294

An X-ray of a barium enema showing the enormously dilated colon in Hirschsprung's disease.

*Treatment* of recent years has been directed to the sympathetic system. Telford has shown that spinal anæsthesia reaching to the anterior root of the 6th dorsal nerve achieves a complete and lasting cure. No explanation is offered for this amazing result, but Telford emphasises that it must be given in the early stage in childhood. A personal communication from him relates to thirteen consecutive successes.

#### ERRORS IN FUNCTION

**Fæcal Impaction** may very rarely occur as a result of prolonged unrelieved constipation and will eventually culminate in so complete a block that acute intestinal obstruction will follow. A large mass of fæces collects in the sigmoid colon and upper part of the rectum,

and as fluid is absorbed, it becomes more solid and hard. The patient, usually a middle-aged woman, complains of colicky pain, distension, nausea and constipation for many days. Examination reveals a hard mass low down in the left side of the abdomen, and a finger in the rectum reveals the faecal nature of the tumour.

*Treatment* consists in the injection of hot glycerin or olive oil enemata, followed an hour later by a large, simple enema. If this fails to produce a result, the mass must be broken up into small pieces by the finger or a spoon under general anaesthesia.

A similar condition may occur in the caecum, but obstruction is not so likely to occur, and the soft putty-like mass can usually be made to pass on by the use of high colonic lavage combined with oft-repeated small doses of Epsom salts.

**Intestinal Stasis.**—Hurst has recently suggested that too much unwarranted importance is attached to chronic constipation and the supposed ill-effects which may arise from it; this is infinitely more true of intestinal stasis. Twenty years ago this was a fashionable complaint, and many diseases, even chronic mastitis of the female breast, were said to be due to toxæmia derived from the putrefaction of stagnant faecal material. Peritoneal bands and adhesions were described and named (*e.g.*, Lane's first and last kinks) and extensive operations for removing the whole or part of the colon practised. At the present time intestinal stasis is not regarded as a clinical entity, and it should be clearly understood that the treatment of constipation is purely medical and that resection of the colon, short-circuiting procedures or the division of adhesions is no longer considered justifiable.

**Visceroptosis** (Glénard's Disease).—It has been shown that the human thorax and abdomen are susceptible of arrangement in several normal types, in one of which the costal margin is unduly long, the epigastric angle unusually acute and the space between the iliac crest and the last rib very narrow. This type predisposes to general visceroptosis. Pregnancy, prolonged illness, sedentary occupation and lack of muscular exercise all play their part in the slackening of the peritoneal mesenteries and ligaments and the gradual sagging of all or certain viscera. Such ptosis is compatible with normal health and does not necessarily produce symptoms, but it may tend to constipation and slight abdominal discomfort. In patients with a neurasthenic background, these mild symptoms may become a fixed obsession of the presence of malignant disease, and the danger exists that a diagnosis of peptic ulceration, cholecystitis, renal disease or intestinal carcinoma may be made. Still more frequently chronic appendicitis is suggested as the cause, and the absence of improvement after appendicectomy leads to an aggravation of the neurosis.

*Treatment.*—This condition is a medical and psychological problem. Many operations for suspension or plication of the mesentery, the mesocolon or the bowel itself have enjoyed a brief popularity only to fall into disfavour, and it must be recognised that surgery has no part in this condition. The use of abdominal belts is also open to criticism. The object of treatment is to strengthen the abdominal

muscles by massage, exercises and faradic stimulation, so that they can support the abdominal contents unaided. A belt merely increases the muscular atony, and should be ordered only for those patients in whom active restoration treatment is contraindicated.

## INFLAMMATORY DISEASES OF THE INTESTINES

### ENTERITIS

Inflammation of the mucous membrane of the small intestine occurs in both children and adults from either irritation, food-poisoning or bacterial invasion. It is therefore of medical interest, except in so far as it follows strangulation of the intestine or the improper use of drainage tubes, as a result of which a fæcal fistula may develop. The symptoms of enteritis are discomfort or griping pain in the abdomen and diarrhoea.

*Treatment* consists in removal of the irritant factor by thorough purgation with castor oil, followed by a bland diet and sedative drugs such as bismuth and opium.

### COLITIS

Colitis also comes under the care of the physician rather than the surgeon, but in some of its more severe manifestations surgery is invoked to enable medical treatment to be given to better advantage. There are two varieties, namely, the mucous or mucomembranous and the ulcerative.

**Mucomembranous Colitis** is characterised by constipation and the passage of mucus in the stools. There is no real evidence of a true inflammatory process in the mucous membrane of the colon as there is in the ulcerative form. It affects women rather than men and particularly those of good social and financial position, in whom there is a marked neurotic element. The symptoms are mild abdominal pain, obstinate constipation and the passage of mucus, which will appear either as a shapeless lump or in a long strip suggestive in appearance of a tapeworm, and in some severe cases an almost complete cast of the colon may be voided.

*Treatment* is medical.

**Ulcerative Colitis** is a much more serious condition. Although no causal organism has been isolated, the pathology suggests a relationship with that group of organisms concerned with bacillary dysentery. Ulcers form in the mucous membrane and may coalesce so that large areas of the bowel are denuded of their lining membrane, but the muscle is unaffected and perforation does not occur (Fig. 295).

*Symptoms.*—After a gradual onset, diarrhoea appears as the most prominent symptom and is characterised by the passage of blood, mucus and pus. In the severe cases there is a mild pyrexia, and the patient may pass as many as twelve motions a day. The loss of blood may be such that a secondary anæmia results. The disease is most intractable, and is subject to remissions and exacerbations.

*Diagnosis* is confirmed by the bright-red ulcerated mucous membrane as seen through the sigmoidoscope. Carcinoma and specific forms of colitis such as amœbic and bacillary dysentery must be excluded before treatment is commenced.

*Treatment* consists in rest in bed, careful dieting and regular colonic lavage daily. Recently sulphapyridine enemata have been used with good results. The injection must be retained for an hour if possible. Polyvalent antidysenteric serum has proved successful in a fair percentage of patients. If medical treatment has failed an appendicostomy should be performed, so that the irrigation can be done from above to ensure that the whole colon is receiving treatment.



FIG. 295

Ulcerative colitis.

manifestations are not common, but a non-suppurative arthritis, particularly in the knee, is a well-recognised complication.

### DYSENTERY

**Amœbic Dysentery** is due to the *entamoeba histolytica*, which affects the colon only, stopping abruptly at the ileocæcal valve. Its clinical picture, diagnosis and treatment should be studied in textbooks of medicine. Its surgical manifestations are local in the bowel itself, including perforation in very acute cases and fibrous stricture as a late sequela, and metastatic in the liver, where an amœbic or solitary abscess may form.

**Bacillary Dysentery** includes several types due to either Shiga's bacillus or one of the five strains of Flexner's bacillus. Its surgical

### TYPHOID

Enteric fever includes typhoid and paratyphoid A and B infections, of which the first is by far the most serious. Both sexes and all ages are liable to the disease, but the years between 10 and 35 are most commonly attacked. It is conveyed by contaminated food, water and milk supplies or by certain solids, watercress having recently gained a sinister reputation.

*Pathology.*—The *B. typhosus* chiefly attacks the ileum, and the Peyer's patches bear the brunt of the infection (Fig. 296). These go through a series of changes, namely inflammation, sloughing, ulceration, granulation and repair. When the sloughs separate, severe hæmorrhage or perforation may occur. The ulcers are elliptical, having their long diameter in the long axis of the intestine. The paratyphoid infections differ only in that they affect the ascending colon as well as the ileum.

*Diagnosis* is made by blood culture and by the Widal and Weil-Felix agglutination tests.

The symptoms and treatment should be studied in textbooks of medicine.

*Surgical Complications.*—1. HÆMORRHAGE of a severe nature may occur, though infrequently, during or at the end of the third week, and may be so copious as to necessitate a transfusion.

2. PERFORATION also occurs at the end of the third week and is usually seen in the more severe cases, though it may affect patients during a recrudescence. The symptoms are a sudden onset of abdominal pain, shock, occasionally a rigor and tenderness and rigidity, especially in the right iliac fossa.

*Treatment* is immediate laparotomy and suture.

3. PHLEBITIS of the femoral vein with thrombosis is the most frequent complication, but does not occur until the patient has safely arrived at the early stage of convalescence. It may result in a permanently swollen leg with an impaired venous circulation.

4. TYPHOID OSTEITIS is not uncommon. It is described in Chap. XLVII.

5. ACUTE SUPPURATIVE PAROTITIS, acute non-suppurative orchitis and otitis media are all rare sequelæ.



FIG. 296

Typhoid ulceration of the ileum showing how the infection affects chiefly the Peyer's patches.

## TUBERCULOUS INFECTION OF THE INTESTINE

Although it is easy to describe the tuberculous affections in the abdomen separately, the clinical picture cannot be arranged so neatly in separate entities, for the intestinal, lymphatic and peritoneal lesions tend to coexist in varying degrees. The intestinal manifestations do sometimes, however, occur without marked involvement either of the peritoneum or the lymph glands.

1. **Tuberculous Ulceration** affects both ileum and colon. The ulcers are frequently multiple and are most numerous in the ileo-cæcal region. They result from the ingestion of tubercle bacilli either in swallowed sputum or infected foodstuffs, especially milk. They exhibit the usual characteristics of the chronic tuberculous ulcer with thin, pale, undermined edges. Starting in a Peyer's patch or lymphoid follicle, they spread round the circumference

of the bowel by following the lymphatic vessels. They are thus placed at right angles to the long axis of the bowel (compare typhoid), and the fibrosis which follows their repair tends to form a fibrous stricture, so that at a later date symptoms of chronic intestinal obstruction may be expected (Figs. 297 and 298).

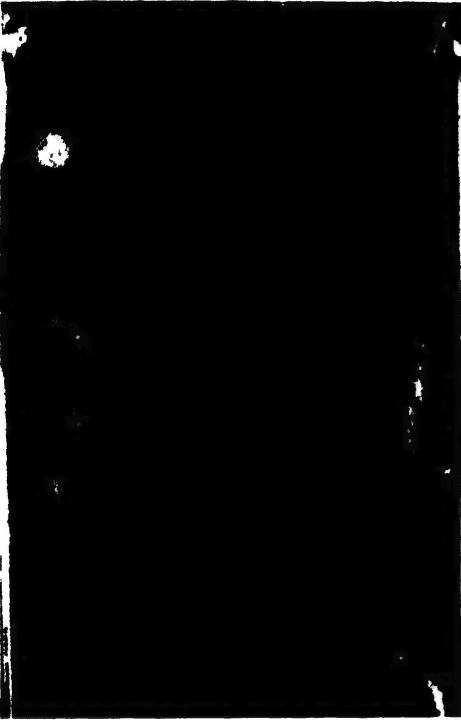


FIG. 297

Tuberculous ulceration of ileum. (Circumferential spread of ulceration is well shown.

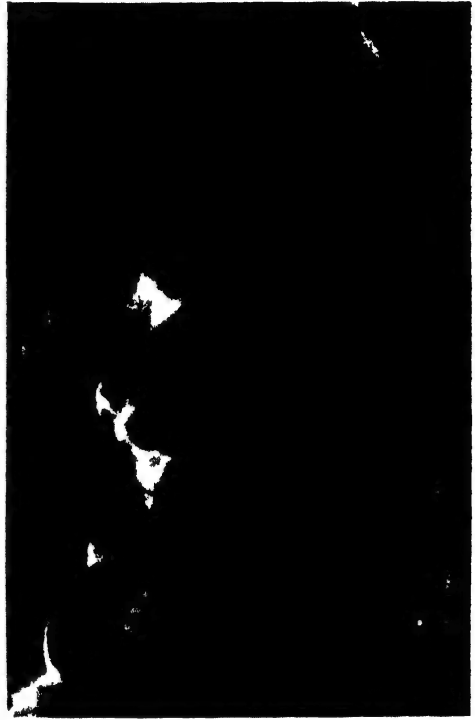


FIG. 298

Tuberculous ulceration of the colon.

*Symptoms.*—The clinical picture is indefinite unless the ulceration is extensive when diarrhoea with loose watery stools occurs without the presence of blood or mucus.

*Treatment* is purely medical until complications arise.

*Complications.*—Perforation with localised abscess formation, which may be either “cold” or secondarily infected with bowel organisms, may be seen, or obstruction from a fibrous stricture or from matting together of contiguous coils may occur, and a faecal fistula would follow the spontaneous rupture of a cold abscess through the abdominal wall. These complications are fortunately rare.

*Treatment* is directed towards the eradication of the infected area, if possible, or to the relief of the obstruction by a short circuit, if adhesions render resection impracticable.

**2. Ileocaecal Tuberculosis** is a rare and curiously specialised manifestation of tuberculosis. It involves the caecum, terminal few



inches of the ileum and the beginning of the ascending colon, and the infection is largely concerned with the submucous coat, in which plane it spreads (Fig. 299). This section of the bowel becomes thickened, indurated and nodular, and an unusual fibrolipomatous deposit is laid down beneath the peritoneum, which is rough and shaggy. In the later stages the mucous membrane consists of hypertrophic masses with occasional ulcers, and eventually chronic obstruction follows the healing fibrosis.

*Symptoms.*—(a) In the introductory stage there is a vague, indefinite history of right-sided pain, indigestion, loss of weight and nausea. Some tenderness may be elicited in the right iliac fossa, and almost inevitably the diagnosis of chronic appendicitis is made. (b) In the intermediate stage, diarrhoea with blood and mucus

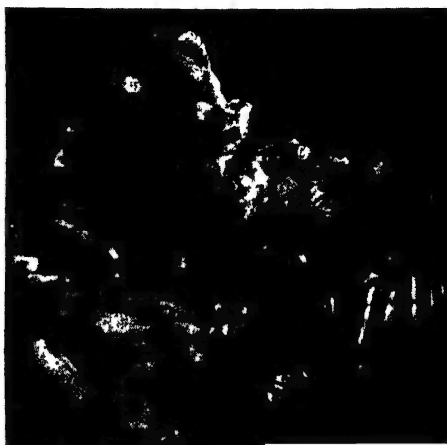


FIG. 299

Ileocaecal tuberculosis. The infection will be seen to be more advanced in the caecum than in the ileum.



FIG. 300

Regional ileitis in which the inflammatory thickening is chiefly in the terminal ileum.

Although originally described as a disease of the terminal ileum, it is also seen in the upper reaches of the small intestine and in the colon.

The bowel is thickened and inflamed in a section between 3 and 6 in. long, the mucous membrane is swollen and ulcerated and small

in the stools alternates with constipation, and on examination a tumour can be felt. The diagnosis of carcinoma of the caecum is certain unless a mild pyrexia and other signs of tuberculosis are present, and inspire a bacteriological examination of the faeces. (c) The final stage is one of an established chronic obstruction which is showing signs of becoming acute.

### REGIONAL ILEITIS

Crohn's disease is an inflammatory condition of the ileum and is now a well-established entity and a large number of cases is on record. It may occur at any time between puberty and old age, but the majority of patients are under 40 years.

localised abscesses may be present in the submucous and muscular coats (Fig. 300). It is characteristic of this disease for an abscess to form at the margin of the gut within the leaves of the mesentery; in a recent case this had subsequently perforated into the other limb of a loop of intestine, thus forming an entero-enteric anastomosis. The pathology of this condition remains unknown.

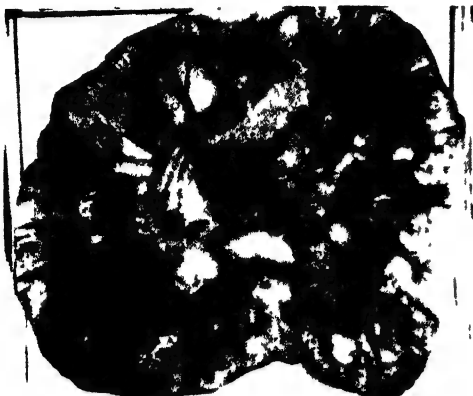


FIG. 301

A coil of ileum with multiple congenital diverticula.

ileum (Fig. 301) and colon. Meckel's diverticulum has been described (p. 614), while the others rarely give symptoms. Acquired diverticulosis of the colon is by no means uncommon and may affect the whole large intestine, but the sacs are always more numerous in (and sometimes confined to) the sigmoid colon. The term diverticulitis is applied to the clinical condition arising from inflammation in the little sacs and is seen chiefly in the sigmoid

Diverticulitis occurs more commonly in men, who are over 40 years of age, fat and habitually constipated. Reference to Fig. 302 will show that at two (possibly three) places in a cross-section of the bowel wall there exists a normally weak spot where the blood vessels penetrate the muscular coat. Chronic constipation leads

to an increased intracolonic pressure, which is greatly increased during the strain of defæcation, and at these times little sacs of mucous membrane may be forced through these weak spots, frequently finding their way into the appendices epiploicæ. In this way a double (or rarely a treble) row of sacs may be found in a length of the colon. They are devoid of muscle fibres in their walls, so that while fæcal material can enter them, it cannot easily find its way

It varies considerably in severity and presents itself clinically in two forms, one closely resembling acute appendicitis and the other producing symptoms of chronic intestinal obstruction.

Treatment is resection of the affected segment of intestine or a short circuit.

### DIVERTICULITIS

(Congenital diverticula are found in the duodenum, jejunum,

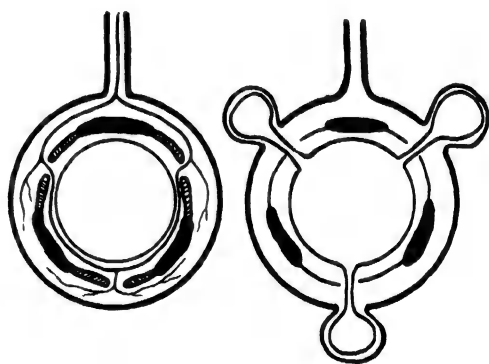


FIG. 302

A diagram of transverse sections of the colon showing the anatomical sites of weakness in the muscle wall and the relation of the diverticular sacs to them.

back into the colon. Impaction of fæces may lead to inflammation, and clinically these patients will seek advice either with symptoms of an acute abdominal emergency or for chronic left-sided pain (Fig. 303).

**Acute Diverticulitis** closely resembles acute appendicitis in the left iliac fossa instead of in the right. The nature of the inflammation, its course and complications are similar to appendicitis in almost every respect. A fat, constipated, middle-aged patient complains of abdominal pain centred around the umbilicus, and may be sick. Later the pain moves to the left iliac fossa, where there will be tenderness and after a time rigidity. Again, just as the appendix may be lying in the pelvis, so may the affected diverticulum be low down in the sigmoid colon near the rectosigmoid junction, in which case the symptoms are those of pelvic peritonitis.

*Treatment.* — The inflamed diverticulum should be removed, but this is rarely possible, and in many patients an abscess will have formed by the time an operation is performed.

In such cases treatment is confined to drainage of the abscess. Convalescence is apt to be retarded by recurrences of pain and pyrexia, and in such cases a temporary colostomy should be performed.

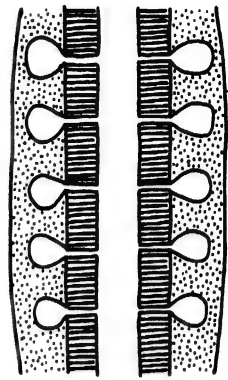


FIG. 303

A diagram of a longitudinal section of the colon with multiple diverticula.

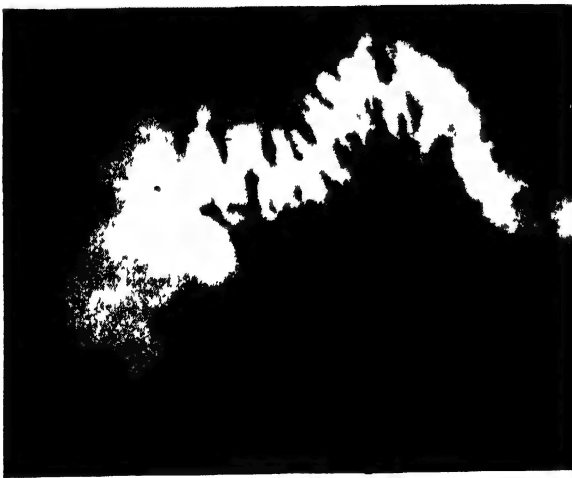


FIG. 304

A barium enema showing the presence of many diverticula in the sigmoid.

existence of a carcinoma of the colon. A bimanual examination reveals a hard and tender swelling in the region of the sigmoid colon, and only a barium enema radiographic investigation can establish a true diagnosis (Figs. 304 and 305).

*Treatment.*—In its early stages no operation is needed, but the

**Chronic Diverticulitis** is produced by a chronic inflammatory process in a row of diverticula, so that a fibrous reaction is laid down in the wall of a section of bowel, which is then thickened and narrowed. These patients will present a vague history of several months duration of slight discomfort, with a tendency to attacks of diarrhoea in which mucus, but not blood, is passed. This is allied to a gradually increasing constipation, which sooner or later must arouse the suspicion of the

condition should be treated on the same lines as mucous colitis. A close watch is kept on the patient, and if signs of chronic obstruction arise, or if the radiographic findings suggest an increase in the size of the infected area or a decrease in its mobility, a resection should be performed if adhesions permit. The operative risks in such cases will be greatly diminished if a preliminary colostomy is performed and the bowel drained for six to eight weeks.

**COMPLICATIONS OF DIVERTICULITIS.**—1. **Acute Perforation.**—The author has recently seen a lady who was undergoing treatment in a clinic to reduce her weight. While seated on the lavatory seat and straining, she was suddenly seized with such violent abdominal pain

that she fainted. Later a pelvic peritonitis developed and led to the formation of a large pelvic abscess. A diverticulum had given way suddenly as the direct result of violent straining.

*Treatment* consists in drainage and repair of the perforation.

## 2. **Fistula Formation.**—

Chronic diverticulitis with mild attacks of perisigmoiditis may lead to adhesions to neighbouring structures, the most important of which is the bladder. A slowly progressive penetrating ulceration continues until the bladder is opened and a vesico-colic fistula results (Chap. XXXVI). Irritability of the bladder combined with the passage of bubbles of gas in the



FIG. 305

An X-ray illustrating the persistence of the barium in the little pockets after an evacuation of the lower bowel.

urine may be the first indication of the diverticulitis.

*Treatment* is difficult but exceedingly important. The slightest suspicion of bladder involvement demands operation, because it is easier to relieve the patient before the fistula has become established. In such cases the bladder must be carefully dissected free and the damaged colon removed if possible, failing which a colostomy is performed to prevent further inflammation and to allow subsidence of that already present. Six months later radiography may reveal so marked an improvement that a resection of the colon may be planned, followed by closure of the colostomy.

If a fistula is present, the infection of the urinary tract is of paramount importance, and all further contaminations of the bladder must be prevented by diverting the faeces by means of a colostomy. At a later date it may be possible to free and repair the bladder.

## FÆCAL FISTULA

A purposely designed colostomy and appendicostomy are examples of intestinal fistulae, but they are not included in the clinical definition

of a fæcal fistula, which term implies that the fistulous communication is the result of a congenital defect, disease or injury. These various causes may be classified as follows :—

1. Congenital fistulæ seen at the umbilicus as the result of a persistent omphalo-mesenteric duct.
2. Traumatic : (a) penetrating wounds in military or civilian practice ; (b) operative factors, including the ill-judged retention of drainage tubes and the bursting open of an abdominal incision as the result of sepsis with the involvement of a coil of intestine.
3. Necrosis of the bowel wall following acute gangrenous appendicitis, acute perforative diverticulitis, an unrecognised strangulation of a coil of intestine or the presence of foreign bodies such as retained swabs, etc.
4. Specific causes such as tuberculosis, actinomycosis and carcinoma.
5. The non-recognition of a distal obstruction during operation for appendicectomy, or intestinal resection and anastomosis.

The fistula has either a long track leading to a deeply placed coil of intestine or no track at all in those cases in which the bowel is directly adherent to the wound. A true fæcal fistula can be readily differentiated from an abscess discharging fæcal-smelling brown pus by the oral administration of a cachet of methylene blue, which will appear on the dressing in the case of a true fistula.

*Treatment.*—Fistulæ will heal spontaneously provided that there is (1) no distal obstruction, (2) no specific infection of the track and (3) no adherence of the mucous membrane to the skin. Treatment is therefore directed to these factors, but in some persistent examples operative closure will be required, and will consist in either an extra-peritoneal repair or a formal resection of the adherent coil.

### STRICTURE OF THE INTESTINE

Stricture of the intestine is not of common occurrence, except for the malignant variety. The following types are found :—

1. Congenital, which have already been described on p. 614.
2. Infective. Tuberculosis in the small bowel and syphilis and dysentery in the colon may lead to such extensive scarring that a fibrous stricture results.
3. Traumatic. The constriction line in a strangulated hernia is sometimes so damaged that its repair by oversewing is needed. The resulting scar tissue may contract sufficiently to cause a narrowing of the gut. Faulty technique in intestinal anastomosis may be followed by a stricture at the suture line. Impacted foreign bodies or gall-stones may produce a linear circumferential ulcer, the healing of which may lead to stenosis.
4. Neoplastic, which are discussed below.

A stricture of the small intestine will give rise to the gradual onset of chronic intestinal obstruction, whereas in the colon the picture is likely to be more acute.

*Treatment* consists in resection and anastomosis or a short-circuiting operation.

## NEW GROWTHS OF THE INTESTINE

**The Small Intestine.**—*Benign* growths are very rare. The connective tissue tumours are fibroma, myoma, lipoma, fibromyoma, fibrolipoma and angioma. The adenoma is the only epithelial tumour. They all tend to become pedunculated either into the lumen or on the peritoneal surface. The former are likely to act as the apex of an intussusception, but apart from this they are unlikely to give symptoms.

*Malignant* growths are also rare, except that lymphosarcoma is by no means infrequent in the ileocaecal angle. Carcinoma in the form of the scirrhus stricture is occasionally seen in the jejunum.

**The Large Intestine.**—*Benign* tumours are rare and are similar to those in the small



FIG. 306

Multiple adenomatous polypi of the colon.



FIG. 307

Multiple primary carcinomata of the colon.

bowel. Multiple adenomatous polypi (Fig. 306) are quite common in the lower part of the colon, but must not be confused with the polypi of irritative or inflammatory origin. They give rise to pain, diarrhoea, the passage of blood and mucus and secondary anæmia. If the symptoms are sufficiently severe and uncontrolled by irrigation, a resection of the affected part of the colon must be considered. They are of importance in that they may be the starting-points of multiple primary carcinomata of the colon (Fig. 307).

## CARCINOMA OF THE COLON

The large intestine is one of the more common situations of cancer affecting both sexes equally.

*Naked-eye Appearance.* — 1. The **Scirrhus Ring Stricture** may occur in any part of the colon except the cæcum, and is the commonest variety. The growth spreads in the submucous coat around the circumference of the bowel. In its external appearance the colon shows little abnormality except that at one point it seems to have had a thick piece of string tied tightly round it; but when it is cut longitudinally a narrow growth is seen encroaching on the lumen (Fig. 308). Its base is rarely more than one and a half inches broad, and there is little evidence of spread either above or below the growth. The gut above bears all the signs of chronic intestinal obstruction, *i.e.*,



FIG. 308

The scirrhus ring carcinoma of the colon.

gut above bears all the signs of distension, hypertrophy and colitis with stercoral ulceration.

2. The **Ulcer** is relatively uncommon. It has raised and rolled edges, it penetrates deeply and spreads to the lymphatic glands more quickly than the other varieties.

3. The **Fungating Growth** is typically seen in the cæcum (Fig. 309), but may occur in any part of the colon. It has a broad base and grows luxuriantly into the lumen of the bowel. It does not cause obstruction and seems to expend its energy in its local growth, for its spread to the lymph glands is of late occurrence (Fig. 310).



FIG. 309

Large ulcerating and fungating carcinoma of the cæcum.

*Microscopic Appearance and Method of Spread.*—These growths are columnar celled, usually of the adenocarcinomatous type,

and tend to undergo colloid degeneration (Figs. 311 and 312).

They are not highly malignant, as they involve the lymph glands quite late and metastases are not common. They do, however, tend to invade neighbouring structures and to form fistulous connections,

*e.g.*, a growth of the transverse colon may spread into the stomach,



FIG. 310

Fungating carcinoma of transverse colon.

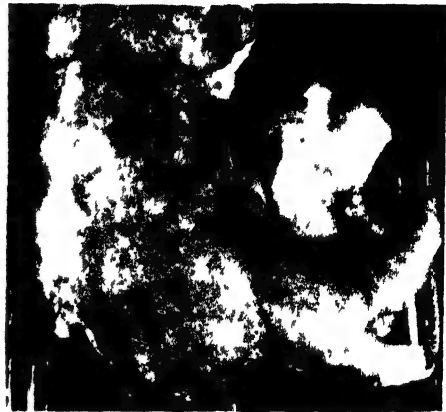


FIG. 311

Colloid carcinoma of ileocaecal region.

that of the lower sigmoid into the bladder and in the fixed parts of the colon extraperitoneal spread with abscess formation may occur.

Coils of small intestine may be involved. Late spread includes lymphatic involvement of the area of the portal fissure and seedling growths on the surface of the peritoneum.

*Symptoms.*—The clinical picture depends on the situation of the growth and its nature. In the proximal part of the colon the contents are liquid and can continue their onward progress through even a greatly stenosed canal, whereas in the distal portion solid faeces are more easily liable to arrest by comparatively minor degrees of contraction.

In the proximal colon, *i.e.*, the caecum, ascending colon, hepatic flexure and proximal one-third of the transverse colon, the growth is more likely to be fungating rather than scirrhus, and these patients will present themselves for one of several reasons: (1) relatives have watched with anxiety the patient getting thin and more easily tired, while the subject remains unaware of any change, save that he feels

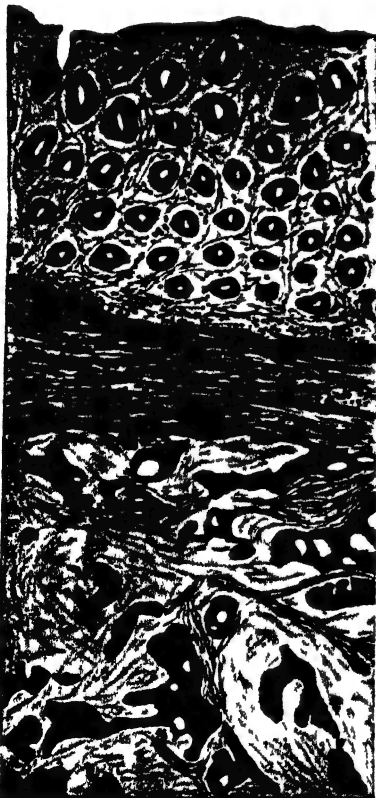


FIG. 312

A drawing of a microscopic section showing a colloid carcinoma of the large intestine. (*Kettle*)



rather "off colour"; (2) there may be vague abdominal pain, loose motions or actual diarrhoea and a feeling of malaise; or (3) the patient suddenly becomes aware of a tumour in the right side. On examination the tumour will be palpable and occult blood will be found in the stools.

In the distal colon, *i.e.*, the distal two-thirds of the transverse colon down to the rectosigmoid junction, the growth is usually an annular stricture, and the picture is that of chronic intestinal obstruction with its gradually increasing constipation alternating with occasional attacks of spurious diarrhoea. The tumour cannot be felt in most cases, but blood and mucus are present in the stools. Rarely carcinoma in any part of the colon is heralded by a severe hæmorrhage, and Grey Turner has emphasised the great importance of this symptom as an early indication of cancer in the colon.

*Examination.*—Radiography combined with a barium enema (Fig. 313) will give evidence of the presence of the annular stricture, and sigmoidoscopy will demonstrate growths of the sigmoid colon. It is necessary, however, to issue this word of warning. The barium enema technique does not, indeed cannot, reveal the existence of all the growths in the colon, and many proved carcinomata have nevertheless yielded a normal radiographic picture. The conclusion to be drawn is that a negative X-ray finding must not be allowed to prejudice the diagnosis when the clinical signs are suggestive.

*Treatment.*—*A.* The growth is discovered before acute intestinal obstruction has set in. The abdomen is opened in the middle line and if the growth is operable the appropriate type of resection and anastomosis is carried out at once (see below). This may be accompanied by a cæcostomy to relieve the suture line of any strain from distension, but this is a matter of individual choice.

If the growth is inoperable, a short circuit should be performed to avoid the subsequent development of obstruction. This will not be possible in those growths low down in the sigmoid colon, for which a colostomy will be required.

*B.* Acute obstruction is present and the site of the growth unknown. An immediate operation must be arranged, and a median sub-umbilical incision having been made, the site and operability of the growth



FIG. 313

An X-ray of a barium enema showing a complete hold-up of the opaque solution by a carcinoma of the transverse colon.

must be investigated. If it is operable, the colostomy most suitable for drainage and for the requirements of the subsequent resection is performed ; but if inoperable, a permanent colostomy or short-circuit operation will be indicated.

The procedure known as "blind cæcostomy," in which the cæcum is drained without exploring the abdomen, must be reserved for those patients whose condition is very grave as a result of late diagnosis and who would probably not survive any extensive intraperitoneal manipulations. It should be regarded only as a life-saving measure in these grave emergencies, and is not to be considered as a routine procedure.

### THE GENERAL PRINCIPLES OF OPERATIVE TREATMENT

1. In the presence of acute obstruction, resection followed by immediate anastomosis is never justifiable under any circumstances whatsoever.

2. Conditions of operability. A growth of the colon is operable if there are no secondary deposits in the liver, distant lymph glands or on the surface of the peritoneum, and provided it has not infiltrated seriously the posterior abdominal wall or other viscera.

3. Technical principles. Each growth must be removed with its lymphatic drainage area, the colon must be mobilised (if necessary) so that the bowel can be brought outside the abdomen prior to resection and anastomosis, and the resection lines must be so placed that the ends prepared for anastomosis have an assured blood supply. Thus, for growths of the cæcum, ascending colon and hepatic flexure, the tissues removed include the last 6 in. of the ileum to the proximal third of the transverse colon together with a wedge-shaped area of peritoneum, including the lymphatic glands.

Growths of the transverse and sigmoid colon are resected with wedge-shaped areas of mesocolon and united by end-to-end anastomosis. Growths of the splenic flexure and descending colon require an excision of the distal third of the transverse colon, splenic flexure, descending colon and part of the sigmoid colon, together with a corresponding wedge of peritoneum.

**Paul's Operation** is reserved for certain cases of carcinoma of the transverse and sigmoid colon, especially the latter, in the presence of acute intestinal obstruction. Occasionally a growth in the freely movable parts of the colon will be so easily delivered out of the wound that the temptation to remove it is almost irresistible. Although not an ideal procedure, it may be countenanced provided that a Paul's tube is tied into each end of the divided bowel. The closure is, of course, left to a subsequent occasion.

R. M. HANDFIELD-JONES.

## CHAPTER XXX

### INTESTINAL OBSTRUCTION

**I**NTESTINAL obstruction is a condition in which the normal onward passage of intestinal contents is prevented either by a mechanical or a paralytic cause and is to be distinguished from constipation, in which the faecal evacuation is merely delayed by a sluggish intestinal musculature. The classification of the causes of intestinal obstruction can never be a simple matter, but it is highly desirable that the mechanical causes should be kept entirely separate from those in which the bowel muscle is paralysed, because the clinical picture and the treatment are so different.

### MECHANICAL OBSTRUCTION

This type of obstruction, which comprises the majority of cases, manifests itself clinically in three varieties, viz., acute, chronic and chronic obstruction which is becoming acute. These must be described separately.

#### ACUTE INTESTINAL OBSTRUCTION

*Causes.*—Acute obstruction is due either to pressure upon the intestine from without, to a pathological or developmental process causing contraction of its wall or to the blockage of its lumen by some solid substance inside it. If the obstruction is due to impaction within the lumen or to stenosis of the gut-wall, or if a peritoneal band passes across the intestine in one place only, a mechanical block will be established ; but if the band passes across the bowel in two places or if a loop of gut is caught by any means, *e.g.*, the opening of a hernial sac, the blood vessels of the mesentery of the loop are obstructed also and “strangulation” will occur. The causes of acute obstruction, therefore, are classified as follows :—

#### *Obstruction with Strangulation (62 per cent.)*

- |  |                                    |
|--|------------------------------------|
| 1. In external herniæ (45 per cent.).  | (c) Appendix.                      |
| 2. In internal herniæ (2·5 per cent.). | (d) Great omentum.                 |
| 3. By bands (11·4 per cent.) :         |                                    |
| (a) Peritoneal adhesion.               | 4. Intussusception (15 per cent.). |
| (b) Meckel's diverticulum.             | 5. Volvulus (2·6 per cent.).       |

Mesenteric vascular occlusion is not included here, as is usually done, but is described under Paralytic Obstruction (p. 642).

*Obstruction without Strangulation (38 per cent.)*

- |  |   |
|--|---|
| 6. Adhesions (7·4 per cent.).                | 8. Impaction of gall-stones (0·7 per cent.).    |
| 7. Congenital malformations (0·6 per cent.): | 9. Impaction of foreign bodies (0·3 per cent.). |
| (a) Atresia of small gut.                    | 10. Growths of intestine.                       |
| (b) Atresia of large gut.                    | 11. Diverticulitis.                             |
| (c) Imperforate anus.                        | 12. Fibrous stricture.                          |
|  | 13. Pressure from without.                      |

**Pathology—A. Without Strangulation.**—The intestine above the obstruction becomes progressively distended with gas and fluid, while the gut below is empty and in spasm, being white and firmly contracted.



FIG. 314

A loop of ileum which had become gangrenous in a strangulated femoral hernia. The constriction line can be well seen. The proximal part of the intestine (on the left) is markedly distended.

The degree of distension depends upon the site of the obstruction and the time which has elapsed since its onset. Generally speaking, it is most marked in large intestine obstruction, and becomes less prominent as the site of the lesion rises in the intestinal tract: the greatest distension occurring in volvulus of the sigmoid colon, the least in a high jejunal obstruction. The gut-wall becomes very thin and at first is pale, then red and cyanotic, but only in neglected cases will it be so damaged as to permit the passage of organisms into the peritoneal cavity.

**B. With Strangulation.**—The intestine above and below the strangulated gut are in the same condition as in simple obstruction, but the loop itself will show the effects of vascular compression (Fig. 314). In the first few hours the pressure is sufficient to occlude the veins only, so that the loop becomes distended, swollen, œdematous and deep blue in colour owing to *venous congestion*. Blood and mucus escape into the lumen, and a blood-stained effusion is secreted by the peritoneal coat. As the tension in the loop rises a time will come when the arterial pressure is overcome and *gangrene* of the loop inevitably follows. The gut now becomes black, the peritoneum loses its shiny lustrous appearance, organisms pass out into the blood-stained exudate and local or general peritonitis rapidly supervenes. It is important to realise that these changes will be more advanced in both time and severity along the line of compression of the intestine by band, hernial orifice or other cause.

**GENERAL SYMPTOMS.**—The general condition of the patient is so

<sup>1</sup> These are only rarely causes of acute obstruction without a previous history of chronic obstruction.

typical that minor adjustments and additions only will be needed in the description of the various causal diseases.

**Pain** is the first symptom. It is severe, abrupt in onset and is centred around the umbilicus or, less commonly, generally over the abdomen. After the first few hours, colicky, griping pains due to violent peristalsis are added to the constant dull, sickening ache. In untreated patients, the picture changes as peritonitis sets in, the colicky pain disappearing and the dull ache diminishing.

**Vomiting** occurs at once and consists of one or more attacks in the first half hour. It then ceases for a time until the true persistent vomiting of obstruction is established. The interval depends on the site of the obstruction, and the vomiting and the distension may be said to be inversely proportional to one another. In high jejunal obstruction distension is slight and the vomiting quickly sets in, while in a low colon lesion the distension may be enormous, but the vomiting does not come on for forty-eight or more hours. At first the vomit consists of stomach contents, then green bile is the chief constituent and later it becomes brown and more and more offensive. In the early stages it is projectile in type, but later large quantities of so-called "fæcal" vomit seem to pour out of the mouth without any apparent movement of the patient.

**Absolute Constipation.**—Within half an hour of onset there may be an action of the bowel, after which neither fæces nor flatus are passed either voluntarily or in response to enemata (see below). **Thirst** soon becomes an urgent symptom, patients ceaselessly demanding more and more to drink.

**GENERAL SIGNS.**—1. **Shock** is present in all cases with strangulation, but is absent or slight in others. The patient wears an anxious expression, the face is pale and bathed in a cold sweat, the pulse is thin and weak, and the temperature is subnormal. The pulse rate slowly returns to normal and gradually becomes quicker, but the temperature will not rise above normal until peritonitis has set in.

2. **Distension** has already been referred to. It is important to differentiate between that formed by the large gut and that by the small, as this may have an important bearing on treatment. It is quite impossible to do so in many patients, and there is no infallible test, but colon distension fills up the flanks and the epigastrium, leaving an impression of a flat, soft, central area in the abdomen; whereas small gut distension balloons the centre and does not encroach to the same extent on the flanks and epigastrium.

3. **Reaction to Enemata.**—Blind reliance on enema tests cannot be too strongly deplored. An enema given soon after onset **may** produce both flatus and a fæcal result. A second enema given half to one hour later will produce no result and will need to be syphoned back. Herein lies the true worth of the enema test, but it must be realised that one enema may prove misleading.

4. **Other Abdominal Signs.**—In many cases there will be none, but in intussusception, external herniæ and some growths the tumour can be felt either through the abdominal wall or per rectum, and in intussusception blood and mucus will be found in the rectum.

**5. Absence of Other Abdominal Signs.**—*Tenderness and rigidity are absent* until the onset of peritonitis, by which time the prognosis is very bad. The abdomen moves with respiration, the anterior abdominal wall is soft, flaccid and only in very rare cases is there localised tenderness. The absence of these two signs is of the greatest importance. Visible peristalsis is sometimes described as a sign of acute obstruction; that is a grave mistake and a most dangerous doctrine. It is true that even normal peristalsis may be seen in very thin, old people, but it is essentially a sign of chronic obstruction, which is threatening to become acute and in which the bowel is hypertrophied as well as distended. We wish to emphasise that it is the absence of these signs which form so strongly positive a link in the chain of early diagnosis.

**6. Late Signs.**—Peritonitis brings its own signs, but one result of the vomiting is a rapid dehydration of the body. The Hippocratic countenance is only too typically present with its pallor, its shrunk features, its hollow orbits and sunken eyes.

These signs are somewhat altered in a strangulated external hernia, in which pain is felt in the swelling, which becomes tense and tender, otherwise the picture is similar.

**GENERAL DIAGNOSIS.**—The high mortality in acute obstruction has always been a matter of grave concern, and much anxious research has been directed to this subject, but the absolute essentials for success are early diagnosis and prompt operation. The clinical picture detailed above should suffice to satisfy the practitioner of the *presence* of obstruction. He should also attempt to define the site and the nature of the lesion. A careful history is taken, all hernial orifices examined, a rectal or vaginal examination performed and the distension investigated to distinguish (if possible) large from small gut obstruction. Percussion and auscultation of the cæcal region may give valuable evidence.

The differential diagnosis includes all acute abdominal emergencies and all types of colic. The absence of tenderness, rigidity and pyrexia excludes all the inflammatory diseases and the ruptures of viscera. The history should suffice to eliminate biliary and renal colic, and tabes is excluded by investigating nervous reflexes and the pupil reactions. There should be no difficulty in differentiating the mechanical from the paralytic obstruction, provided the small intestine is carefully listened to by stethoscope. Excessive gurglings are heard in mechanical obstruction, silence reigns in paralytic ileus.

**GENERAL TREATMENT.**—Acute obstruction being a clinical syndrome and not a pathological entity, treatment is directed to the relief of its cause and the evacuation of the distended bowel. When the nature of the obstruction can be recognised, *e.g.*, a strangulated external hernia or an intussusception, appropriate treatment is adopted, but in many patients the lesion is intra-abdominal and neither its site nor its type can be defined. In such cases the abdomen is opened in the middle line below the umbilicus and the cæcum is palpated. Its distension points to a large gut obstruction and its collapse to a small gut lesion. The most distended coil of small gut tends to present

in the wound, and if this is gently followed the obstruction should be close at hand and quickly discovered and appropriate measures taken to overcome the cause. If the general condition is bad and the gut greatly dilated, a jejunostomy used to be practised to drain away its toxic contents. Similarly a patient may be so gravely ill, having vomited persistently for several hours, that any operation seems too dangerous; a cæcostomy or jejunostomy used to be advised. To-day these procedures have given place to intestinal drainage via a Ryle's duodenal tube, the full technique of which is given on p. 643.

### STRANGULATED HERNIÆ AND STRANGULATION BY BANDS

1. **Strangulated External Hernia** (Chap. XXVII, p. 565).

2. **Strangulated Internal Hernia.**—There are a number of peritoneal fossæ, into which coils of intestine may enter. They are situated around the duodenojejunal junction, near the cæcum and in the mesentery of the sigmoid colon. The foramen of Winslow has been known to transmit coils of small gut into the lesser sac, and the opening in the transverse mesocolon in the operation of posterior gastro-jejunostomy has also served as a hernial orifice. Strangulation in all these situations may occur and will give a typical picture of acute obstruction with no indication of its cause (Fig. 315). Immediate operation is needed to release the intestine.



FIG. 315

A hole in the mesentery of the small intestine through which coils of intestine have prolapsed. An example of a strangulated internal hernia.

3. **Bands.**—(a) Peritoneal bands and adhesions result from previous inflammation of the peritoneum, appendicitis, peritonitis and inflammation of the mesenteric lymph glands being common examples. Surgical trauma may also produce adhesions, particularly if raw areas are left in the abdomen uncovered by peritoneum. Such adhesions may be wide, flat, thin sheets or single rounded cords, the latter being more likely to cause acute obstruction than the former. These bands stretch usually from the mesentery to some other abdominal structure or to the parietes.

(b) Meckel's diverticulum may be attached to the umbilicus by a fibrous cord, or its end may have been free but have become secondarily attached by inflammation to the mesentery, a coil of intestine or the posterior abdominal wall. In any of these conditions the diverticulum may act as a band, around which a coil of small intestine may become strangulated.

(c) The appendix, or the great omentum, may become attached in a similar manner and likewise act as a band.



Bands of whatever nature act in one of two ways, either a short tense band compresses a loop of intestine, which has slipped beneath it, or a long lax band forms a noose through which the loop slides.

*Symptoms.*—This type of acute obstruction usually affects the small intestine in the lower reaches of the ileum. The onset is abrupt, the pain severe, the shock marked, the distension moderate, and persistent vomiting is established within a few hours.

*Treatment.*—The abdomen having been opened below the umbilicus, free fluid, either clear or blood-stained, is immediately apparent, and the site of the obstruction is quickly found by gently following the most distended loop. The band is carefully examined, and when proved to be a thickened peritoneal adhesion (and not a coil of intestine) it is divided. The appendix, or Meckel's diverticulum, if acting as the band, should be removed unless the general condition prohibits anything but essentials. The strangulated coil is carefully examined and, if its vitality is assured, the compression groove is closely scrutinised for it may show early gangrene, ulceration or perforation long before the loop is affected. Any weakness at this line can be remedied by oversewing. If the viability of the loop is in doubt, the wisest procedure is to wrap it up in hot, moist packs for three minutes, after which its colour and pulsation will have returned and peristalsis may be elicited. When the gut is obviously gangrenous, a resection with end-to-end anastomosis is performed. The amount to be removed depends on the degree of distension in the proximal coils. The gut below the dead loop is divided as near to the compression line as is convenient, but the proximal gut must not be cut across until a reasonably healthy coil has been discovered. Before the abdomen is closed a careful search must be made to exclude the presence of a second band.

*After-treatment* is important. Immediately on the patient's return to bed, a continuous intravenous drip saline is started. As soon as he has recovered consciousness, he is placed in Fowler's position. Small sips of iced water are given as frequently as desired. Morphia may be given only if essential and then in doses of  $\frac{1}{2}$  gr. The following medicine is to be given in doses of  $\frac{1}{2}$  oz. four-hourly:—

R	Eserin Sulphate	.	.	.	.	aa gr. $\frac{1}{16}$ .
	Strychnine Hydrochlor.	.	.	.	.	
	Syrup : Limonis.	.	.	.	.	3fs.
	Aquam.	.	.	.	.	ad 3fs.

If all goes well a turpentine enema is given at the thirty-sixth hour. This should give a good result, vomiting should have ceased and the danger be over. If vomiting persists, and the general condition fails to improve, the patient is threatened with paralytic ileus and treatment is directed toward that condition (see below).

### INTUSSUSCEPTION

This remarkable condition entails the invagination of one part of the intestine into that immediately below it, followed by a progressive



advance of the invaginated portion, so that more and more intestine is drawn up within the outer layer.

It will readily be understood that in the affected segment three layers of intestine are involved. The part which first becomes invaginated is known as the *apex*, and this remains constantly in the lead of the advancing invagination. The three layers are the *entering layer*, which turns over at the apex to become the *returning layer* and this in turn joins the *outer* or *ensheathing layer*. The invaginated portion, viz., the entering and returning layers, is also referred to as the **intussusceptum**, and the sheath as the **intussusciens**. As the invagination proceeds onwards its increase in length is always at the expense of the outer ensheathing layer. Clearly, the intestine cannot take part in this process without dragging with it its mesentery, which comes to lie between the entering and the returning layers. The point at which it enters is known as the *neck*. As more and more gut becomes drawn in, the congestion at the neck increases till finally the veins of the mesentery are compressed. The intussusceptum is, therefore, closely analogous to the loop of a strangulated hernia, with the one exception that it is not a closed loop but points forward into the distal intestine (Fig. 316). The venous congestion leads to swelling and oedema of the intussusceptum, most marked at the apex, as a result of which blood and mucus are poured out into the distal intestine. Later the peritoneum is involved and a local plastic peritonitis fixes together the entering and returning layers, and the intussusception is now **irreducible**. If the condition remains unrelieved the intussusceptum finally become gangrenous, and cases are recorded in which it sloughed away and was passed per rectum.

**The Causes of Intussusception** include any source of irritation or a tumour in the lumen or in the wall of the intestine, which stimulate over-violent peristaltic waves in an effort to expel the trouble. In practice the acute intussusception, in infants, is almost always due to swollen Peyer's patches and the chronic variety to either benign or malignant tumours (Figs. 317 and 318).

**The Types of Intussusception** are classified as follows :—

1. Enteric (10 to 15 per cent.).
2. Enterocolic (75 to 80 per cent.)  $\left\{ \begin{array}{l} \text{ileocaecal.} \\ \text{ileocolic.} \end{array} \right.$
3. Colic (5 to 10 per cent.).

The **ENTERIC** type affects the small gut only. It occurs in children under 10 years of age in whom it is always due to an obvious cause, such as a polyp, a Meckel's diverticulum or a tuberculoma, and in adults who have a malignant tumour of the intestine.

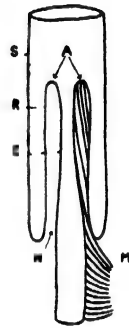


FIG. 316

A diagram showing the component parts of an intussusception.

S, the sheath ;  
R, the returning layer ;  
E, the entering layer ;  
A, the apex ;  
N, the neck ;  
M, the mesentery.



FIG. 317

A lymphosarcoma of the ileum causing an intussusception.

ileum, which act as the stimulus to violent peristalsis.

*Symptoms.*—The baby is seized with an attack of abdominal pain of a colicky nature which lasts about a minute. These attacks recur at regular intervals of a few minutes. During the attack the child curls itself up and screams loudly, while its face becomes very pale. In the intervals the colour returns, but the child lies unusually quiet. The bowels will be emptied during the first few spasms, and later blood and mucus may be passed. Vomiting is not a feature of this condition.

*Signs.*—The abdomen is soft and the tumour should be palpable as a soft sausage-shaped swelling which rapidly hardens as the spasm of pain comes on. It is slightly curved, having the concavity facing towards the umbilicus.

The COLIC type affects adults only and is due to a malignant tumour of the colon.

The ILEOCÆCAL type is commonest of all. In it the ileocæcal valve is the apex and the ileum is invaginated into the colon. In infants the mesentery is so long that the intussusception may go right through the colon and present at the anal orifice. In the Ileo-colic variety the invagination begins as a pure enteric intussusception about 6 ins. from the ileocæcal valve. Soon the ileal apex passes through the valve and the colon is then involved (Figs. 319 and 320).

*Acute Intussusception* occurs in infants under 2 years of age, more commonly in boys than girls, and usually chooses the fittest and fattest. It is preceded by a mild attack of enteritis with relaxed and foul-smelling motions. This leads to engorgement and swelling of the Peyer's patches in the terminal



FIG. 318

Multiple papillomata causing an intussusception.

The right iliac fossa gives the impression of being curiously empty. If any difficulty is experienced a finger should be introduced into the rectum, when a bimanual examination can hardly fail to discover the tumour, and, in addition, the finger will be covered with blood and mucus. The diagnosis should never be in doubt, though Henoch's purpura may give difficulty in rare cases.

*Treatment.*—Immediate laparotomy is called for. The abdomen is opened through a right paramedian incision and two fingers are introduced and seek for the apex. Gentle pressure from below causes the invagination to slide backwards, and in some cases it runs back so quickly that the finger fails to keep in contact with it. Reduction is complete except for the apex, and the tumour is now withdrawn from the abdomen and surrounded with hot, moist packs and gently and progressively compressed from the distal aspect. Under no circumstances whatever may the entering layer be pulled on. Within a minute or two the reduction is complete, but care must be taken to ensure that the last dimple is reduced.

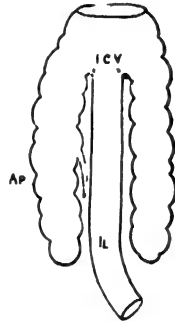


FIG. 319

A diagram illustrating an ileocaecal intussusception. I.C.V., the ileocaecal valve; Il., the ileum; and Ap, the appendix.

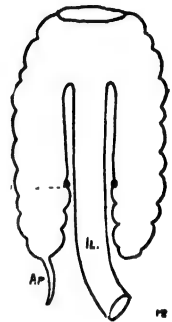


FIG. 320

A diagram illustrating an ileocolic intussusception. The references are the same as those in Fig. 319.

If the intussusception is irreducible and its walls gangrenous, the whole area must be resected, but the results are very bad; if the sheath is not gangrenous it is opened vertically and the intussusceptum is cut off at the neck, the cut margins being united by a circular stitch (Jeset's operation). Fortunately very few cases are irreducible, for in them the mortality rate is high.

Contrary to all expectation, recurrence after reduction is exceedingly rare.

**Chronic Intussusception** is a rare condition in adults over 60 years of age, being usually due to carcinoma of the colon. The story is that of chronic obstruction and may last for several weeks. Blood and mucus appear in the stools, constipation is present, and mild colicky pains occur. The condition is readily diagnosed by a barium enema and the treatment is directed to its cause. Multiple and retrograde intussusception are described, but occur only as a terminal manifestation at the approach of death.

## VOLVULUS

A volvulus is produced by the twisting of a coil of intestine on the axis of its own mesentery, so that not only is the lumen obstructed at each end but the vessels are compressed. It occurs in the sigmoid colon, the caecum and the small intestine.

**Volvulus of the Sigmoid Colon** is unlikely to occur in a normal

sigmoid colon and mesosigmoid. The freely movable sigmoid loop is fixed at each end, above at its origin from the descending colon and below at the rectosigmoid junction. These fixed attachments are normally some distance apart, but perisigmoiditis leads to the formation of thickened bands in the mesocolon. The contraction of these bands draws the fixed ends of the loop into close contact. A narrow pedicle is thus formed, allowing facile rotation of the loop. An overloaded sigmoid colon will hang down into the pelvis and some irregular movement may cause it to turn over. The lumen at each end becomes obstructed, gas rapidly forms, and in a short time untwisting is not possible. The analogy of the closed-loop pathology is here perfect (see general pathology above), as both the gut and its vascular supply are obstructed, and the results are similar in all respects.

*Symptoms.*—Volvulus of the sigmoid colon occurs in both sexes after the age of 45 years. There is a sudden attack of abdominal pain with an initial single vomit. The symptoms are not so severe as in other forms of strangulation, but the distension rapidly becomes enormous, the huge sigmoid loop filling the entire abdominal cavity and causing both respiratory and cardiac embarrassment. Its extent must be seen to be believed, and it provides a sure guide to early diagnosis.

*Treatment* consists in immediate operation under spinal anaesthesia. A large paramedian incision exposes the distended loop. An attempt is made to pass a flatus tube from below, while the surgeon endeavours to guide its nozzle into the loop. This is rarely successful, and must not be tried if the distension is very great. The rotation must be untwisted, but this will not be possible in many cases until the gas has been let out through a small puncture in the gut. If nothing else is done the volvulus is likely to recur, and the sigmoid loop should be fixed. The only satisfactory method is to do a colostomy, which has the advantage of draining the loop. At a later date an extraperitoneal closure gives permanent fixation with a restored lumen.

**Volvulus of the Cæcum** occurs only when the mesocolon of the ascending colon persists. The twist of the cæcum produces a picture of acute obstruction, but the exact diagnosis is unlikely to be made until operation.

*Treatment* consists in laparotomy and untwisting the cæcal rotation.

**Volvulus of the Small Intestine** is very rare but very severe. The picture is that of a small gut obstruction, and the abdomen must be opened at the earliest opportunity.

### GALL-STONE OBSTRUCTION

Gall-stones large enough to become impacted in the intestine enter the duodenum or the hepatic flexure of the colon as the result of an ulcerating cholecystitis. The colon usually transmits the stone without impaction, but in the small gut the stone is held up at its narrowest part, 39 in. from the ileocaecal valve (Fig. 321). Elderly women are most commonly affected and they have suffered from recurrent attacks

of gall-stones and cholecystitis (but not of colic or jaundice) during the preceding few years. The impaction produces an attack of acute abdominal pain, but shock and distension are not marked. Vomiting is a prominent symptom. It may be possible on rare occasions to feel the stone in the left iliac fossa. No difficulty will be experienced at operation, the stone being gently pushed upwards into a healthy coil of gut and removed.

A similar clinical picture is given by other foreign bodies, such as enteroliths and those swallowed, but these are very rare.

### CHRONIC INTESTINAL OBSTRUCTION

Chronic obstruction is due to a slowly increasing narrowing of the intestinal canal, so that the passage of its contents is rendered more difficult. It is usually met with in the colon. Its causes may be classified as follows :—

#### A. Extrinsic—

1. Adhesions.
2. Pressure from without as from tumours, etc.

#### B. Intrinsic—

3. Strictures—
  - /Inflammatory.
  - Traumatic.
  - \Neoplastic.
4. Carcinoma of colon and rectum.
5. Chronic diverticulitis.
6. Chronic intussusception.
7. Chronic volvulus.
8. Ileocaecal tuberculosis.
9. Ileocaecal actinomycosis.
10. Faecal impaction.
11. Hirschprung's disease.
12. Chronic regional ileitis.



FIG. 321

A large gall-stone firmly impacted in the intestine causing acute intestinal obstruction.

*Pathology.*—The intestine above the obstruction is moderately distended and greatly hypertrophied (Fig. 322). A catarrhal inflammation of the mucous membrane follows and, later, stercoral ulcers develop.

*Clinical Picture.*—The cause of the obstruction is not always apparent, but in many patients it will produce its own symptoms before the obstruction has become a prominent feature. In the unheralded variety the patient complains of a gradually increasing constipation and after a considerable time (possibly many months) there will be a mild degree of colicky abdominal pain, distension, flatulence and gurgling sounds inside, which may be embarrassingly

evident. Later still, alternating with the constipation, there are attacks of "spurious" diarrhoea, due to the colitis, stercoral ulceration and putrefactive changes in the delayed fæces. The patient complains of malaise, loss of weight, and although unable to be precise, he will be convinced that "something is not quite right inside."



FIG. 322

A portion of ileum showing a tuberculous stricture, above which the gut is distended and greatly hypertrophied. The patient came to operation with acute obstruction due to the impaction of a damson stone (in the test tube) in the stricture.

An abdominal examination reveals a moderate degree of distension with visible peristalsis, which may even produce the typical "ladder" pattern. Careful investigation by palpation, percussion, auscultation and a rectal examination should indicate the site of the obstruction. These cases terminate in acute obstruction, in peritonitis due to the perforation of a stercoral ulcer or from reasons connected with the primary cause.

*Treatment* is essentially operative and is directed to the cause. If this should prove to be inoperable, a colostomy or an anastomosis will be needed.

CHRONIC OBSTRUCTION BECOMING ACUTE hardly merits the distinction of a separate category. A study of the pathology of chronic obstruction suggests that a complete blockage is likely to occur, if appropriate treatment of the cause has not been undertaken at a sufficiently early stage.

## PARALYTIC OBSTRUCTION

The cessation of all intestinal movements due to paralysis of the muscles produces a clinical picture closely akin to acute intestinal obstruction, but no form of mechanical blockage is present. It can unquestionably be called the most terrible complication of abdominal disease. There are two types, viz., paralytic ileus and mesenteric vascular occlusion.

### PARALYTIC ILEUS

This may be the result of the following conditions :—

1. Inflammatory, as a complication of local or general peritonitis.
2. Toxic, as in uræmia and lead poisoning.
3. Neuropathic, when the spinal cord and the peripheral nerves are diseased or compressed.
4. Traumatic, after rough handling of intestine or pulling on the mesentery during operations.

The inflammatory type of ileus usually occurs after operation for some abdominal lesion accompanied by spreading peritonitis. The more rapid and more virulent infections are characterised by a dirty, offensive sero-purulent effusion into the peritoneal cavity with few protective adhesions. These are the conditions which favour the onset of ileus. Thick yellow pus, however offensive in smell, is not of such dangerous significance. This type of paralysis is in the nature of a protective reflex, much as is muscular rigidity in diseases of joints. Active peristalsis must spread inflammatory exudates, whereas immobility of the intestine tends to localise the peritonitis.

*Symptoms.*—After operation, the post-anæsthetic vomiting having subsided, a period of twenty-four to thirty-six hours follows in which the patient's progress seems as favourable as could be expected. At the end of this period the improvement is not maintained, the abdomen becomes more distended and the patient has occasional attacks of vomiting. There is some slight pain, temperature and pulse rate are slowly rising and enemata fail to produce a result. Such is the picture of a threatened paralytic ileus. If treatment fails the distension increases and the vomiting continues, the pulse becomes weaker and more rapid and later the temperature falls below normal. Profuse effortless vomiting of the fæulent type follows, a profound toxæmia develops and death rapidly ensues.

*Treatment—A. Prophylactic.*—Experience rapidly teaches a surgeon to recognise the type of abdominal emergency which is likely to develop ileus. He must also be satisfied that the peritoneum has been efficiently drained and that no pockets of pus remain untapped. These patients should be given an eserine and strychnine mixture (p. 636) every four hours until the danger is past. Sulphapyridine is useful in infections due to *B. coli* and hæmolytic streptococci.

*B. Early Cases.*—As soon as there is a definite threat of ileus, active steps must be taken to support the patient's strength until the infection has been mastered and intestinal tone restored. No attempt is to be made to produce evacuation of the bowel by aperients given by mouth, but gaseous distension of the colon may be relieved by the passage of a rectal tube or a simple enema. Two grave conditions demand attention; first, marked dehydration, which is a feature of this disease, and second, the toxæmia from the highly poisonous contents of the small intestine. A constant intravenous drip of intravenous glucose saline will be started immediately the diagnosis is made. At the same time a Ryle's duodenal tube is passed via the external nares and swallowed by the patient until its nozzle is in the duodenum. Through it the toxic intestinal contents are constantly aspirated by an automatic overflow device. This procedure has greatly improved the prognosis and replaced older and less satisfactory methods such as a jejunostomy operation. Both intestinal drainage and intravenous drip may be continued for four or five days, after which intestinal peristalsis returns and an evacuation will occur probably without any other assistance beyond a copious enema.

*C. Late Cases.*—If this treatment is not proving successful or if the surgeon has not been called in until the disease is far advanced,

the prognosis is extremely grave and more energetic measures are called for, the best of which is Wilkie's technique. One cubic centimetre of "Prostigmin" is given intramuscularly every hour for six hours, and after the third and sixth injection a glycerin enema is administered. Should the bowel act at any time the remaining injections are not given. The use of ox bile enemata, pituitrin and eserine and spinal anæsthesia have little value in this last desperate stage. *B. welchii* serum has proved to be useless.

**Toxic Ileus** may occur as a terminal manifestation in many toxic states, but is of outstanding importance when associated with an unsuspected failure of renal function. Many patients are referred to a surgeon as cases of acute intestinal obstruction who in reality are suffering from uræmic ileus. This syndrome deserves more attention than it receives and must always be borne in mind whenever cases of obstruction without obvious cause are seen.

### MESENTERIC VASCULAR OCCLUSION

Embolism and thrombosis of the superior mesenteric vessels are seen in middle-aged men and women. They may result from endocarditis, pyæmia or atheroma or from venous thrombosis in association with portal cirrhosis and peripheral infection. The picture is that of acute intestinal obstruction, but one or more actions of the bowel usually occur and a quantity of blood may be voided. The length of bowel involved depends on the site of the vascular lesion, varying from a few inches of ileum to the whole small intestine and ascending colon (see p. 541).

*Treatment* is immediate operation with resection of the gangrenous intestine if possible.

R. M. HANDFIELD-JONES.



## CHAPTER XXXI

### THE RECTUM AND ANAL CANAL

**A**NATOMY.—The Anal Canal is developed from an invagination of the perineal skin and is lined with squamous or transitional epithelium. It is between  $\frac{3}{4}$  and  $1\frac{1}{4}$  in. long. It is surrounded by and closely related to the muscles which control defæcation. These are the external and internal sphincters and the levatores ani.

THE EXTERNAL SPHINCTER has been described by Milligan and Morgan as being composed of three separate parts, viz., subcutaneous, superficial and deep (Fig. 323).

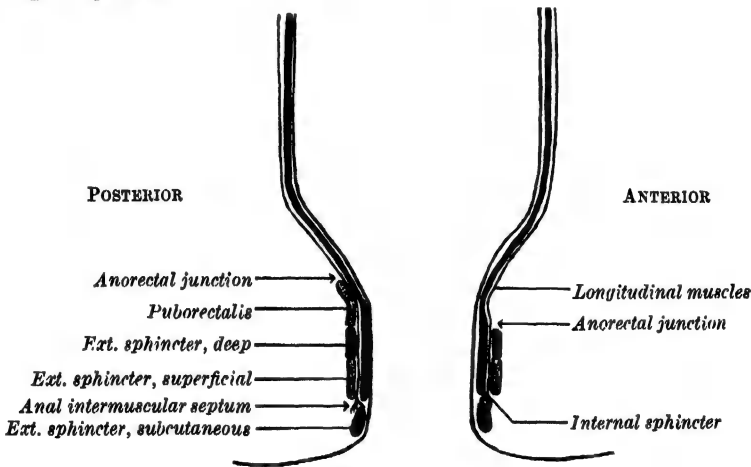


FIG. 323

The anatomy of the anal canal as seen in section.

1. *The Subcutaneous Segment* is an annular bundle of muscle fibres surrounding the lower part of the canal. It lies immediately beneath the skin and can be both seen and felt guarding the anal entrance. By its action the anal skin is thrown into several puckered folds. It is not attached to the coccyx but surrounds the anus, the fibres of each side decussating with each other both in front and behind. Its nerve supply is from the inferior hæmorrhoidal nerve derived from third and fourth sacral trunks. This muscle lies on the same plane as the internal sphincter, from which it is separated by a well-marked ring of fibrous tissue—the anal intermuscular septum, which can be easily palpated.

2. *The Superficial Segment* lies above and outside the former and between it and the deep part of this muscle. It surrounds the lower and middle thirds of the internal sphincter, from which it is separated by the downward prolongation of the longitudinal fibres of the rectum. It arises from the coccyx and ano-coccygeal raphe, encircles the bowel and is attached to the central point of the perineum. It is supplied by the perineal branch of the IVth sacral nerve.

3. *The Deep Segment* is closely associated with the puborectalis fasciculus of the levator ani; indeed its upper border is often inseparable from the latter. It invests the longitudinal coat and the upper third of the internal sphincter. It decussates behind but is attached in front to the ascending ramus of the ischium. Its nerve supply is from IIIrd and IVth sacral.

THE LEVATOR ANI also consists of three parts, ileococcygeal, pubococcygeal and puborectalis, of which the last is the only one of interest here. It arises from the pubis and triangular ligament and encircles the anal canal without gaining attachment to the coccyx. It blends with the upper border of the deep fasciculus of the external sphincter and marks the junction of anal and rectal canals.

THE INTERNAL SPHINCTER is formed by a slight thickening of circular muscle coat and surrounds the whole of the anal canal down to the subcutaneous part of the external sphincter. It does not exist as a narrow annular band or ring as is frequently described.

**The Rectum** is developed from the postanal gut and is  $5\frac{1}{2}$  in. long, stretching from the top of the anal canal to a point opposite the 3rd sacral segment. At its upper limit, where it joins the pelvic colon, the bowel loses its mesentery, the three longitudinal muscle bundles spread out to clothe the whole circumference of the bowel, and the superior hæmorrhoidal artery divides into its two branches. The peritoneal relations of the rectum are of great importance. In its upper third the anterior and lateral walls are clothed by peritoneum, in the middle third the anterior surface only is covered and in its lower third the rectum lies altogether below it.

The mucous membrane is of columnar epithelium containing mucus secreting goblet cells. From the anorectal junction twelve to fourteen longitudinal processes stretch a short way upwards in the rectal mucous membrane; these are known as the columns of Morgagni, between which are the crypts of Morgagni. In the upper two-thirds of the rectum are two posterior and one anterior well-defined folds called the valves of Houston.

The rectum is supplied by the superior hæmorrhoidal branch of the inferior mesenteric artery, by the middle and inferior hæmorrhoidal vessels from the internal pudic artery and by the middle sacral artery. The venous return is from the hæmorrhoidal plexus by the superior hæmorrhoidal veins which enter the portal circulation, and by the middle and inferior hæmorrhoidal veins which deliver blood into the general systemic circulation. This is one of the most important anastomoses between the systemic and portal circulations. The lymphatics from the rectum pass exclusively upwards to the glands in the hollow of the sacrum, to those along the inferior mesenteric artery and to the aortic glands. Those of the anal canal pass to the inguinal regions of both sides.

*Methods of Examination* may be visual, digital and instrumental. It cannot be too strongly stressed that every symptom of rectal disease, however trivial, and every apparently minor ailment of the anal region may be an early sign of carcinoma of the rectum, and no treatment must be undertaken till a thorough examination of the rectum has been made. The use of a rubber finger-stall and of a proctoscope should be within the scope of every practitioner.

Visual and digital examinations may be performed in the left lateral, the lithotomy or the knee-elbow positions. A variation of the last is preferable as it is less embarrassing to the patient, who stands beside a couch, and bending well over leans comfortably on elbows and forearms. The buttocks are widely separated and the presence of pruritus ani, prolapsed piles, thrombosed external piles, fissures and the external opening of fistula-in-ano will be readily detected. The gloved finger, well lubricated

with jelly, is gently inserted, a little time being occupied in overcoming the spasm of the sphincter. The subcutaneous part of the external sphincter is easily felt, and above it the finger appreciates the anal intermuscular septum. Higher still no difficulty is experienced in recognising the anorectal junction (Fig. 323). Note will be taken of any abnormality within, and on withdrawal the presence of blood or mucus will be observed.

Instrumental examination should be preceded by an enema to clear out the bowel. The knee-elbow position is adopted because the viscera then gravitate towards the diaphragm, and when the instrument is introduced air passes in and distends the rectum. A strong head lamp is essential and either a Kelly's proctoscope or a short sigmoidoscope is passed into the bowel.

### CONGENITAL ANOMALIES

**Imperforate Anus.**—1. *Persistence of Cloacal Membrane.*—The anal canal and the rectum are normally developed, but the intervening membrane remains unabsorbed. Meconium causes it to bulge through the anus and present as a bluish swelling. The sphincters are normally developed and removal of the membrane affects an instant and permanent cure.

2. *Absence of Anal Canal.*—The rectum is properly formed, but the anal ingrowth is absent and consequently the sphincters are not present (Fig. 324).

3. *Absence of Rectum.*—The colon ends as a blind sac within the peritoneum, and the anal canal may or may not be properly formed.

Should these anomalies pass unnoticed at birth, the non-appearance of meconium will quickly draw attention to them, and the infant will suffer from intestinal obstruction. The persistence of the cloacal membrane is a trivial matter, but the other conditions depend for their treatment on the presence or absence of the sphincters. If these are absent, no attempt should be made to bring the bowel to an opening in the perineum, for incontinence must result, and a colostomy is to be preferred. If the anal canal with its sphincters is present, every effort must be made to restore the continuity of the bowel, but it may be safer to do a colostomy first and delay the plastic operation until the child is old enough to withstand operation more easily.

**Recto-urogenital Fistula.**—In certain developmental anomalies the rectum does not end in a blind cul-de-sac but has a fistulous communication with some part of the urinary system, in the male at any point between the bladder and the penile urethra, and with



FIG. 324

A section through the pelvis of a female infant which shows complete absence of the anal canal and large bowel ending in a blind cul-de-sac at the level of the posterior fornix of the vagina.

the bladder or vagina in the female. In the male, the fistula must cause a urinary infection and a colostomy should be done; in the female, a fistula into the vagina can safely be left, provided the opening is sufficient to allow proper evacuation without obstruction. On the approach of puberty the question of plastic operation or colostomy will need to be considered.

### INJURIES

The rectum may be injured in a variety of ways: (1) in obstetrics when the perineum is torn during delivery or the parts are injured by forceps; (2) by falls on spiked objects; (3) by the unskilled use of the sigmoidoscope or rectal tubes; (4) during operations on the male urethra and prostate; and (5) by a variety of instruments used by criminals or lunatics.

The *symptoms* are primarily shock, pain and hæmorrhage, and later those due to consequent infection of the pelvic and ischiorectal cellular tissues or of the peritoneal cavity.

*Treatment* is directed to an immediate recognition of the extent of the damage. An anæsthetic must be given and the rectum carefully examined. Minor tears require nothing but local cleaning and rest in bed, with a careful watch for any inflammatory complication. Severe tears will need suturing, and tears into the peritoneal cavity call for laparotomy, suture and a temporary colostomy till the rent has healed. In all penetrating wounds of the rectum, anti gas gangrene serum and sulphapyridine should be given.

The rectum may also be injured by foreign bodies which have been swallowed, *e.g.*, tooth plates, small bones or pieces of shell or claws of shellfish, or, again, by those formed in the body, such as gall-stones or enteroliths. Sharp foreign bodies are usually driven into a crypt of Morgagni during defæcation and turn over so that they lie horizontally across the lumen at the anorectal junction. The symptoms are typical. During defæcation sudden intolerable pain is experienced in the anal canal, and in spite of complete rest it continues without cessation. Immediate relief follows the removal of the foreign body, but careful watch must be kept for five days lest ischiorectal inflammation or a fissure-in-ano result.

### PROLAPSE OF THE RECTUM

Prolapse of the rectum may be complete or incomplete. Incomplete prolapse is the commoner and consists in the protrusion of a cuff of mucous membrane beyond the anal margin. In complete prolapse the whole thickness of the rectal wall protrudes, and two degrees are described; in the first the peritoneum is unaffected, while in the second the lowest part of the pouch of Douglas is drawn down between the prolapsed layers.

Prolapse in children is a common occurrence. The underlying weakness of the muscles is always an indication of a debilitated condition following illness or malnutrition. Accessory factors are those which cause undue straining, such as diarrhœa, constipation, thread-

worms, rectal polypi, whooping-cough, chronic bronchitis, phimosis or vesical calculi.

Prolapse in adults is common in women as a sequel to the weakening of the pelvic muscles during childbirth. In men it is a symptom of local disease, *e.g.*, hæmorrhoids, rectal polypi, carcinoma of the rectum or enlarged prostate.

The *diagnosis* is made by defining the continuity of the prolapsed mucous membrane with the anal skin. The protruding apex of an intussusception or a prolapsed rectal polyp alone can cause confusion.

*Treatment* should be directed towards the cause. Simple local treatment is certain to fail unless the predisposing factor has been removed. Small children should be made to pass their motions lying on their side, after which the prolapse is replaced and the buttocks firmly strapped together. In women whose rectal prolapse is merely a part of a general weakening of the pelvic floor, perineorrhaphy and colporrhaphy will cure the rectal condition. In other cases partial prolapse and minor degrees of complete prolapse may be cured by four linear cauterisations of the mucous membrane in its long axis. More severe cases are treated by injection of sodium morrhuate or a solution of quinine into the ischiorectal fossæ and into the hollow of the sacrum. Cases with a very patulous anus are treated by a plastic operation upon the external sphincter behind the bowel.

### INFLAMMATORY DISEASES OF THE RECTUM

**Proctitis** is due to the same causes as colitis, and the two conditions may coexist. Catarrhal proctitis is due either to downward spread of a mucous colitis, irritation caused by thread-worms or bilharzia, chronic constipation, injury from scybalous masses, foreign bodies or hæmorrhoids. It is invariably present in conjunction with carcinoma of the rectum. Dysenteric proctitis is secondary to either amœbic or bacillary dysentery in the colon. Gonococcal proctitis occurs in women owing to the spread of infection from the vulva, and in men from sexual perversions. Syphilitic and tuberculous proctitis are ulcerative in type (see below).

The *symptoms* are pain in the perineum and in the pelvis, sometimes referred down the thighs, tenesmus and mucoid diarrhoea with pruritus or excoriation of the anal skin.

*Treatment* is directed toward the cause. Local treatment consists in rest in bed, hot hip baths twice daily and irrigation of the rectum with a warm solution of 1 : 10,000 silver nitrate or of sulphapyridine. Very mild aperients, such as liquid paraffin, are to be used.

### FISSURE-IN-ANO

**Fissure-in-Ano** is a narrow, elongated ulcer at the mucocutaneous junction, lying within the sphere of action of the subcutaneous segment of the external sphincter muscle, resulting from the tearing of the mucous membrane by a hard fragment of fæces or by a foreign body. It is

narrowly triangular in shape, its apex reaching Hilton's line, its lower limit the true skin at the anal margin, while its long axis is at right angles to the fibres of the external sphincter. It usually lies in the middle line of the posterior surface of the anal canal or a little to its right or left. Usually superficial, it may expose the fibres of the external sphincter in its base.

It cannot be seen until the margins of the anal opening are separated, when it appears as a purple-coloured ulcer with thin edges and a few weakly granulations. Its lower limit is often overlapped by a small fold of torn-down skin, known as a "sentinel pile." It is exquisitely tender and examination is difficult.

*Symptoms.*—Severe pain is experienced when the bowel is emptied, and continues for five to fifteen minutes in early cases but may last for two hours in long-standing ones. A little blood and mucus may be noticed.

*Treatment* is palliative and operative. Palliative treatment should be reserved for small recent fissures which have not exposed the fibres of the external sphincter. Constipation must be overcome, so that a regular soft stool is voided daily. At stool, wool is used instead of paper and the parts are afterwards washed with soap and water, dried and anointed with a mild mercurial ointment.

Treatment by injection has become the standard method and rarely fails to bring about healing. Gabriel claims that recurrences are probable afterwards, but this has not been our experience. Ten cubic centimetres of proctocaine are injected in such a way that the tissues immediately subjacent to the fissure are infiltrated and then the posterior third of the circumference of the subcutaneous external sphincter is injected.

Operative treatment consists in removing the sentinel pile, excising the fissure and dividing the fibres of the muscles at right angles to their long axis, so that it is temporarily thrown out of action and the ulcer thus enabled to heal. The sphincter may also be paralysed by stretching, but this should never be done except by the expert, as incontinence has been known to follow overstretching. The healing process is accelerated by the use of infra-red radiation after operation.

## ULCERATION OF THE RECTUM

1. **Dysenteric Ulcers.**—In dysentery the mucous membrane becomes congested and oedematous and numerous small ulcers form. These rapidly coalesce to produce a large ulcer with a sinuous margin and smooth floor. The condition may lead to perirectal suppuration and ischiorectal abscess, and later to stricture.

2. **Tuberculous Ulcers** are found usually in the crypts of Morgagni in sufferers from other forms of tuberculosis, especially in the lungs. The ulcer itself is typical, having pale undermined edges and greyish weakly granulations, but in the rectum it is surrounded by a ring of unusually firm satellite tubercles, which give an induration rarely met with in other tuberculous lesions. Ischiorectal abscess and fistulæ are very common sequelæ.

3. **Syphilitic Ulcers** are not so frequent as was previously believed and as is still taught by the French pathologists. The primary chancre is occasionally seen at or just within the anal orifice in both sexes. It is a painless, indurated ulcer accompanied by painless, hard and discrete glands in the inguinal regions. Condylomata are a common secondary manifestation. Gummatous ulcers are seen at the anal margin, where they present the typical punched-out appearance. In the anal canal and lower end of the rectum a massive induration can occur with one or more gummatous ulcers ; this type tends to stricture formation later.

*Symptoms.*—Rectal ulceration occurs after 35 years of age, in women more frequently than men. The symptoms depend more on the situation of the ulcer than on its cause, for all varieties produce a nearly identical picture. The higher the ulcer, the less is the discomfort. Those near the sphincter give severe pain, tenesmus, diarrhoea and a rapid deterioration of the general health.

The patient will first notice that immediately on getting out of bed in the morning there is an urgent desire to empty the bowel, but a disappointingly small stool of thin, watery mucus is passed without much relief. Tenesmus continues and only after several attempts will a satisfactory faecal stool be evacuated, and comparative comfort assured for the rest of the day. Later on, as the ulcer extends, there will be a constant dull, aching pain, with tenesmus persisting throughout the day. The constitutional effects are serious, the patient being mentally distressed as well as physically weak.

*Treatment.*—The underlying cause of the ulceration must be energetically treated. Specific treatment, if applicable, will do more good than local measures, which latter are directed chiefly to the relief of symptoms. The patient must be put to bed and the action of the bowel regulated so that a soft well-formed stool is passed daily. Irrigation with warm boracic lotion, or with a 1 : 10,000 silver nitrate solution, brings considerable temporary relief. Single ulcers may be scraped or excised after stretching the sphincters, but in severe cases a colostomy is required to keep the rectum clean before the ulcer will heal.

A careful watch must be kept for such complications as ischiorectal abscess, fistula or stricture.

### STRICTURE OF THE RECTUM

Stricture of the rectum is due to cicatricial contraction, which may result from any of the following causes :—

1. **Congenital.**—These defects have been dealt with (p. 647). Congenital narrowing at the level of the cloacal membrane may pass unnoticed until late in life, when chronic constipation and difficult defaecation cause patients to seek advice. Examination reveals a narrow ring within the anal canal, the margin of which may show one or more fissures.

2. **Inflammatory.**—Rectal ulceration due to dysentery, tuberculosis, gonorrhoea or syphilis may lead to stricture.



3. **Traumatic.**—Penetrating wounds and operations for prolapse or hæmorrhoids (*e.g.*, Whitehead's) are sometimes followed by contraction of the scar. Those following radium treatment come in this category.

4. **Neoplastic.**

5. **Pelvirectal cellulitis** and **abscess** resulting from puerperal sepsis may lead to a horse-shoe arrangement of indurated tissue around the bowel.

The stricture is either annular or tubular; carcinoma gives a fibrous ring-constriction, while dysentery is followed by tubular thickening over a considerable length of rectal wall. It is more common in women than men, and occurs mostly between the ages of twenty-five to forty-five years. The bowel above the stricture is distended and hypertrophied and the mucous membrane is in a state of chronic inflammation with small stercoral ulcers. The state of the gut below depends on the site of the obstruction; when the stricture is high up the rectum is thin-walled, atrophic and "ballooned," with internal hæmorrhoids prominent, whereas low strictures cause weakening of the sphincters with incontinence and prolapse of mucous membrane.

*Symptoms.*—There will be a history of the causative disease. High strictures give the picture of chronic intestinal obstruction, *i.e.*, gradually increasing constipation with alternating attacks of "spurious diarrhoea," the passage of blood and mucus, but an absence of pain. In the low stricture pain is severe, control is lost and blood and mucus escape apart from defæcation, prolapse of the mucous membrane and internal piles being present. If scybalous masses collect above the stricture, frequent attacks of diarrhoea occur without affording any relief, while later the obstruction may become complete and the symptoms and signs of acute intestinal obstruction appear.

The stricture is easily located by the finger or the protoscope, and its nature is determined by its appearance and its history.

*Treatment.*—If the causative disease is still active, appropriate measures must be directed to it, and if the stricture is due to carcinoma, radical removal must be advised.

The treatment of the fibrous stricture is not satisfactory. Gradual dilatation with gum-elastic bougies should be tried first. If the stricture yields, bougies must be passed and retained in position for five minutes every day for a fortnight and then on alternate days for one month, after which once a month for a year should suffice.

Internal proctotomy consists of one or more linear incisions into the stricture from inside the rectum. This is a dangerous procedure owing to the liability to both intramural and perirectal inflammation. It should be reserved for annular constrictions within  $1\frac{1}{2}$  in. of the anal opening. External proctotomy is a better method and should be used for all tubular strictures, the rectum being exposed from behind and the stricture divided completely in the midline posteriorly. A certain number of patients will need to have a colostomy performed as the only method of overcoming the obstruction.



### PERIRECTAL AND PERIANAL ABSCESSES

Abscesses around the rectum and anal margin are of common occurrence in adult life, males being affected more frequently than females. The infecting organisms tend to be of low virulence and the pus spreads along the paths of least resistance to reach either skin or mucous membrane, so that sinuses or fistulæ are likely to follow. The common infecting organisms are *Bacillus coli*, staphylococci and streptococci, usually in mixed culture, and in a certain number of cases tubercle bacilli will also be identified. Very rarely gas-forming organisms are responsible for a fatal form of perirectal infection.

**Perianal Abscess** differs in no way from a simple boil and only assumes importance owing to its tendency to burrow and so lead to a superficial sinus or an ischiorectal abscess. It follows infection of a hair follicle, sebaceous gland or thrombosed external pile: an abrasion from riding a horse or a bicycle or from rowing may be a predisposing factor.

These abscesses occur at or near the anal margin and give rise to pain and irritation, which are made worse by sitting and walking. They appear as small, red, shiny and fluctuating swellings which are very tender. As defæcation is painful, the patient is constipated.

*Treatment.*—Early incision is needed to prevent sinus formation and burrowing into the ischiorectal fossa. A T-shaped incision is made and the edges trimmed away, pus evacuated, necrotic debris scraped out and the cavity lightly packed with paraffin and flavine gauze. The patient must be kept in bed until healing is well advanced, and the period is sensibly shortened by infra-red radiation.

**Submucous Abscess** forms beneath the mucous membrane of the lower part of the rectum, and follows trauma by a foreign body or hard fæcal mass, or an ulcerating internal pile or polypus. Pus forms on one side wall of the rectum and does not spread round the bowel but tracks downwards towards the external sphincter, where it will burst through, leading to an internal sinus.

The patient complains of dull throbbing pain inside the bowel, which is greatly aggravated during defæcation, but which is relieved when the abscess bursts. It is immediately recognised by a digital examination as a soft, fluctuating and tender swelling projecting into the lumen of the bowel.

*Treatment.*—The sphincter must be dilated under general or low sacral anæsthesia and the mucous membrane incised in the whole length of the abscess cavity, which is lightly packed with paraffin and flavine gauze. The patient must be kept in bed for at least a week and the bowel should not be allowed to act for four days.

### ISCHIORECTAL ABSCESS

The ischiorectal fossæ lie on either side of the rectum and communicate with each other behind, but are separated in front by the genital canal in each sex. The boundaries of the fossæ are (1) above and internal—the

levator ani muscles; (2) below and internal—the external sphincter muscles; (3) external—the obturator internus muscle; (4) below—the skin of the anal region; (5) above—the junction of the levator ani and obturator internus muscles. At the apex of the fossa in front a small cul-de-sac rests on the triangular ligament (Fig. 325).

**An Ischiorectal Abscess** can be a complication of every type of rectal inflammation and ulceration, but the usual form is the result of minor septic conditions in and around the anal canal, *e.g.*, infection of crypts of Morgagni, fissures, perianal abscesses and hæmorrhoids. Organisms are carried to the fossa by the lymphatics, and infection soon spreads throughout it owing to the poor resistance of the fat. If the abscess is not opened, pus tracks behind the rectum and invades

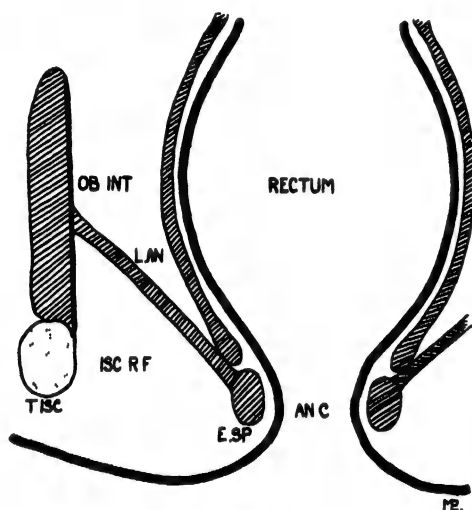


FIG. 325

Diagram showing the relations of the ischio-rectal fossa. Reference to the text will make the diagram perfectly clear.

the opposite fossa, giving rise to the "horse-shoe abscess"; pus also burrows towards the rectum and finding its weakest point—the gap between the internal and external sphincters—bursts through into the bowel. In this way a fistulous track is established as soon as the skin is incised or eroded. Certain of these abscesses are tuberculous and are slower and more insidious in their onset and progress.

*Symptoms* are of acute onset. Pain, which at first is a dull ache, becomes severe and throbbing and is greatly increased by sitting, walking and defæcation. So painful and tender do the parts become that the patient cannot find relief in any position. A red, hot and

tender swelling appears in the ischio-rectal region, and a rectal examination reveals a tender, fluctuating mass which does not bulge into the bowel. All symptoms disappear when the abscess bursts, but as the opening is usually small, they return as the pus collects again. Retention of urine is a common complication.

*Treatment.*—The danger of a fistula forming is so great that the abscess must be opened with the least possible delay. A crucial incision is made and the right-angled corners are snipped away. A finger is introduced to break down all loculations and a strip of rubber tissue introduced. The patient must be kept in bed till healing is well advanced. The cavity is lightly packed daily with paraffin and flavine gauze and irradiated with the infra-red lamp. The skin must not be allowed to heal until the granulation tissue from the healing cavity is flush with the surface.

**Pelvirectal Abscess** lies above the levator ani in the connective tissues, continuous with those of the pelvic cellular tissue planes. It is not usually the result of rectal disease, but follows infections of

the bladder and of the female genital organs (*e.g.*, puerperal pelvic cellulitis). Appendix abscesses and diverticulitis may also lead to pelvirectal abscesses.

The *symptoms* are those of the causative disease and of the pelvic cellulitis. The actual abscess is usually discovered during a rectal examination in these very worrying cases, or when it bursts into the rectum. In late cases, the pus may track and present above Poupart's ligament.

*Treatment* depends upon the cause. The abscess should be opened either through the rectal wall or, in women, through the posterior fornix of the vagina. In spite of the gravity of the illness, it is wise to wait for a localised collection to form before operating.

### SINUS AND FISTULA-IN-ANO

The imperfect drainage of perianal and perirectal abscesses will lead to the formation of tracks lined by pyogenic granulation tissue. Healing is prevented by the constant muscular movement in this region and, if the track communicates with the bowel, a permanent source of reinfection is established. The term *fistula* denotes a track opening at one end on the skin and at the other on the mucous membrane. Those tracks which open at one end only are *sinuses*, and the old term of blind fistulæ will be discarded.

**An External Sinus** follows a perianal or ischiorectal abscess which has been opened on the surface and which has failed to heal. A small opening can be seen and pus may be expressed from it. It is by no means common.

**An Internal Sinus** follows a submucous abscess and is still less common. It can be identified as a narrow, elongated area of induration in the rectal wall with an opening at its lower margin, from which pus can be seen oozing.

**Fistula-in-Ano** is more common than it should be. It results from ischiorectal or pelvirectal suppuration. The opening into the bowel may have occurred before the patient seeks advice, and the skin incision into a supposed simple ischiorectal abscess merely completes the fistula, which may only be discovered later when the wound refuses to heal. The etiology is precisely similar to that of perirectal inflammation, and men are much more frequently affected than women. The possibility of the infection being tuberculous must always be borne in mind.

The track may take a direct line from the bowel to the skin, or it may be tortuous or even branched. A well-recognised example of the complicated variety is the *horse-shoe fistula*, in which the track passes round the rectum behind from one fossa to the other, the external opening being on the opposite side to that into the rectum. Three types of fistula are described: (A) the submuscular or subcutaneous fistula runs from skin to anal mucosa and is below the level of the external sphincter; (B) the intermuscular fistula is the commonest of all, the track reaching the rectum between the internal and external sphincters; (C) the supramuscular—the rarest and most serious—

enters the rectum high up above the levator ani, through which the track passes (Fig. 326).

The *symptoms* are discomfort due to the leakage of pus and fæcal-stained mucus, and intermittent attacks of pain and tenesmus, when pus collects under tension.

*Treatment* consists in a complete opening-up of the track in the whole of its extent, together with any ramifications. When it is a simple direct tunnel, a malleable probe-pointed director is passed up it and made to project into the rectum, the point then being brought out of the anal orifice. The director is carefully arranged so that it passes exactly at right angles to the long axis of the fibres of the external sphincter, and the track is laid completely open. The walls are curetted with a sharp spoon, or if very indurated are excised, and the wound is packed with paraffin and flavine gauze and compelled to heal from the bottom.

Horse-shoe fistulæ are more difficult to deal with, but the same general principles apply. The external sphincter is divided at the point where the track passes over it to enter the rectum. It should be divided only in one place, and never under any circumstances in more than two.

The supramuscular fistula, which traverses the levator ani, cannot be treated in a similar manner, because incontinence inevitably follows the division of both sphincters and part of the levator ani. A two-stage

operation is performed, the upper part of the track above the levator ani being opened, scraped and plugged, and at a later date the track below the levator is laid open. The prognosis in these cases is not good.

In all cases healing can be obtained and recurrences prevented only by the most rigorous after-treatment, in which the dressings are painful and tedious and may easily be done inefficiently. Every day and after each motion the wound is cleansed and packed with gauze soaked in paraffin and flavine or red lotion. The bowel must be confined for four days after operation, when a soft formed stool must be arranged for by suitable aperients. Patients must be kept in bed until healing is complete. This may be greatly accelerated by the use of the infra-red lamp.

Complete excision with suture can be used in simple direct fistulæ and in many cases of tuberculous infection.

**Fistulæ into other Organs.**—Communication between the rectum and bladder, vagina, male urethra, Fallopian tubes and appendix may follow infection or malignant new growth of these structures.

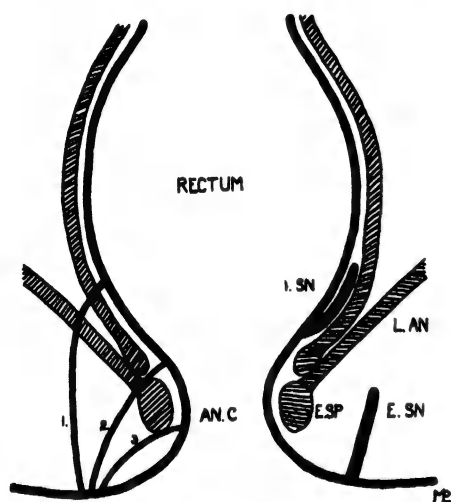


FIG. 326

Diagram illustrating on the right side an internal and an external sinus (I.S.N and E.S.N); on the left side the three types of fistula-in-ano, indicated by the figures 1, 2, 3.

*Treatment* is directed towards the cause and a colostomy may have to be considered.

### HAEMORRHOIDS

**Internal Piles.**—The lower end of the rectum is one of the chief sites of venous anastomosis between the portal and systemic circulations. The superior hæmorrhoidal veins, which are devoid of valves, pass into the portal system, whereas the middle and inferior hæmorrhoidal veins join the systemic circulation. This anastomosis under certain conditions becomes the seat of varicose dilatation, in this way establishing the condition known as internal hæmorrhoids or piles.

Each internal pile, therefore, consists of a nodule of varicose veins and one of the terminal branches of the superior hæmorrhoidal artery, which are surrounded by a sparse amount of connective tissue and covered by rectal mucous membrane. One internal pile or more may be present, but in well-established cases three major piles will always be found, one situated anteriorly and the other two postero-laterally. Internal piles cannot be felt with any certainty on digital examination, but are seen through the proctoscope as purple projections from the lower rectal wall, which grow steadily larger and more engorged for the first few moments after the speculum is introduced.

Internal piles are seen in both sexes between the ages of twenty and sixty-five years, and are due to many causes which may be classed in four groups.

1. Idiopathic hæmorrhoids occur in people with no local or general disease and are due to a familial predisposition, as are other forms of varicose veins.

2. Constipation is the commonest cause owing to the pressure of inspissated fæcal masses on the valveless veins.

3. Straining due to enlarged prostate, urethral stricture, etc.

4. Back-pressure in the veins, *e.g.*: (a) in the rectal wall, by carcinoma of the rectum, inflammatory and ulcerative diseases and strictures; (b) in the portal vein, from cirrhosis of the liver, heart disease and splenomegaly; (c) in pelvic congestion, from the gravid uterus, uterine and ovarian tumours and diverticulitis.

The complications to which internal piles are liable are thrombosis, phlebitis, periphlebitis, ulceration and strangulation.

*Symptoms.*—These are hæmorrhage, mucous discharge, prolapse and pain.

**Piles of the First Degree** do not come outside the sphincters. These will cause the appearance of a few drops of fresh blood with the passage of each motion. There are no other symptoms, and if the motions are kept soft and regular there may even be little or no bleeding.

**Piles of the Second Degree** come down with each act of defæcation, but return spontaneously or can be easily replaced by the patient. In this stage the piles are well-formed polypoid tumours with a broad base, and bleeding becomes a prominent symptom. When the piles have come out, their bases are gripped by the external sphincter and intense venous congestion results. As long as they remain out, venous

blood steadily oozes from them, but the bleeding ceases as soon as they are returned into the rectum. If the surface is ulcerated, bright arterial bleeding may be detected and this tends to continue for a time after the piles have been replaced, in which case blood collects and will be passed later, either alone or with a fæcal motion. The hæmorrhage in second degree piles is often sufficient to cause a marked degree of secondary anæmia. Moderate pain or discomfort is present and some mucus will be passed.

**Piles of the Third Degree** are combined with a lax sphincter and a state of chronic partial prolapse is found. As a result of constant irritation and infection, the mucous membrane covering the piles is converted into transitional or squamous epithelium. Bleeding is trivial or non-existent, but the mucous discharge is profuse and leads to irritation and pruritus ani. Pain both locally and in the back is pronounced, and patients suffer considerable mental distress and will spend much time daily in a futile effort to replace the prolapsed piles.

*Treatment.*—No treatment should be considered until it has been proved that the piles are idiopathic and are not due to any local or general disease. It cannot be emphasised too strongly that internal hæmorrhoids are frequently not a disease, but merely a symptom of other pathological lesions. There can be no excuse for the treatment of hæmorrhoids, while an operable carcinoma of the rectum passes unnoticed.

*General Treatment.*—If the cause can be effectively treated, the piles will disappear in due course. In pregnancy piles give trouble in the earlier and the later months, but no treatment should be advised, unless they are causing secondary anæmia; in cirrhosis of the liver and in other causes of portal obstruction, local treatment may be undertaken if the piles are causing real distress, but the patient must be told that the relief is temporary and that recurrence is to be expected within six months.

Constipation will need attention in every case. The weakest dose of a mild aperient, which effects a result, should be aimed at, good examples being liquid paraffin, phenolphthalein, senna or one of the preparations of biliary and intestinal extracts, such as eulaxase or taxol. A careful regulation of the diet will also tend towards a satisfactory result.

*Local Treatment—A. Palliative.*—Mild first degree piles may need no attention beyond regulation of the bowels and the use of suppositories or ointments containing gallic acid, tannic acid, ichthyol or witch hazel.

*B. Injection.*—Other piles of the first and second degrees should be treated by injection. This method has great advantages, being reliable and safe in careful hands, needing no anæsthetic, and producing little or no pain. Further, it can be done in the consulting room with no loss of time from work for the patient. A special syringe with a long narrow barrel, a grooved speculum and a good headlamp are required. The sclerosing fluid is injected into the base of the pile and not into its most prominent part, and great care must be taken to ensure that the needle is introduced into the rectal mucous

membrane and not into the anal skin. The following preparations are suitable for the purpose :—

R	Ac. carbolici	.	.	.	.	3i.
	Zinci chlor.	.	.	.	.	gr. i.
	Ol. olivæ	.	.	.	.	3v.

5 to 8 minims into each pile.

or

R	Sodii biboratis	.	.	.	.	3i.
	Ac. salicyl	.	.	.	.	3i.
	Glycerini	.	.	.	.	3i.
	Ac. carbolici	.	.	.	.	3iiij.

5 to 8 minims into each pile.

At the first treatment it is wise to inject one pile only and to observe the reaction. On subsequent occasions two may be safely injected unless the patient has had a marked reaction to the first injection. The contraindications to injection are thrombosis, ulceration and strangulation. The method must never be used for external piles.

**C. Operative.**—Operation is needed in those cases which are not suitable for injection. Piles of the third degree may be improved temporarily by injection, but will eventually come to operation. The method generally practised in this country is that of “ligature and excision.” The three main piles are removed with the redundant folds of anal skin, no attempt being made to remove any lesser pile lest a fibrous stricture follow.

*Complications* are strangulation, thrombosis and ulceration.

1. **Strangulation.** A patient, who has hitherto been able to replace the prolapsed piles after each motion, finds that the mass cannot be pushed back. The use of lubricants and the prone position fail to bring about reduction, and the feeling of congestion and discomfort rapidly passes into severe pain, from which no relief is obtainable. Examination reveals a violet mass of intensely congested and swollen piles, firmly gripped above by the sphincter and projecting beyond the anal margin. Gangrene may follow in some cases.

*Treatment.*—The patient must be put to bed, the end of which is raised on blocks, and hot compresses applied to the prolapsed mass. An injection of morphia and atropine is given. Within four hours an attempt is made to replace the piles. In a small number of cases this can be done without an anæsthetic, but the pain, tenderness and spasm are so great that a general anæsthetic is usually necessary. The prolapsed mass is replaced and the sphincter stretched. The patient is kept in bed till all swelling has subsided, after which removal of the piles should be advised.

2. **Thrombosis.** An internal pile can become inflamed as the result of phlebitis and periphlebitis. Patients complain of pain, tenesmus, a mucous discharge and some œdema of the anal margin. The condition lasts a few days and is usually followed by a general improvement, as the affected pile will shrink up as the thrombus organises.

3. **Ulceration.** Strangulated and thrombosed piles may become ulcerated, when pain and tenesmus are more prolonged than in simple



thrombosis. Patients should be kept in bed, the bowels carefully regulated and suitable suppositories or ointment applied locally. An excellent ointment consists of

R	Ung. ac. tannici . . . . .	} āā ʒi.
	Ung. stramonii . . . . .	
	Ung. belladonnæ . . . . .	

**External Piles.**—A TRUE EXTERNAL PILE consists of a varicose perianal vein contained in a redundant fold of skin. This may be present independently of, or coexist with, internal piles. It gives rise to no symptoms until it becomes “thrombosed,” when the vein ruptures as the result of some severe strain and a clot forms in the fold of skin. This thrombosed external pile presents at the anal margin as a tense, spherical, dark-blue swelling, which is so exquisitely painful and tender that the patient can find no relief.

*Treatment* is incision under a local anæsthetic, the clot being shelled out. Healing is rapid.

A FALSE EXTERNAL PILE consists of a redundant fold of skin without any venous component. Several of these tags may be present, and give no symptoms except a little local irritation.

**Pruritus Ani.**—Pruritus ani is a condition of itching of the skin surrounding the anus, and in women is confluent with pruritus vulvæ. The itching may become so intense that the patient’s life is made an almost intolerable burden and the general health suffers from constant irritation and sleeplessness. It is always worse at night and in hot weather, but in later stages is constantly present. Although it may be an indication of general disease, *e.g.*, gout or diabetes, it is usually a symptom of rectal disease, and no case must be diagnosed as idiopathic until a complete examination has proved the rectum normal.

In early stages the skin is dry, wrinkled and powdery white in colour, but in many cases patients have scratched themselves so vigorously that œdema and surface ulceration are present. Treatment is directed to the cause, and if none is found, the condition is likely to be very intractable. X-ray therapy often achieves a cure, and the following ointment is of value :—

R	Menthol . . . . .	gr. xv.
	Plumbi acet. . . . .	gr. v.
	Calomel . . . . .	gr. iii.
	Paraff. molle . . . . .	ad ʒi.

Gabriel advises extensive subcutaneous injections of proctocaine all round the anal region, especially posteriorly.

### NEW GROWTHS OF THE RECTUM

The growths of the rectum are :—

Benign—

Epithelial—adenoma.

Connective tissue—fibroma, lipoma, hæmangioma.

Malignant—

Carcinoma and sarcoma.

Carcinoma of the anal margin.



**Adenoma.**—The benign tumours, of which adenoma is by far the most common, are frequently referred to as rectal polypi, and this term includes any of these growths which have a pedicle and are covered with epithelium (Fig. 327).

The adenoma occurs at any age but is especially frequent in children under twelve. It is composed of glandular acini lined with columnar epithelium and its surface is either smooth, fissured or deeply furrowed. As it increases in size its pedicle becomes longer



FIG. 327

Microscopic drawing of an adenomatous polyp. The high columnar epithelium with goblet cells is well shown.

and narrower, until, although its point of attachment is several inches up the rectal wall, the tumour may project through the anal opening. Viewed through a speculum it appears as a soft, red mass projecting into the lumen of the rectum.

Multiple adenomata are sometimes seen in the form of soft, reddish growths with a fissured, warty surface, either scattered diffusely over the rectal mucous membrane or covering large confluent areas. They are called diffuse papillomatous adenomata. It is probable that such a condition must be regarded as precancerous.

Some hypertrophic adenomatous conditions in the rectal mucosa are not truly neoplastic, but are due to chronic irritation or infection, an example of which is that caused by rectal bilharzia.

*Symptoms.*—The simple pedunculated adenoma may give no symptoms for some time, but ultimately there is a sense of discomfort

or irritation, tenesmus, hæmorrhage and prolapse. In the multiple type the symptoms are more severe and hæmorrhage will cause a profound secondary anæmia, while pruritus ani will result from mucous discharge.

*Treatment.*—The single pedunculated “polyp” should be removed by placing a ligature round the base of its pedicle and dividing it with curved scissors. Broad-based papillomatous growths are excised with the diathermy knife, while diffuse multiple adenomata may possibly require removal of the rectum in exceptionally severe cases.

**Fibroma**, arising in the submucous coat, and pushing the mucous membrane in front of it as it enlarges, may give rise to a rare form of rectal polyp. None of the other benign connective tissue tumours warrant description.



FIG. 328

An ulcerating carcinoma of the rectum.

### CARCINOMA

Cancer of the rectum occurs in both sexes, males being affected slightly more commonly than females. It is usually seen after the age of 40 years, but it appears to be on the increase in young people. There have been three cases between the ages of 16 and 26 in St Mary's Hospital within the past eighteen months.

*Pathology.*—Naked-eye appearance. Carcinoma of the rectum assumes three varieties: (1) The scirrhus type, in which the growth is of low malignancy and the fibrous reaction well developed. The tumour cells tend to spread round the circumference of the gut and a stricture results. Superficial ulceration occurs, but does not penetrate deeply. (2) The ulcerating type, in which cellular activity is greater and deep ulceration occurs with heaping-up of the edges in a manner comparable to that of a squamous-celled carcinoma of the tongue (Fig. 328). No stricture occurs, but the growth spreads outside the rectal wall at an early stage. (3) The fungating type, in which a polypoid mass projects into the lumen, superficial ulceration and deep invasion both being present (Fig. 329).

Microscopically, the growth is composed of columnar epithelium and is usually arranged in an alveolar and adenomatous manner. Colloid degeneration is common.

The growth spreads by continuity in the rectal wall and to adjacent structures in the pelvis, by lymphatics to the glands in the hollow of the sacrum, to those along the superior hæmorrhoidal artery and to the aortic glands, and finally by the portal vein to the liver. Low rectal growths will invade the anal skin and spread by lymphatics to

the inguinal glands. The late results of direct spread will include fistulæ into the bladder and vagina, and involvement of the peritoneum in the pelvis.

*Symptoms.*—The onset is insidious and the patient may be unaware of serious trouble until the growth is extensive. The symptoms depend to a great extent on the type of growth.

1. The appearance of, and the gradual persistent increase in, constipation will be the predominant feature of the scirrhus growths, together with alternating attacks of "spurious" diarrhœa. There will be some abdominal discomfort with flatulence and slight distension. Some patients pay so little attention to these symptoms, regarding them as an inevitable sign of age, that an attack of acute intestinal obstruction first brings the gravity of the situation home to them.

2. Tenesmus is present in all growths of the lower part of the rectum, but is less marked if the upper half is affected. It is particularly prominent with ulcerating and fungating growths.

3. Hæmorrhage will occur in all growths, but in many cases it is trivial and late in appearance. The passage of large quantities of mucus is present in both ulcerative and fungating tumours.

4. Pain is not a prominent feature. Vague discomfort will be experienced by many patients, but only in the malignant ulcers of the lower part of the rectum will pain be pronounced. In later cases, when the growth has infiltrated surrounding structures in the pelvis, pain from nerve involvement may be very severe, and it may be for "sciatica" that the patient first seeks advice.

5. Hæmorrhoids are so common a concomitant of rectal carcinoma that their presence should serve immediately to direct attention to the possibility of a malignant growth.

These symptoms occur in varying degrees of intensity in all cases, and it must be borne in mind that sooner or later in every case infection occurs and proctitis and periproctitis add their quota to the picture. Rectal carcinoma has no specific syndrome, but presents a story of chronic intestinal obstruction, of rectal ulceration, or of a combination of the two. In any case the history should not fail to lead to an immediate and exhaustive examination of the rectum and colon.

*Examination.*—A digital examination will either reveal stenosis of the bowel, palpate the hard edges of an ulcer or a fungating polypoid mass with an indurated base. If nothing abnormal is found, a sigmoidoscopy and barium enema examination should be performed.



FIG. 329

A fungating carcinoma of the rectum, which may be seen to have grown downwards into the anal canal.

*Diagnosis.*—The obstructive group will need to be distinguished from the many causes of chronic intestinal obstruction, while those with pain, tenesmus and passage of blood and mucus must be differentiated from diseases which produce rectal ulceration.

*Treatment.*—Whereas it will be evident in certain cases from a digital examination that the growth is inoperable, it is not possible to decide that the tumour is suitable for removal on a rectal examination alone. An operation for colostomy must precede every removal of the rectum, and this enables the surgeon to examine the growth from above and to explore the abdominal contents for secondary deposits. Enlarged lymph glands in the hollow of the sacrum are not a contra-indication to operation, but fixation of the growth to the sacrum, prostate or bladder, invasion of the peritoneum or of the pelvic floor, involvement of the lumbar lymphatic glands or of the liver render any radical operation unjustifiable.

Operable growths are dealt with by excision of the rectum, which may be performed in two ways. Lockhart Mummery's perineal excision gives admirable results if the growth is within 4 in. of the anal margin, and has a small immediate mortality. Miles' abdomino-perineal method comprises two procedures, in the first of which the upper part of the rectum is freed and mobilised by an intraperitoneal operation, while in the second the removal of the rectum is completed by dissection from its surroundings in the perineal region. Both operations entail the establishment of a permanent colostomy. A modification of this procedure is the perineo-abdominal method and its latest refinement allows simultaneous attack by two surgeons, one below and the other above. Recently attempts have been made to preserve the anal sphincters and so avoid the need for a colostomy, the cut end of the lower sigmoid colon being brought down and sutured to the rectal stump just above the sphincters.

The treatment of inoperable growths is a matter of controversy. If there is a definite stenosis a colostomy must be performed to avoid the possibility of obstruction becoming acute, but in those growths without stenosis the value of the colostomy is open to question and many surgeons advise strongly against it. It does, however, prevent the passage of faeces over the growth and allows the inflammation of the bowel above and around the tumour to subside. As a result, some patients show a marked improvement in their general health, due to the lessening of toxæmia and the relief from pain. Each case must be judged strictly on its merits and the deciding factor will be the relative comfort of the patient with or without the colostomy. Radium therapy in some patients gives considerable relief, even if only temporarily.

*The Management of a Colostomy.*—Dread of the physical discomfort and the mental distress induced by a colostomy is frequently expressed—not only by patients but by their medical attendants—in the words, "It is better to be dead than to have an artificial opening." Ignorance alone can inspire such an opinion, for a properly trained and managed colostomy needs attention in the morning only and places no restraint on the patient's activities.

Education begins on the 12th day after operation, when the bowel

is firmly fixed and the skin incision healed. In the morning the colon should be completely emptied and washed out. A soft rubber catheter is passed well up into the colon and a pint of warm water is slowly run in; within a minute a profuse action occurs and about twenty minutes later a second smaller one follows. These are passed into a bowl or directly into the lavatory by means of the St Thomas' Hospital colostomy horn (Fig. 330) which is strapped in position by a belt around the waist. The opening is then gently cleaned, dried and powdered, and as soon as the patient gets up a special belt, *e.g.*, the "Agordian" belt, is worn in which no cup and no perineal straps are required. An occasional accident will occur at first, but within six weeks the bowel should be trained to perfect behaviour.

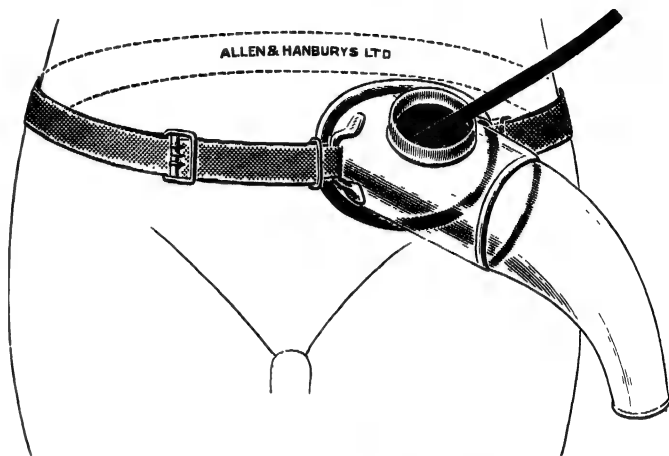


FIG. 330

The colostomy horn shown *in situ* with the rubber catheter entering the upper segment of the bowel for irrigation. (*Allen & Hanburys.*)

Attention to diet will be important, and individual patients will learn by experience what foodstuffs and fluids must be avoided. Colostomy patients are compelled to spend between thirty and forty minutes each morning in attention to their bowel action, but apart from this they should be in no way handicapped or distressed.

**Sarcoma** is a very rare growth in the rectum, being seen in children or in adults past 40 years of age. It is a tumour of the submucous tissue and grows inwards towards the lumen, thus producing single or multiple rounded swellings. Red, soft masses will be seen through the proctoscope, and invasion of the muscle coats occurs. Metastasis is widespread and early.

The *symptoms* are pain, discharge of blood and mucus and tenesmus.

*Treatment* is rarely practicable.

**Carcinoma of the Anal Margin** is a squamous-celled carcinoma, which takes the form either of a warty tumour or of a typical ulcer. The growth is usually slow and enlargement of the inguinal group of lymph glands will be the first sign of metastasis. The appearance of the growth is so typical that it is unlikely to cause difficulty in diagnosis, tuberculous and syphilitic lesions being possible alternatives.

*Treatment* is by excision, and as this will usually need to include the sphincters a colostomy is a necessary preliminary. The inguinal glands should also be removed. Deep X-ray therapy should be employed for inoperable cases.

R. M. HANDFIELD-JONES.

## CHAPTER XXXII

### THE DISEASES OF THE APPENDIX

**S**URGICAL ANATOMY.—The vermiform process or appendix is a narrow tube opening at one end into the cæcum and being closed at the other. Its length varies from 2 to 20 cm., the average being 9 cm., its thickness in the normal state from 3 to 7 mm. The natural contents are mucus and the small quantity of liquid fæces that enters it from the cæcum. The wall of the organ is similar to that of the colon, being composed of like elements. The *serous coat* is normally complete except at the attachment of the meso-appendix, but the normal peritoneal relations vary greatly and any part or whole of the organ may lie extra-peritoneally. The *muscular coat* has an outer layer of longitudinal fibres, continuation of the assembled tæniæ coli, and an inner circular one, these together forming a complete investment except in some appendices, in which muscular defects leave serous and mucous coats in contact. The *submucous coat* is thick and consists of an uninterrupted layer of lymphoid tissue disposed in nodules, which throw the mucous layer into irregular folds. The *mucous lining* resembles that of the colon, being a mucus secreting columnar epithelium. At the cæcal orifice there is sometimes a semilunar fold that has the appearance of a valve guarding the opening.

*Peritoneum, Blood Supply and Lymph Drainage.*—The meso-appendix is a small triangular fold passing from nearly the whole length of the organ to the postero-inferior aspect of the cæcum and to the lowest part of the left leaf of the mesentery. In its free border runs the appendicular artery, accompanied by the vein, passing behind the ileum as it runs from the ileocæcal artery to reach the meso-appendix. Passing from the front of the meso-appendix to the cæcum is the inconstant inferior ileocolic fold, forming when present the anterior wall of the internal ileocolic fossa, a small pyramidal cavity bounded by the ileum above and open to the left. This fold is sometimes named the “Bloodless Fold of Treves,” inappropriately since it usually bleeds when cut. The *lymph drainage* of the appendix passes by channels accompanying the blood vessels to the ileocæcal glands that drain the lower end of the ileum and the cæcum. Sometimes the appendicular lymphatics are interrupted by lymph nodes in the base of the meso-appendix. From the ileocæcal glands the efferents pass upwards to the lymphatic plexus near the head of the pancreas, where there is an intercommunication between the channels from the appendix, duodenum, biliary apparatus and pancreas.

*Anatomical Variations.*—There are many variations in site and disposition of the appendix, as also in the conformation of the adjacent cæcum. Thus its orifice may be so close to the ileocæcal valve that its base is in contact with the ileum as it turns upwards in approaching its termination, there may be a small pouch of cæcum between the appendix and the valve, or the appendix may form an axially placed prolongation arising on a conical base from the mid-point of the cæcal apex. The length of the appendix may be several times that of the base of the meso-appendix, in which case the

organ is apt to be bent or coiled, such normal flexures being at times responsible for obstruction to the lumen or constituting factors in the precipitation of inflammatory disease.

*Its position* may be such that it points *upwards and to the right* behind the cæcum, *upwards and to the left* behind or less commonly in front of the lower ileum, *downwards and to the right* in the iliac fossa, or *downwards and to the left* so that it lies wholly or partly in the pelvis. This pelvic position is of clinical importance; the organ may pass over the pelvic brim from an iliac cæcum or lie wholly in the pelvic cavity if the cæcum is there also. The position of the appendix naturally depends upon the many anatomical variations of the cæcum and on the extent of the developmental rotation of the gut around the superior mesenteric artery. Thus it is not unusual for the cæcum and the appendix to lie in contact with the under surface of the liver or in any site between this position and the pouch of Douglas. The cæcum may be congenitally large or distended by disease, so that the base of the appendix is carried downwards, unless it is fixed in an extraperitoneal situation in the iliac fossa, when the cæcum becomes folded backwards on itself. All the above positions should be borne in mind when disease of the organ is in question, and it is important to remember that about one-fifth of all appendices are pelvic in position.

Finally, the appendix may occupy a small peritoneal pouch, retrocæcal or ileocæcal, the inconspicuous entrance to which may necessitate a little dissection before its display is possible.

*Surface Marking.*—From the foregoing considerations it is obvious that no surface marking can indicate the position of the appendix in any but too vague a way for surgical approach; nevertheless its origin in the cæcum in many cases underlies MacBurney's point, *i.e.*, the junction of the middle and lateral thirds of the right spino-umbilical line.

*Function of the Appendix.*—Like the colon, the appendix absorbs water from its contents and, though the quantity is inconsiderable, no other function is known in man. In disease the water-absorbing function is nevertheless important, because it often results in solidification of the contents with partial or complete obstruction of its lumen. Contraction of the appendix may be seen in radiographic examinations, but where obstruction of the lumen exists, peristalsis may be incompetent to expel the contents into the cæcum, when continued dehydration results in the formation of small scybalous masses named stercoliths (Fig. 331). *Foreign bodies* do not easily enter the appendix unless small or having a motility of their own; thus the only ones found at all commonly are small shot, seasonally in the game-eating classes and usually passed spontaneously, and threadworms which the appendix harbours with the rest of the colon in afflicted persons.

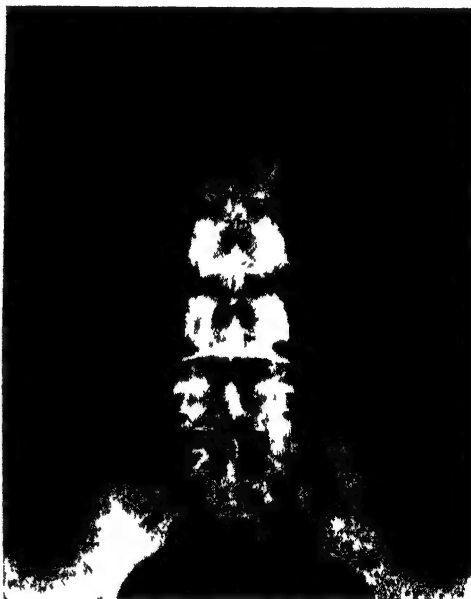


FIG. 331

X-ray showing a large appendicular stercolith, lying free in the peritoneal cavity, after the rupture of a gangrenous appendix.



## APPENDICITIS

By appendicitis is meant an acute or chronic inflammation of the organ.

### ETIOLOGY

**A. Racial.**—Inflammation of the appendix seems to be a disease of modern western civilisation and is found especially in peoples whose nutritional habit is the large consumption of cooked protein food but of relatively little cellulose. Even in such races there are groups where the incidence of the disease is low, of which the recognised examples are the inmates of institutions providing for reasons of economy low protein dietary and greater preponderance of cellulose-containing foods. Thus prisoners, lunatics, orphans and others cared for by Poor Law and some charitable authorities have been instanced as rarely suffering from it. The example of lunatics is noteworthy in connection with the fondness of many of them for swallowing indigestible objects, because small ingested foreign bodies were at one time thought easily to enter the appendix and so to produce disease. It must be remembered that the refinements of diagnosis in appendicitis are at the present time so efficient, that it may be recognised in all its grades from slight congestion to gangrene, and that whereas serious appendicitis is diagnosed straight away, the minor examples that focus the attention of the private practitioner and constitute the greater proportion of the incidence among his patients are less likely to be brought to the notice of the medical attendant in institutional than in private practice. This is certainly the fact among the mentally afflicted, because they can often give no account of their troubles and the diagnosis must be made on physical signs alone. Nevertheless there seems to be little doubt that in Eastern Europe and Asia, where among the country folk the dietaries contain minimal animal protein or none, appendicitis is uncommon, but when the same people give up their natural frugality with emigration or increasing domestic prosperity, the incidence rises to that prevailing among their new associates. A possible explanation of these facts is that the rich cooked food of western civilisation fails to stimulate the colon to its healthy motor activities, the appendix thus failing to discharge its contents, which when solidified become a possible cause of obstruction to its lumen. Appendicular obstruction is nevertheless only one cause of inflammation and is probably not the sole determining factor in the racial incidence.

**B. Age.**—Appendicitis is rare before the age of three years, but it may be seen in infancy sometimes with the slightest of physical signs, so that to deny the probability of its existence in a sick baby is a reprehensibly dangerous attitude. It is also said to be rare in old age, but the old are certainly not exempt, merely less commonly affected than young. Appendicitis becomes common after the age of three years, perhaps because the character of the child's diet begins then to approach that of his civilised parents, and its frequency then increases rapidly until the age of ten years, when it remains an



extremely common disease until the onset of old age. It is said that the end of adolescence coincides with the peak of the incidence.

**C. Sex.**—Unlike constipation, which we are accustomed to assign to a like etiology, appendicitis is about twice as common in men as in women.

All these generalisations are interesting facts, but none of them should be uppermost in the practitioner's mind when attempting diagnosis in an acute abdominal condition.

### PATHOLOGY

Acute appendicitis is due to the entrance of micro-organisms into the wall, the principal germs being the colon bacillus and the streptococcus in any of its breeds. Anaerobes are met with also and may be responsible for the offensive smell of pus from appendicular abscesses, but they are often saprophytic secondary growths in the exudate rather than pathogenic organisms; thus a patient, in whose abdomen there exists a large abscess containing unpleasantly offensive pus, may suffer a relatively slight toxæmia. Organisms may arrive in the appendicular wall by the two routes of direct spread from the contents or from the blood stream. Evidence of the existence of *hæmatogenous infections* is mostly clinical and rarely confirmed by culture, being found, for example, in the sequence of appendicitis following quickly in the trail of upper respiratory infections, so that when influenzal attacks are epidemic so also it may be said is acute appendicitis. Such respiratory infections appear primarily in the lymphoid tissues of the pharynx, and when appendicular metastasis occurs the lymphoid nodules of the submucosa are the sites selected. There are also the much rarer cases in which similarly inflamed appendices occur as incidents in declared septicæmia. *Infection of the appendix from its contents* also affects the lymphoid tissue, though its primary cause is in a large number of cases of an obstructive nature, such as any of the following: (a) there may be a stercolith in the lumen which prevents evacuation of mucus, or much more rarely a foreign body; (b) there may be a sharp bend in the appendix due to the conformation of the peritoneal folds, and if the distal part becomes distended such a bend may become an impassable kink with resulting complete obstruction. With stagnation of the contents and increasing distension of the organ, the contained mucus becomes highly infected and the starting point of inflammation; (c) earlier attacks of inflammation may have produced scarring, constricting the lumen.

In typical examples these two modes of infection result in distinct morbid appearances. In blood-stream infections the whole length of the appendix is commonly swollen or, if part only, the affected portion fades into the normal without sharp dividing lines. The wall is thickened by œdema and exudate is seen on the surface, the organ is turgid and no longer flexible or contractile, yet the mucosa may be little affected in an early case and the contents neither increased nor greatly altered in character. As the disease progresses the mucosa may become as

intensely inflamed as the other coats, so that the condition of the appendix now becomes indistinguishable from one due to the other type of infection, *i.e.*, that coming from the lumen. Such acute blood-borne infections vary in gravity from the mild to the intensely toxic and are often streptococcal. As a sub-group of this variety are those uncommon cases, in which a streptococcal septicæmia settles down in the appendix, the appendicitis then having much the same relation to the generalised infection as has a pyæmic or what is called a "fixation" abscess. Hæmatogenous infection of the appendix may proceed from inflammation to gangrene, necrosis appearing first in the mucosa after the contents have become severely infected, but until this happens the liability to perforation is slight. Nevertheless, peritonitis is common in the absence of perforation, which is not surprising since an inflammation of the substance of the wall may reach the peritoneum before affecting the mucosa. In the blood-borne group there should probably be included those septicæmic cases, where there is an associated phlebitis spreading from the appendicular vein to the ileocolic and possibly even to the superior mesenteric vein.

Appendicitis from infection from the contents is often, but not necessarily, the result of obstruction to normal evacuation, which indeed if not the activating cause may prove a potent factor in determining the evolution of the disease. In a typical obstructive case there is a kink proximal to which, if the obstruction is away from the base, the appendix is normal at the onset and only later affected by spread of inflammation in the wall. The mucosa rapidly becomes congested, then hæmorrhagic and at length gangrenous. The organ distends and its wall becomes secondarily infected, so that at one stage the mucosa may be on the verge of gangrene, but the muscular and serous coats are hardly affected. With the progress of the disease all the coats are inevitably inflamed, and unless subsidence occurs gangrene of the wall initiates perforation with escape of highly infected contents. Such a perforation may occur anywhere, but is most typically seen at a point where pressure of a stercolith has precipitated infective gangrene of the mucosa. It is a curiosity of morbid anatomy that the tip of the appendix habitually escapes until a late stage, though it might have been expected that distension would early cut off the blood supply to the distal extremity. The consequences of perforation are noticed in relation to the effects on the peritoneum (see p. 546); nevertheless a common site for perforation is the attachment of the meso-appendix, in which an abscess then develops often causing localised thrombosis of the appendicular venules. In obstructive appendicitis, when such changes occur but stop short of perforation and then subside, scarring of the appendix will result, the consequences of which will be described among the effects of chronic appendicitis.

#### THE EFFECTS OF APPENDICITIS ON THE PERITONEUM

Inflammation of the appendix affecting the serous coat causes local peritonitis, having the usual characters of fibrinous exudate and fluid

effusion. The character and progress of the peritonitis depend upon the nature of the organism and the route by which infection reaches the serous membrane. Thus sometimes there is little fibrin and consequently little localisation of the inflammation by adhesive peritonitis, such infections being often streptococcal in origin. Though many are of the severest and most dangerous types, others are mild infections which patients are easily able to overcome. At other times there may be a fibrinous exudate that completely seals the infected area, and again many of these are trivial while others are of the greatest severity. In the former cases the sealing process rapidly brings an end to the spread of the disease, and with the subsidence of the appendicular infection the patient effects a speedy natural cure. In the latter it may be that the localisation of the disease by fibrin results in the walling-off of an *abscess*, which once formed may behave in several ways. Thus it may be completely absorbed, it may grow to an enormous size as a localised abscess, the remainder of the peritoneum being unaffected or it may rupture, either into the peritoneal cavity, into a hollow viscus or on to the surface; in the peritoneal rupture, reinfection of the cavity with a new and diffuse peritonitis results. Between the two extremes of watertight sealing and complete failure to localise there are numerous cases of partial sealing, in which the degree of adhesive peritonitis is insufficient to prevent diffusion, and in which as a consequence a partially localised abscess occupies the centre of a diffusing peritonitis. These cases may end either in final complete localisation followed by absorption and recovery or in progressive diffuse peritonitis with all its attendant horrors. It follows from what has been said that, if a distended inflamed appendix bursts when adhesions to its serous surface are already well formed, a localised abscess is the likely consequence, but that with rupture of such an appendix into a peritoneal cavity so far unaffected or the seat of a serous effusion, the results of the entrance of grossly infected appendicular contents are likely to be perilous. Herein lies the danger of obstructive appendicitis as has been clearly pointed out by Sir David Wilkie, and if, as is easily possible, the symptoms of the disease when confined to the appendix itself are undervalued in their importance, the explosion of the highly infected contents into the peritoneal cavity may change the clinical appearances from those of a trivial disorder into the indications of sheer disaster. The general effects on the peritoneum and its contained organs receive reference in the chapter on Peritonitis (p. 546).

The structures that by adhesion may prevent the spread of peritonitis are naturally the other abdominal contents of which the omentum, small intestine, cæcum and ascending colon, mesentery and pelvic organs are all common examples. When the appendix lies behind the cæcum or ascending colon the extraperitoneal fat forms the posterior wall of an abscess, the colon the anterior. Where an abscess has been permitted to undergo spontaneous absorption, there is usually death of some of the omental, epiploic or extraperitoneal fat, which then presents the white appearance of necrotic and saponified fat.

### CHRONIC APPENDICITIS

By this term is meant the clinical state resulting from effects either of recurring mild acute or subacute attacks, or of dysfunction owing to the disposition, peritoneal relations or conformation of the appendix associated possibly with depression of the motor activities of the colon. Nevertheless a true chronic progressive inflammation of the appendix is occasionally seen in which the appendix is thickened, firm, fibrous and of an ivory colour, sometimes by its appearance even suggesting the possibility that the infection may be by the tubercle bacillus, a supposition very rarely borne out by bacterial or histological investigation. In the milder degrees of chronic appendicitis first referred to there are the effects of kinks in producing appendicular pain and what must be called reflex effects on certain sections of the alimentary canal. With grosser change there may be thick fibrous strictures and even complete replacement by fibrous tissue so that the appendix becomes a thin, firm white strand. There is also the effect of sloughing of the mucosa with resulting obliteration of the lumen in one place, so that the distal part of the organ becomes distended with mucus. If the appendicular wall is weak, either from previous localised destruction of the muscle or from a congenital defect, and mucus be secreted under pressure, the mucosa may herniate to form a diverticulum or a mucous cyst, or alternatively may burst with the eruption of mucus into the peritoneal cavity. Mucous cysts form round themselves by irritation thick fibrous walls, whereas mucus bursting into the peritoneal cavity may cause irritative chronic peritonitis in which globules of mucus are surrounded by fibrous tissue and to which the name of pseudomyxoma peritonei has been applied.

### ACUTE APPENDICITIS

#### CLINICAL PICTURE

The symptoms and signs of acute appendicitis may be grouped according to their pathological origins and for their interpretations may be conveniently assessed as due to the following primary factors : (1) The direct effects of acute inflammation of the appendix itself ; (2) the effects on other parts of the alimentary canal ; (3) the effects on neighbouring structures directly involved by local spread ; (4) the effects on the peritoneum ; and (5) septicæmic effects.

**A. The Direct Effects** of acute appendicular inflammation are pain, associated abdominal rigidity and deep tenderness, fever and coated tongue.

1. *Pain* often and typically begins as a generalised upper abdominal or umbilical (*i.e.*, central) one. It is frequently of no great severity at the onset, but may increase greatly with the passage of a few hours. During the course of a few to twenty-four hours it changes its site, settling down in that place where the inflamed appendix lies, thus

often in the right iliac fossa. The surgeon does not often see the patient before this transference of pain, but where he is so fortunate he will usually find that, while there may be slight epigastric tenderness, there is usually tenderness over the inflamed appendix before the pain is unequivocally centred there. The early tenderness that is directly due to inflammation of the appendix is *deep*, is commonly unassociated with any noteworthy degree of cutaneous hyperæsthesia and is situated over the organ. Thus if the appendix lies between the umbilicus and the anterior superior spine, there also is the tenderness. With a high-lying appendix the tenderness is above the umbilical level in the right hypochondrium, or if lowly placed it may be appreciated just above the inguinal ligament or the right pubic bone, according to whether the appendix is lying in the false or true pelvis. The pain may be entirely pelvic and tenderness may then be difficult to discover by abdominal palpation, though there is usually some rigidity of the lower right rectus muscle. Rectal or vaginal examination may be necessary to assess the degree of inflammation and position of the appendix. Wherever it may lie, if the inflammation is of any severity and sufficient time has elapsed for its evolution, there is local rigidity of the abdominal wall of an intensity corresponding to that of the pain, a *rigidity* which increases in degree and extent with the onset of peritonitis. The pain of acute appendicitis is probably associated with distension or turgidity of the organ, and consequently with traction on its mesentery and the parietal peritoneum. It may be constant and increasing from the early hours or it may be colicky in character with intermissions, later becoming constant should the attack fail to subside. It is a common event for such pain after increasing for some hours or even days to become rapidly better, so that the patient thinks that recovery is at hand, when in fact gangrene has supervened with death of the peritoneal coat and rupture of the appendix. Rupture entails peritonitis, but in many cases, as noted in the section on pathology, this long precedes rupture, especially in the cases of blood-borne infection. Disappearance of the pain, unaccompanied by improvement in the patient's general condition or associated with further deterioration, is a serious event indicating the onset of gangrene and imminent, if not actual, rupture. Appendicitis pain varies in intensity and type, not only with the severity of the infection but also with its anatomical, hæmatogenous or obstructive origin. In the early stages of the last variety pain may be slight and colicky and may thus fail to excite the attention which its origin demands in view of the fact that the obstructed appendix is more prone to rupture than the clear one. Herein lies its danger, for the early symptoms of appendicular obstruction with inflammation may be slight and no more severe than the patient has accustomed himself (and unluckily, perhaps, his doctor) to neglect for a few days on previous occasions, when in fact the attacks have been appendicular in origin. Yet rupture at once changes the situation so that the patient rapidly becomes gravely ill from peritonitis, as the result of which the pain returns, but now assumes the characters and distribution typical of peritoneal involvement. In this short account of the pain of appendicitis the

writer has made no attempt to assess the claims of the various theories of origin and nature of visceral pain, but has merely described the well-known clinical manifestations of the diseased appendix.

2. *Fever*.—In mild cases the temperature may rise to  $99^{\circ}$  F. or perhaps not at all, with moderate severity it rises to  $99.4^{\circ}$  or more, and in the infrequent examples of extremely severe infection from the onset it may rise to  $102^{\circ}$  or  $103^{\circ}$  F., such early pyrexia being a serious sign and commoner in hæmatogenous cases than others. With the rise of temperature the *pulse* has an increased frequency according to the intensity of the infection, but in the absence of peritonitis it is not often more frequent than 80 per minute, nor is it of poor volume.

3. *The Tongue* in the early hours of acute appendicitis is a valuable clue to the nature of the disease. When we are attempting to distinguish between the early appendicitis and a mild gastro-intestinal upset, a tongue just dry and coated with a film of fur so thin and transparent that the natural pink of the tongue may be seen through it provides a diagnostic sign of great importance. It is so slight as hardly to be noticeable, unless a knowledge of its value has quickened the practitioner's attention. As the disease progresses, the tongue becomes drier and more thickly coated, and these later changes will be described with the effects of the progress of the disease.

**B. The Effects on other Parts of the Alimentary Canal.**—These may be called reflex, and are as follows :—

1. *Nausea and Vomiting*.—As with any other abdominal catastrophe, vomiting (or nausea) is the rule at the onset. It is often a single vomit and in any case is unlikely to be persistent unless due to one or other of the causes named under headings *C* and *D*. It is frequently absent in patients in whom the disease is mild and also in those who are suffering from an exacerbation of acute inflammation in an already diseased organ.

2. *Colicky Pain* of intestinal origin is often seen and may be difficult to distinguish from appendicular pain, which is itself undergoing recurring exacerbation and remission. Such colicky pain is frequently seen in pelvic appendicitis.

3. *Constipation* is usual when the attack is well established, but earlier there are often one or more actions of the bowel. It is not so complete as to prevent the passage of gas in the absence of more serious complications. It is easily overcome by the injudicious exhibition of aperients.

4. *Diarrhœa* sometimes occurs early with a few actions, but when persistent it is due to one of the causes noted in groups *C* and *D*. Diarrhœa of the reflex type produces normal or fluid stools without either blood or mucus.

**C. Effects on Neighbouring Structures by Direct Involvement.**—

1. *The Urinary System*.—Pelvic appendicitis causes pain and frequency of micturition as soon as the bladder wall is involved in the inflammation. There may be a few red blood cells and pus cells in the urine, but visible pus or blood points to an unfortunate delay in establishing the diagnosis of appendicitis. The right ureter is occasionally involved with similar symptoms.



2. *The Small Intestine.*—Escape of toxic contents from a pelvic appendix may result in dysfunction of the small bowel. Colicky pains are common in neglected patients, and partial obstruction may be due to kinking of the ileum should its coils become involved in the formation of an abscess wall. Between the onset of the colicky pain and the development of obstruction, abdominal distension appears and with it vomiting of an irritative character. If the obstruction is not relieved, the vomiting takes on a regurgitant nature.

3. *The Large Intestine.*—When the colon is affected the usual effect is diarrhoea with the passage of mucus, and if the rectum is involved there is tenesmus with the passage of large quantities of mucus and often of blood. An associated difficulty in getting rid of faeces is due to the simultaneous involvement of the small intestine, and this combination of mucous tenesmus with intestinal obstruction has been named by Sampson Handley “*ileus duplex*.”

4. *The Female Pelvic Organs* may possibly show signs of involvement by the presence of leucorrhœa or menorrhagia.

5. The right iliacus, psoas and obturator internus muscles may go into spasm. As a result the right hip may be flexed or there is pain on or resistance to hyperextension and rotation of the joint.

**D. Effects on the Peritoneum.**—The majority of cases of acute appendicitis show evidence of the existence of peritonitis of some grade, which may be local, diffuse or spreading. The signs of its presence are pain, rigidity and tenderness. Where peritonitis is strictly localised to the serous coat, the pain, tenderness and rigidity are hardly greater in extent than when there is no peritonitis at all, but when this is spreading these three signs affect a larger area of the abdomen, corresponding to the extent of the spread. They are not necessarily seen together in equal intensity and each merits a short individual reference.

*Pain* is severest in the early stages of peritonitis while it is spreading to neighbouring parts of the cavity. It remains intense until the infection has become widespread or until localisation into a well-walled abscess has occurred. In the former event, with the advent of severe toxæmia and other effects of general peritonitis, all pain may disappear, but when it does so the patient is moribund.

*Tenderness* in peritonitis is essentially deep and not a superficial hyperæsthesia, except where the peritoneum of the anterior wall is directly involved, when there appears an intense hyperæsthesia corresponding in extent to the area of involved parietal peritoneum. This is often seen where an inflamed appendix is in contact with the anterior peritoneum, and the commonest example is when it lies low down in the iliac fossa and far out, where the shallowness of the abdominal cavity results in contact with both anterior and posterior walls. Hyperæsthesia may be present in a mild degree with a deeply lying appendix, but it is then overshadowed by the deep tenderness and is not therefore of diagnostic importance.

*Rigidity* is the consequence of peritonitis and, when this is strictly limited to the appendix, rigidity is likewise localised. With diffusion

the rigidity rapidly increases both in intensity and extent. It is often, though by no means always, accompanied by immobility of the belly-wall and also by localised distension of the abdomen due to local arrest of intestinal movement.

The foregoing signs of appendicitis with peritonitis are of the first importance as they give the examiner information as to the exact site of the appendix in all but late cases, as well as the extent of the peritonitis. As regards their value in estimating the position of the appendix, important exceptions are those fulminating cases in which the disease appears to begin with perforation and go on at once to diffuse generalised peritonitis, a variety that may be impossible to distinguish from perforation of a peptic ulcer.

*Other Clinical Effects.*—The temperature rises to 102° F. and does not fall until the patient recovers or his condition deteriorates seriously. The pulse rate quickens with the onset of peritonitis and loses volume. In estimating the degree of the affection, the pulse is probably the most important guide, a poorly sustained small pulse with a frequency of over 120 being of serious import. The tongue becomes dry and thickly coated and in general peritonitis its centre is brown and the edges red and glazed. The face betrays pain and moderate toxæmia at first, but later the typical Hippocratic facies is seen.

Vomiting at some time is the rule. Thus where the attack is initiated by temporary vomiting, with the onset of peritonitis this symptom returns. At first irritative in character, it later becomes regurgitant and is then an indication of a grave prognosis. Local abdominal distension has been noted. General and marked distension indicates the existence of partial or complete intestinal paralysis and is of the gravest significance. Diarrhoea occurring in appendicitis with peritonitis is a sign of some gravity and an indication that operative relief is urgently needed.

Local peritonitis and abscess formation. Peritonitis may be confined to the region of the appendix from the beginning and may proceed to the formation of an intraperitoneal abscess, which appears as a tender swelling. From the preceding account of the pathology, it follows that a more diffuse area of peritonitis with an area of tenderness several inches square may resolve into a much smaller localised abscess, though it must never be assumed that such a subsidence may be surely anticipated. There are certain clinical phenomena that accompany this localisation of a more diffuse peritonitis which are noteworthy. The pain is apt to diminish, the tongue becomes clean and moist and the temperature rises. At the same time the more diffuse tenderness and rigidity of the abdomen, which make it difficult to be certain of the presence of a swelling, give place to an easily felt localised mass.

*E. Septicæmic Phenomena.*—Septicæmia may occur as a feature of most infections, but in abdominal inflammation and especially in appendicitis it tends to assume one well-known form, viz., infective phlebitis of the radicles or the trunk of the superior mesenteric vein, leading perhaps to pylephlebitis and multiple abscess of the liver. Some examples of appendicitis seem to be septicæmic from the



beginning, the infection tending to spread along the veins. Clinically such cases have high fever and frequent pulse, and as soon as the phlebitic process starts a rigor occurs. Tenderness spreads upwards from the appendix, and even before it reaches the liver this organ may have become enlarged. The picture of appendicitis with infective thrombosis is thus a very clear one. It should be particularly noted that in appendicitis rigors are so rare that their occurrence is almost certainly diagnostic of infective phlebitis.

#### CLINICAL VARIETIES OF ACUTE APPENDICITIS

There are a number of common clinical types of appendicitis, made up of the various features described. As regards general severity and progress the following groups may be seen :—

1. Appendicitis with little or no peritonitis, followed by subsidence.
2. Appendicitis with early diffusing peritonitis and later abscess formation.

3. Appendicitis with diffusing peritonitis and partial localisation only. Such are likely to progress to general peritonitis.

4. Appendicitis with general peritonitis.

5. Cases of catastrophic violence of onset, in which the early stages are so rapid in evolution that they can hardly be said to be recognisable, the disease appearing to start with perforation. Such cases may be difficult to distinguish from perforation of a peptic ulcer.

6. Pelvic appendicitis presents a slightly different picture. Pain may be absent from the right iliac fossa and is to be expected above the pubes or even in the left iliac fossa from the spread of infection from the pelvis upwards into the abdomen along the pelvic mesocolon. The tenderness is low down above the pubes and immediately above one or both inguinal ligaments, and is more prominent on rectal or vaginal examination. Upper abdominal pain and vomiting are likely to be persistent owing to the affection of the loops of ileum in the pelvis. Later there will be colicky pain due to obstruction. Still later all tenderness except the rectal may disappear, the patient then presenting the appearance of intestinal obstruction of unknown origin. Careful attention to the history and a thorough pelvic examination should provide the correct diagnosis. In women neglected pelvic appendicitis may cause serious disease of the tubes and ovaries.

7. Retrocæcal appendicitis. An appendix lying behind the cæcum is commonly kinked and thus likely to become obstructed. Preliminary warnings in the form of pain likened to stitch are usual, and with the onset of inflammation, if the appendix is extraperitoneal, not only is tenderness sometimes difficult to discover, but actual abscesses may be overlooked owing to their deep situation.

8. Chronic appendix abscess. An appendix abscess may become chronic, the early phase failing to receive adequate attention. In such patients a tumour develops in the right iliac fossa and a neoplasm of the cæcum or ascending colon is suspected, and a radiological examination may be required to settle the diagnosis.

## DIFFERENTIAL DIAGNOSIS

**Other Gastro-intestinal Diseases** cause some confusion, as for example, enteritis. The condition of the tongue has been referred to. The localisation and extent of the tenderness are suggestive, being more diffuse in enteritis. Pelvic examination must never be omitted. It is especially important to remember that in children the signs may be so slight as to deceive the inexperienced observer. Enteric fever may present difficulty in its early stages.

**Peritonitis Arising from Disease of other Organs.**—Perforation of all hollow viscera present more severe clinical pictures and should not be a source of confusion except in the unusual cases of appendicitis of fulminating onset described above. A consideration of the symptoms usually enables a correct diagnosis to be made, and it should be remembered that a perforated appendix causes a more rapid intoxication with deterioration of the pulse than does the early stage of the perforated peptic ulcer. If the appendix is high in position beneath the liver, it may be confused with the gall-bladder as the site of inflammation, and here the past history and the nature of the onset of the present illness, together with the more persistent vomiting of cholecystitis, will be helpful.

Acute salpingitis provides great difficulty, especially in cases of pelvic appendicitis. The history, the presence of a vaginal discharge, the higher temperature, the later involvement of the tongue, painful and frequent micturition and the findings on vaginal examination should serve to distinguish between the two conditions.

**Peritonitis of Hæmatogenous Origin** is seen in streptococcal septicæmia, and gonococcal infections may be either ascending from the female genital organs or be carried by the blood stream. Pneumococcal peritonitis is the affection with which we are most often concerned, and it is described in Chap. XXVI. It can never be differentiated from appendicitis with absolute certainty, and the diagnosis of pneumococcal peritonitis has too frequently led to the death of the patient from an unrecognised appendicitis. All patients, therefore, in whom pneumococcal peritonitis is suspected must be regarded as possible subjects of acute appendicitis and treated as such.

**Intraperitoneal Hæmorrhage** from any cause, but especially when due to a ruptured ectopic gestation or a Graafian follicle, may give rise to some doubt. A careful assessment of the symptoms and the clinical signs should suffice to make the diagnosis clear.

**Other Swellings in the Right Iliac Fossa.**—The distinction between chronic appendix abscess, carcinoma, ileocæcal tuberculosis and regional ileitis can usually be made by a barium-meal examination.

**Diseases of the Right Kidney** provide the greatest difficulties in the diagnosis of appendicitis. In urinary infection with or without stone the temperature is higher and there will be one or more rigors. The full diagnostic problem is discussed in the section on pyelitis (p. 728); suffice it to say here that in every case in which the pain starts in the side, a renal lesion should be suspected in preference to an appendicular one.

**Pneumonia.**—The variety of pneumonia, which is confused with acute appendicitis, is that which produces severe right-sided abdominal pain and rigidity without appreciable signs in the chest. The diagnosis may be extremely difficult, the quickened respiration rate, the laboured working of the *alæ nasi* and high-placed areas of hyperæsthesia not seen in appendicitis should draw attention to the chest.

#### PROGNOSIS OF ACUTE APPENDICITIS

It is probably true that the large majority of patients suffering from acute appendicitis recover, but that the disease is highly dangerous to life needs no emphasis. In the absence of serious peritonitis the late effects are adhesions and scarring affecting the whole or part of the organ, both of which may lead to kinking, the possible consequences of which have been described. It is rare that the appendix is destroyed by the inflammation, and it may be said, therefore, that a single attack predisposes to others and that with every succeeding attack this predisposition is accentuated.

Where there is peritonitis, recovery occurs either by subsidence or with the formation of an abscess, which is later drained or absorbed. Recovery from diffusing peritonitis is certainly not the rule without operative intervention, and it is in these early cases of spreading peritonitis that early diagnosis and immediate operation have changed the outlook from one of gloom to the prospect of almost certain recovery. Again, the absorption of abscesses cannot be anticipated with certainty, because there are many possible untoward happenings in the process. Of these the most important is the production of a small intestine obstruction due to pelvic abscess, the prognosis of which is always grave.

#### TREATMENT OF ACUTE APPENDICITIS

As a general statement the treatment of disease of this apparently useless but undoubtedly dangerous organ is its removal by operation. Hence it may appear surprising that the management of the various grades and stages of this extremely common disorder is not yet standardised, and that in certain events the relative merits of expectant and operative treatment are still hotly discussed.

**For all Early Cases** it is universally agreed that immediate appendicectomy is the correct treatment, unless operation implies a high risk on account of other considerations, such as disease of the lungs or heart, diabetes, old age or obesity.

**Appendicitis with Spreading Peritonitis without Localisation.**—Again, in these patients it is the universal practice to remove the appendix without delay, except in the presence of such complications as invalidate any operative intervention. It is necessary to stress the overwhelming importance of applying these principles to children without delay.

**Cases with Peritonitis showing Signs of Localisation.**—It is around these patients that discussion still rages. It is certain that a large number will proceed to complete localisation with or without abscess

formation; further, amongst those in whom an abscess forms, a proportion continue to spontaneous recovery by the absorption of the abscess. It is therefore suggested by some surgeons that this process of natural cure should be given free scope and not interfered with by operation, which carries with it the danger of spreading infection to unaffected parts of the peritoneum. They therefore advocate delay, with the removal of the appendix at a later date.

Thus in the practice of those who adopt this expectant method, the first forty-eight hours of the attack are somewhat arbitrarily taken as the safe period for immediate appendicectomy, when they agree that operation should be performed. After the elapse of forty-eight hours they are of opinion that localisation should be awaited. To form a judgment as to the success of this natural localisation the patient must be watched minutely, the most important observation being the pulse. If the pulse is increasing in frequency from hour to hour, then delay is proved to be useless and operation is undertaken. If the pulse is steady and there is no real deterioration in the general condition, all food by the mouth is withheld, no aperient is given, and a continuous intravenous drip glucose saline is administered. Morphia or similar drug is absolutely forbidden lest its action should mask the effects of a progressive peritonitis at a time when operative relief might be successful. This rule must be as strictly observed as that which enjoins the withholding of morphia from patients suffering from an acute abdominal lesion, which has as yet been undiagnosed.

The advocates of delay mention three months as a suitable period to elapse between the subsidence of the abscess and the removal of the appendix. This may frequently prove a safe procedure, but unfortunately there are many difficulties and not a few accidents. The abscess may fail to absorb and produce recurrent pyrexial attacks, or in the female pelvis it may cause intractable suppuration of the adnexa, necessitating their eventual complete removal and finally it may lead to intestinal obstruction. Further recurrence of the appendicitis may be seen during the prescribed three months, and the difficulty of removing the three-month-old inflamed appendix is greatly underrated and may result in the stirring-up of the infection with suppuration of the wound. If the expectant treatment proves a failure, then it cannot be denied that it is the delaying method which is directly responsible for the deterioration of the patient, and further that the life and health of such a patient have been jeopardised by the treatment adopted. Finally, it is generally conceded that expectant treatment can only be carried out with safety under the eye of a surgeon or of his assistant, so that it is impracticable in a large proportion of a surgeon's practice.

The alternative procedure is to operate on every case without delay provided the patient is fit for it. In the writer's opinion (with which the editors are entirely in agreement) the advantages of the expectant treatment cannot be proved to outweigh its manifest disadvantages; further, the drawbacks to immediate operation are not apparent to him for the following reasons: 1. It is said that in an abscess it may be impossible or injudicious to remove the appendix, the cause of the

suppuration. In practice this is quite uncommon, and in any case this is what happens if a long-delayed abscess needs to be opened.

2. It is said that infection may be disseminated by opening an abscess into the general peritoneal cavity. This might be true, but is extremely uncommon. It is obvious that a direct approach to the abscess by a suitable incision is less likely to disseminate infection than a more circuitous one, for example, a midline incision for an abscess in the iliac fossa. Undoubtedly some suppuration occurs in the abdominal wall, but this is rarely serious and is common to both methods.

Before deciding to operate the surgeon will of course satisfy himself that the patient is fit for it. Where there is severe toxæmia with marked dehydration, or the patient is collapsed as a consequence of a recent perforation, resuscitation methods for a few hours are desirable. These include the use of a radiant-heat cradle and the infusion of glucose saline into a vein.

**Operative Treatment.**—The aim of the surgeon should be to remove the appendix and, except in the very long-delayed case, this is always possible for a surgeon of requisite experience. No special anæsthetic is indicated.

The incision should provide the shortest route to the inflamed appendix and should be of no more than adequate length. Several kinds are available and in his choice the surgeon should be guided by the position of the appendix in individual patients, for which reason his clinical examination should be conducted with this aim in view. The recognised incisions are: (1) an oblique muscle-splitting approach, often inexplicably named the “grid-iron” incision, suitable for all uncomplicated cases in which the appendix is in the iliac fossa near McBurney’s point; (2) the right semilunaris incision, usually known as “Battle’s,” suitable for all appendices, especially those in the pelvis and (3) the midline incision, which should be reserved for abscesses definitely palpable above the pubes in the middle line. If these methods are observed, the cæcum need not be freely delivered on to the abdominal wall, and the ileum will be seen but not handled.

It is unfashionable to stress the value of drainage, but it is of the greatest service, when obtained through a properly placed lateral incision, whereas an incorrectly planned or a median incision gives the reverse results. Another advantage of these lateral incisions has been pointed out by Gordon Taylor, that they are rarely the cause of the late peritoneal bands in which the midline incisions so frequently result.

**The Conduct of the Operation.**—If the incision is suitably placed, the appendix or the abscess will be easily found. The abscess is opened by gentle manipulation with the finger or the sucker tube. All free fluid and all the contents of the abscess cavity are aspirated with the sucker, thus avoiding all the trauma caused by mopping. The appendix is mobilised by dividing the vessels in the meso-appendix and its base is then crushed and ligatured. The appendix is removed and its stump cauterised and buried by an invaginating stitch. When it is very swollen and fixed tightly by its mesentery and by inflammatory adhesions, it is often advisable to divide the base first and the meso-

appendix afterwards, for by this means the danger of rupture is avoided.

**Treatment of Abscesses.**—The decision as to the propriety of removing the appendix from an abscess cavity is one which must be settled in each individual patient. With thick walls and an old-standing abscess, removal is often out of the question, but apart from these the surgeon of experience can nearly always remove the appendix, which is the cause of the suppuration. In every case where perforation has occurred, the bed of the appendix must be carefully inspected in case a stercolith may have escaped from its cavity and be lying free in the peritoneum (Fig. 331).

**Drainage.**—In the presence of localised pus or free offensive fluid, the site of the appendix or the parietes may need to be drained, the walls being loosely closed around the drain. The desirability of lateral drainage has already been emphasised. After removal of the appendix from an abscess cavity, there is often some bleeding which must be checked completely before the wound is closed.

**After-care and Progress.**—In general the less interference the better for the patient, but rectal saline is not disturbing and does not prevent the rest and sleep that are the most urgent needs in the early hours of recovery. Small doses of morphia will usually be necessary, but should be discontinued as soon as possible, as this drug causes nausea and vomiting. Intravenous infusions are needed only in the presence of severe toxæmia in peritonitis or for those suffering from persistent vomiting. Tube drainage in the peritoneal cavity should not be left in longer than four days, and two days is the average period during which it carries on useful functions. Patients so treated make quick recoveries, but there are certain post-operative complications which need mention.

**Complications.**—1. *Pneumonia*, massive pulmonary collapse and pulmonary embolism account unhappily for a proportion of deaths following acute appendicitis.

2. *Residual Abscesses.*—Where there is unlocalised peritonitis, and after removal of the appendix, pus may form in more remote parts of the peritoneum, and a residual abscess is thus established. Such collections either disappear spontaneously, burst into the bowel or need drainage by operation. The clinical course and treatment of such conditions have been fully described in Chap. XXVI.

3. *Fæcal Fistula.*—When a suppurative peritonitis or abscess has developed before operation, whether appendicectomy is carried out or not, there may be a profuse stinking discharge from the wound sometimes difficult to distinguish from fæces. Such a discharge presages a fæcal fistula from the cæcum or appendix, which is usually temporary and undergoes spontaneous closure. Rarely they persist and need operative repair after the suppuration has subsided.

4. *Ileus.*—This dread complication has been fully discussed in Chap. XXX in the section on Paralytic Obstruction of the Intestine.

5. *Infective Ileocolic Phlebitis* has already been described.

6. *Spreading Gangrene of the Abdominal-wall.*—This very rare complication is not confined to operations for appendicitis. It is described on p. 177.

## CHRONIC APPENDICITIS

*Clinical Picture.*—Recurring attacks of abdominal pain may be due to subacute appendicitis, in which case each attack is a miniature bout of mild acute appendicitis and differs from the description given above in no essential feature. Similar attacks may be due to recurring appendicular obstruction without inflammation, in which the pains are commonly felt only in the right iliac fossa as sharp pricking pains of short duration. A chronic unending pain of wider distribution is not usually due to this cause and is more commonly a sign of constipation without appendicular obstruction. A valuable feature in the clinical history of true appendicular obstruction is the presence of free intervals with no discomfort, although the underlying constipation is perpetual.

Associated with this type of trouble there may be, in the majority of sufferers, digestive difficulty described as indigestion. It consists of pain immediately after food, usually without vomiting.

*Diagnosis* is not easy, because such affections as old tuberculous glands may give almost identical symptoms. Idiopathic right-sided hydronephrosis is also a pitfall; indeed chronic appendicitis should never be diagnosed until the right kidney has been proved to be normal. Skiagraphic examination of the appendix with an opaque enema and meal is sometimes valuable (Fig. 332).

*Treatment* of chronic appendicitis is the removal of the organ, and if the diagnosis has been correctly made, the result is most gratifying.

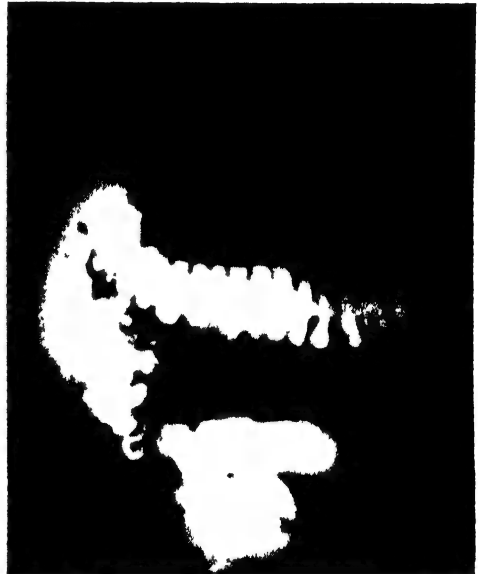


FIG. 332

A barium-meal examination demonstrating a long appendix with several kinks and filling defects suggesting the presence of stercoliths.

## MUCOCELE AND PSEUDOMYXOMA PERITONEI

Mucocele and pseudomyxoma peritonei are rare forms of chronic appendicitis, the pathology of which has been described in the section with which this chapter opened. The clinical history is that of recurring attacks of right-sided abdominal pain, after many of which a progressively increasing abdominal distension makes its appearance. This is due in the case of the mucocele to the presence of a mucus-containing cyst with a thick wall and of great capacity.



The cyst is filled with mucus by the ruptured appendix or appendicular diverticulum, communication with the cæcum having been closed by previous inflammatory attacks affecting the base of the organ. In the case of the pseudomyxoma the mucus escapes into the general peritoneal cavity, where the widespread mucous masses lead to diffuse inflammatory reactions causing chronic peritonitis. In either case removal of the offending appendix cures the disease, and it should be noted that it is possible for even an experienced surgeon to open the abdomen and mistake the appearances for a tuberculous peritonitis or a diffuse colloid carcinomatous involvement of the peritoneum.

#### ACTINOMYCOSIS OF THE APPENDIX

The appendix is affected more commonly by this streptothrix than any other part of the human body, with the exception of the buccal cavity. It seems to be a complication of acute appendicitis and has the characters of the disease as seen elsewhere, a spreading patchily suppurating indurative inflammation that involves every tissue that it meets, respecting none. It spreads into the tissues of the right iliac fossa and leads to multiple intestinal fistulæ.

Clinically it is seen in the form of sinuses appearing or often persisting unexpectedly after appendicectomy. The sinuses lead into an indurated mass affecting all the structures in the vicinity. It is best treated by opening abscesses when they appear, by the exhibition of large doses of potassium iodide (up to clv. gr. daily) and by nursing the patient in the open air by night as well as by day. About 50 per cent. of patients recover.

#### TUMOURS OF THE APPENDIX

The least uncommon tumour of the appendix is the rare "carcinoid" of the mucosa (the argentiform tumour). It is a small yellowish outgrowth from the mucosa and it does not involve the more external coats. It is hardly more than a pathological curiosity.

JULIAN TAYLOR.



## CHAPTER XXXIII

### THE LIVER AND BILIARY SYSTEM

**A***NATOMY.*—The **Liver** occupies the dome of the diaphragm and is to a large extent under cover of the lower ribs and costal cartilages on the right side, the ensiform cartilage, and the 6th, 7th and 8th costal cartilages on the left side. It is reddish brown in colour, firm but friable, and has a smooth surface. It weighs between 40 and 60 oz., and in adults is equal to one-fortieth of the total body weight. It has superior, anterior, right lateral, posterior and inferior surfaces.

The anterior, superior and right lateral surfaces are in contact with the diaphragm. The superior and anterior surfaces are divided into right and left lobes by the falciform ligament.

The inferior surface looks downwards, backwards and to the left, is divided into right, left and quadrate lobes, and has the portal fissure near its junction with the posterior surface. The left lobe lies to the left of the round ligament and is in relation to the stomach; the quadrate lobe lies between the round ligament and the gall-bladder, and is in relation to the first part of the duodenum and pylorus. The gall-bladder occupies the cystic fossa of the right lobe, which is also related to the hepatic flexure of the colon, the right kidney and the second part of the duodenum. The portal fissure contains the hepatic artery, the hepatic ducts, the portal vein, lymphatics and nerves of the hepatic sympathetic plexus.

The posterior surface presents a concavity which lodges the convexity of the 10th and 11th dorsal vertebral bodies. The left lobe shows an impression for the œsophagus, between which and the groove for the inferior vena cava is the Spigelian lobe. This is covered by the peritoneum of the lesser sac, and is in relation to the aorta. To the right of the inferior vena cava is a depression for the right suprarenal capsule.

The hepatic artery is a branch of the celiac axis, and divides into a right and left branch in the portal fissure to supply the two lobes. The portal vein, formed by the union of the splenic and mesenteric veins, also enters the portal fissure and divides into two main branches. The hepatic veins enter the inferior vena cava. The lymphatics enter the glands in the portal fissure, and their radicles run to the glands around the celiac axis artery or to the receptaculum chyli direct.

The right and left hepatic ducts drain the two lobes of the liver, and in the portal fissure unite to form the common hepatic duct. One inch below the liver this joins the cystic duct to form the common bile duct.

**The Gall-bladder** is a pear-shaped sac lying in the cystic fossa on the inferior surface of the liver and projecting slightly beyond the anterior border at the level of the 9th right costal cartilage. It is attached to the liver by loose areolar tissue, the rest of its surface being covered by peritoneum. Its neck narrows to enter the cystic duct, which unites with the hepatic duct to form the common bile duct. At its junction with the duct the neck forms a pouch, named Hartmann's pouch, in which stones may become impacted. The relations of the gall-bladder are: above and in front, the liver; to the left, the pylorus; below, the beginning of the transverse colon and the first

part of the duodenum ; and to the right, the hepatic flexure. It is supplied by the cystic artery which is a branch of the hepatic.

**The Common Bile Duct** is formed by the union of the hepatic and cystic ducts. It runs in the right free margin of the gastrohepatic omentum in front of the foramen of Winslow with the hepatic artery on its left and the portal vein behind and to the left. It passes behind the first part of the duodenum, and then runs along the inner margin of the second part in a groove in the pancreatic lobules, to unite with the pancreatic duct when it forms a small dilatation called the Ampulla of Vater. The ampulla has an opening into the duodenum which is surrounded by the sphincter of Oddi. The length of the component parts of the biliary duct system are as follows : common hepatic, 1 to  $1\frac{1}{4}$  in. ; cystic,  $1\frac{1}{2}$  in. ; common bile duct,  $3\frac{1}{2}$  in.

## THE LIVER

### ANOMALIES OF FORM AND POSITION

The liver may have a tongue-shaped extension of the right lobe downwards towards the right iliac fossa, known as Reidel's lobe. It is occasionally seen in connection with an enlarged gall-bladder containing stones, but it may be present in the absence of any pathological condition.

Displacement of the liver—hepatoptosis—is sometimes seen as a part of generalised visceroptosis.

### INJURIES OF THE LIVER

**Penetrating Wounds** are caused by bullets, shell splinters, bayonets and knives. The immediate danger is hæmorrhage and later infection. If the portal vein is injured, the hæmorrhage is usually fatal. In accordance with the fundamental principles of wound treatment, EVERY wound of this type MUST be enlarged and explored, the tear in the liver sutured and the peritoneal cavity drained.

**Subcutaneous Ruptures** are due to falls from a height, crushing accidents, direct blows and sudden acute flexion of the trunk. A liver which is enlarged from disease will rupture more easily than a normal one. The right lobe is injured six times more often than the left, and the majority of tears are on the superior and anterior surfaces. The dominant feature is intraperitoneal hæmorrhage, and its severity may be grouped thus : (a) severe bleeding without localising signs ; (b) a similar type but with pain, tenderness and rigidity of the right upper quadrant of the abdomen ; (c) mild bleeding without typical evidence of internal hæmorrhage and having localising signs in the upper abdomen followed a few days later by enlargement of the liver and mild jaundice. In the first two groups the patient presents the classical picture of intraperitoneal bleeding (see pp. 143 and 531).

*Treatment* consists in an immediate laparotomy through a midline incision above the umbilicus. The tear in the liver is sutured with mattress sutures of thick catgut introduced on a special liver needle.

Gauze packing may also be needed to secure hæmostasis. In cases of severe bleeding, this may be controlled as soon as the abdomen is opened by compressing the hepatic artery and portal vein between the thumb and a finger in the foramen of Winslow.

### INFECTIONS OF THE LIVER

Infecting organisms may reach the liver by : (1) the portal vein, (2) the hepatic artery, (3) the bile ducts, (4) the lymphatics, and (5) by direct spread. The following conditions are found :—

- |          |   |                                      |
|----------|---|--------------------------------------|
| Acute    | { | (a) Acute suppurative pylephlebitis. |
|          |   | (b) Acute suppurative cholangitis.   |
|          |   | (c) Subacute cholangitis.            |
|          |   | (d) Catarrhal cholangitis.           |
| Specific | { | (a) Gas gangrene.                    |
|          |   | (b) Tubercle.                        |
|          |   | (c) Syphilis.                        |
|          |   | (d) Actinomycosis.                   |
|          |   | (e) Amœbic dysentery.                |

### ACUTE SUPPURATIVE PYLEPHLEBITIS

This condition, known as portal pyæmia, has become very rare. It may occur in infective conditions in any part of the gastro-intestinal tract drained by the radicles of the portal vein. Acute appendicitis in the past accounted for most of the cases, but sepsis in the rectum still leads to an occasional example of portal pyæmia, and rarely a septic umbilical cicatrix in infants is the cause. In systemic pyæmia the infection is carried by the hepatic artery and produces a similar pathological and clinical picture. The liver is enlarged and studded with multiple small abscesses, each arising around the end of a portal capillary. Later adjacent abscesses may fuse to form one of some size. In portal pyæmia the organisms are bacillus coli and streptococci, and in general pyæmia usually staphylococci and streptococci.

*Clinically*, it is noticed that, after an operation for appendicitis, hæmorrhoids or other gastro-intestinal sepsis, the patient's progress is slow and not altogether satisfactory. After a few days the general condition begins to deteriorate and the temperature, which has never settled, begins to rise steadily. There is rapid loss of weight, pain in the liver area and, sooner or later, rigors. The severity and duration of the condition varies greatly. Fulminating cases will be dead in seventy-two hours, while others may linger on for weeks.

*Treatment* is of little or no avail. In streptococcal cases sulpha-pyridine should be given. The real treatment is prophylactic, *i.e.*, earlier diagnosis of the causal acute abdominal condition, and a more wise preparation for and choice of rectal operations. Julian Taylor has recently advised ligature of the superior mesenteric vein, a courageous effort to overcome an otherwise fatal catastrophe.

## ACUTE SUPPURATIVE CHOLANGITIS

This disease is caused by infection reaching the liver, either by the lymphatics from an infected gall-bladder or by the biliary ducts as in an obstruction to the common bile duct by stones. The liver is enlarged and riddled with multiple small abscesses arising round the radicles of the biliary capillaries. The ducts themselves are dilated, thickened and filled with thick purulent bile. The clinical picture consists in high fever, rigors, enlargement of the liver and jaundice, a fatal issue being a matter of days.

*Treatment* is of no avail when the condition is established, but should be prevented by more certain diagnosis and earlier surgical intervention.



FIG. 333

Gas gangrene of the liver.

## SUBACUTE CHOLANGITIS

This is localised to the bile ducts and their capillaries, a less acute variety of the former condition, in that the infection does not spread into the liver and no abscesses are found. Gall-stones may remain in the common bile duct for long periods, giving rise to periodic attacks of colic, and in the intervals causing either no obstruction or only a partial one. The bile is infected, but as long as it can enter the duodenum no signs of infection are evident, although a mild degree of jaundice may persist. When, however, the stone impacts complete biliary obstruction results, with a rapid increase in the virulence of the infection.

Owing to the huge surface area of the biliary ducts, there is a great absorption of toxic bile in a short period. The attack of colic is accompanied, therefore, by an abrupt rise of temperature to 104° or 105° F., and later an increase takes place in the depth of the jaundice. As soon as the stone disimpacts, biliary drainage is re-established and the temperature falls almost as abruptly as it rose. So steep is this rise and fall that the name "steeple chart" is well merited. The stones must be removed and the bile ducts drained, lest an acute suppurative cholangitis supervene.

## CATARRHAL CHOLANGITIS

Catarrhal jaundice is a medical condition which has a surgical interest only in so far as it presents a problem in diagnosis.

## GAS GANGRENE

Gas gangrene of the liver is seen occasionally in cases where this organism causes infections in the portal area. The liver is riddled with small abscesses containing gas, and the condition is rapidly fatal (Fig. 333).

## TUBERCULOSIS

This is rare and may take the form of miliary tubercle, a caseous abscess or perihepatitis in tuberculous peritonitis. The liver may be enlarged and if there is a mass in the portal fissure there will be jaundice. If an exploratory laparotomy is decided upon in an obscure case, it may be possible to evacuate an abscess cavity.

## SYPHILIS

Gummata may be seen in both congenital and acquired syphilis. They are either single or, more commonly, multiple, and attack the rounded superior and anterior surfaces and the region of the portal fissure. They have an area of perihepatitis over them or a zone of fibrosis around them. The liver is slightly enlarged and nodular thickening may be felt. There is pain and the liver is tender; ascites is present only if the portal vein is obstructed, and jaundice is rare.

The *treatment* is that of tertiary syphilis.

Perihepatitis, hepatitis and syphilitic cirrhosis are described in textbooks of medicine.



FIG. 334

Actinomycosis of the liver, showing the typical honeycomb appearance.

## ACTINOMYCOSIS OF THE LIVER

The liver is the fourth commonest organ in the human body to be attacked. The infection reaches it either by venous or more rarely lymphatic spread from the ileocaecal region or occasionally by direct spread from the lungs through the diaphragm. Abscesses may be single or multiple, and present the typical honeycomb appearance (Fig. 334). The disease is described in Chap. IV.

## AMÆBIC ABSCESS

Liver abscess is a well-recognised complication of amœbic dysentery, and is seen in the tropics, the Near East and South-East

Europe. It has been called a "tropical" or a "solitary" abscess, but as it is not confined to the tropics and as it is multiple in 40 per cent. of cases, it seems wise and more pathologically correct to use the term "amœbic abscess."

The infection reaches the liver by the portal vein, the amœbæ migrating from the colon. It is rarely seen in patients in whom the diagnosis of dysentery was made at once and who were efficiently treated with emetine. It may occur in cases of mild diarrhœa in which the diagnosis of dysentery has never been discussed. The liver can be affected at any time after the original bowel infection, and as long an interval as two years may elapse.



FIG. 335

Amœbic abscess of the liver.

The abscess is single in 60 per cent. of cases and usually affects the upper and posterior area of the right lobe. Suppuration is preceded by a gradual inflammatory softening of the liver tissue, and if this is near the surface an area of perihepatitis results. The wall of the abscess is devoid of any fibrosis and consists of disintegrating liver cells with a leucocytic reaction (Fig. 335). The pus is sterile, but amœbæ can be recovered from scrapings of the abscess wall. It increases slowly until a large size is reached and, as this enlargement is usually upwards, the diaphragm is displaced upwards and finally becomes adherent. In this way the abscess may erode the diaphragm, enter the lung and be evacuated via a bronchus. The pus is either pale grey-green from admixture with bile or anchovy sauce colour from the presence of blood.

*Symptoms and Signs.*—The onset is insidious and obscure and a large abscess may develop before it is diagnosed. So successful is medicinal treatment in the early stages that it is imperative that the early clinical picture be clearly understood. There is first a complaint of too easily induced fatigue, loss of weight and slight but persistent aching deep beneath the lower ribs on the right side, usually in the posterior scapular line. This pain may be referred to the right acromioclavicular joint. There is an earthy sallowness of the skin. The subjective signs are few in this stage. A history of exposure to infection and of mild attacks of diarrhœa should always instigate a search for amœbæ or cysts in the fæces. Later the pain becomes more severe from perihepatitis, and the liver is tender on deep palpation or heavy percussion and on lateral flexion and extension of the trunk. The temperature is raised to 100° and 101° F., and night sweats occur. An examination of the blood reveals a polymorphonuclear leucocytosis

with an eosinophilia. There may be a tendency to diarrhoea, and the signs of compression of the base of the right lung with a pleural effusion should lead to a searching examination in every patient who has had, or might conceivably have had, dysentery. X-ray photographs show fixation of the right cupola of the diaphragm at a raised level.

*Treatment.*—The stage before suppuration and the small abscess are cured by emetine, whereas in the large single abscess the mortality is 20 per cent. and in the multiple ones the outlook is hopeless. In every case a full emetine course is given, viz., one grain of emetine sulphate intramuscularly for twelve successive days. In the early stages this kills off the amœbæ and the lesion cicatrises up, or if an operation is needed later, the patient's general condition will have been greatly improved. If operation is decided on, the abscess is approached from the side by resecting part of the 9th rib in the mid-axillary line, and by going across the pleural cavity which will be obliterated by adhesions. The abscess is opened, the pus evacuated and the cavity irrigated with a solution containing ten grains of quinine hydrochloride. The liver is then sutured and a small drain left down to the suture line for two days. The abscess cavity must never be drained unless secondarily infected. In some cases aspiration through a cannula and irrigation with quinine have proved successful. During convalescence, three grains daily of emetine bismuth iodide should be given from the tenth to the twenty-second day in gelatin capsules.



FIG. 336

An adenoma of the liver or "hepatoma."

### GROWTHS OF THE LIVER

These may be classified as follows :—

	<i>Benign.</i>	<i>Malignant.</i>
A. Primary . . . . .	{ Adenoma. Angioma.	{ Carcinoma. Sarcoma.
B. Secondary . . . . .		{ Carcinoma. Sarcoma. Teratoma.

**Primary Growths** are very rare. The adenoma or "hepatoma" (Fig. 336) is an encapsuled tumour growing either from the liver cells or from the bile capillaries. They will rarely be operable, although Grey Turner did a brilliantly successful removal in a boy. Angioma is common and may grow to considerable size, but it gives no

symptoms and requires no treatment. The primary liver cell carcinoma is rare and of several types, one arising in the regeneration nodules seen in cirrhosis. Sarcomata occur as large pinkish white soft tumours. They produce enlargement of the liver and soon prove fatal.



FIG. 337

Secondary carcinoma of the liver.

VI and here only the local clinical features will be dealt with. The liver is more frequently attacked than all other regions taken together, between 57 and 63 per cent. of cysts being hepatic (Fig. 339). There may be one or more cysts which can occupy any part of the liver, the upper part of the right lobe being the commonest site. The clinical picture varies according to their size and the complications to which they may be subject. A single hydatid will grow to large size without symptoms, causing merely dragging pain from its weight or evidence of pressure on surrounding structures. If it becomes infected, the clinical picture is that of a liver abscess, and if it bursts into the peritoneal cavity an acute abdominal disaster, with great pain and vomiting, has occurred. A slight leakage is suggested by attacks of urticaria. It may rupture into a neighbouring coil of intestine and a spontaneous cure result.

**Secondary Growths** are common in every form of malignant neoplasm, particularly carcinoma of the stomach, gall-bladder, intestines, rectum, uterus and breast, and also sarcoma of all types, particularly melanotic. Secondary nodules of carcinoma occurring on the surface are sometimes umbilicated and may be palpable. All give rise to a rapid enlargement of the liver with jaundice and ascites (Figs. 337 and 338).

### CYSTS OF THE LIVER

**Hydatid disease** is discussed in Chap.



FIG. 338

Secondary melanotic sarcoma of the liver.



*Treatment* consists in the removal of the cyst when possible, and failing this it is exposed, the fluid withdrawn and replaced by 1 per cent. formalin. The wound is then carefully packed off and the germinal lining removed whole or piecemeal.

Very rarely a **single cyst** of developmental origin may be found growing from the lower surface near the anterior border. It may be mistaken for a mucocele of the gall-bladder, a hydatid or mesenteric cyst. **Congenital polycystic disease** is occasionally associated with the similar condition in the kidneys.

#### THE SURGICAL TREATMENT OF CIRRHOTIC ASCITES

The Talma-Morrison operation of omentopexy was first successfully performed by Rutherford Morrison in 1895. The obstructed portal circulation can be relieved if an efficient collateral anastomosis is established between the portal and systemic venous circulations. Omentopexy consists in suturing part of the great omentum into the abdominal wall. In successful cases an enormous development of the superficial veins of the trunk occurs and blood is drained into the common femoral and axillary veins. The selection of patients is important; the cirrhosis must be alcoholic and the patient free of cardiac, renal or pulmonary disease. They must become absolute teetotallers afterwards. Under these conditions the operation is safe, but the results are variable.



FIG. 339

Hydatid cyst of the liver.

### THE GALL-BLADDER

*Methods of Investigation—A. Clinical.*—An enlarged gall-bladder produces a pear-shaped swelling, the long axis of which is directed downwards and inwards from the tip of the 9th right costal cartilage towards the umbilicus, or to a point a little below it. It moves with the liver on respiration and may be moved from side to side unless adherent from infection. The swelling is dull on percussion and this note is continuous with the liver dullness above, while there is resonance on either side of it. A diseased gall-bladder, which is not enlarged, will be tender on deep palpation, and this may be made more apparent by Murphy's test. This is done with the patient sitting up, while the examiner's fingers press deeply around the costal margin. Inspiratory movements are suddenly checked as the tender gall-bladder

comes into contact with the fingers, and the patient experiences a sharp jab of pain. This test may also be done in the semi-prone position, when the sign may be elicited in a similar manner.

**B. Radiography.**—X-ray photography reveals but a small proportion of gall-stones (Figs. 340 and 341), and a shadow of the gall-bladder is difficult to obtain. Barium-meal examinations may suggest gall-bladder disease from deformities of the pyloroduodenal shadow.

**C. Cholecystography.**—In 1924 Graham introduced a drug which was excreted only in the bile and was opaque to X-rays. Further researches led to the adoption of sodium tetraiodophenolphthalein, a drug with slight toxicity but with a satisfactory density in radiography. It is given either by the mouth or intravenously. The latter is the more reliable route, but occasionally produces shock, vomiting, shivering and general malaise. The dose is 3 grm. dissolved in 50 c.c. of distilled water, injected slowly over a



FIG. 340

Three gall-stones in the gall-bladder of a boy aged 7 years. They will be seen in a straight line in contact with the shadow of the last rib. Their faint outline is characteristic.

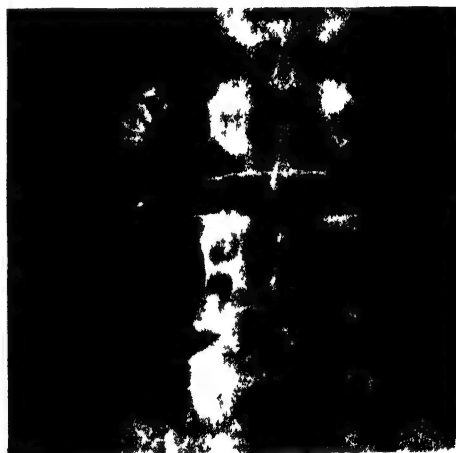


FIG. 341

Multiple gall-stones in an adult. It is rare for the shadows to be so distinct.

period of 15 minutes, and photographs are taken at 2, 4, 8 and 24 hours. The shadow will be at its largest in 4 hours, smaller but denser in 8 hours and no longer visible in 24 hours.

The technique of oral administration varies in different hands. The following has been proved to give satisfactory results. At 7 P.M. the patient is given a meal rich in carbohydrates but lacking in fats, and with it swallows the keratin-coated capsule containing 4 to 5 grm. of the drug. The following morning at 9 A.M. a photograph is taken, after which a meal rich in fats is given and a second photograph taken at 2 P.M. Success depends upon absorption of the drug from the intestinal canal, and this may be hindered either by precipitation of an insoluble compound by the acid of the gastric juice or the too rapid elimination by diarrhoea or vomiting.

The drug, being secreted by the liver, reaches the gall-bladder where the bile is concentrated. The shadow observed, therefore, is of the gall-bladder only, and in health is oval or pyriform in shape and homogeneous in density. If occlusion of the ducts prevents bile entering the gall-bladder, or if this is tightly packed with many calculi, then no shadow will be visible. In gall-bladders with a few stones the shadow presents a mottled appearance,

and lastly tumours or adhesions may cause deformities in the shape of the shadow. If the gall-bladder fills, but fails to empty in normal time, chronic cholecystitis may be inferred. While this method marks a great advance in biliary diagnosis, it must be understood that a negative result, *i.e.*, absence of shadow, does not necessarily prove disease. Positive results are alone reliable (Figs. 342 and 343).

**D. The Lyon Duodenal Catheterisation Test.**—A copious flow of bile can be produced by introducing a 25 per cent. solution of magnesium sulphate into the duodenum through an Einhorn's tube. The bile can then be withdrawn for examination. In health it should be sterile, contain no cholesterin



FIG. 342

The appearance of a normal gall-bladder as seen in a cholecystogram.



FIG. 343

The mottled appearance produced by gall-stones in a cholecystogram.

crystals and a very small amount of mucin. In disease there may be infection with coliform organisms or streptococci, an excess of mucin and cholesterin crystals.

In conclusion, let it be fully understood that by careful clinical observation a correct diagnosis of gall-bladder disease may be made in 65 to 70 per cent. of all cases, and that simple radiography will raise the percentage to 75. It is only in the remaining 25 per cent. that the auxiliary tests should be done. It is not only unnecessary, but quite unjustifiable, to submit every patient with gall-bladder disease to the full routine of cholecystography and duodenal catheterisation.

### ANOMALIES OF THE GALL-BLADDER AND THE CYSTIC DUCT

The gall-bladder itself is rarely the subject of anatomical variation, but the cystic duct and artery provide a number of anomalies which

may be a real danger in operating unless constantly kept in mind. The duct may vary in length and in the level at which it enters the common duct, and may even pass behind it and enter its left side. The cystic artery can arise from the hepatic artery, either of its branches or the gastroduodenal trunk, and it may have almost any relation to the cystic duct and gall-bladder. So many are these variations that no cholecystectomy clamps should be applied until the exact limits of the cystic duct and the common bile duct have been defined and the latter protected.

### INJURIES TO THE BILIARY SYSTEM

These are rare except in company with other intra-abdominal injuries or in penetrating wounds. In such cases the major injuries overshadow those of the bile ducts, but in those rare cases of uncomplicated injury some days or weeks may elapse before a cyst containing bile makes its appearance in the subcostal region. In all cases suture of the tear is required, and if the common bile duct is divided it should be ligated and a cholecystgastrostomy performed.

The common duct is sometimes injured during operations in this region, and either a persistent biliary fistula or jaundice of gradually increasing intensity occurs. If the gall-bladder is present a cholecystgastrostomy is performed, but if it is absent the problem is one of great difficulty. The proximal end is identified and anastomosed to the duodenum, using a flap from the latter and a small rubber tube.

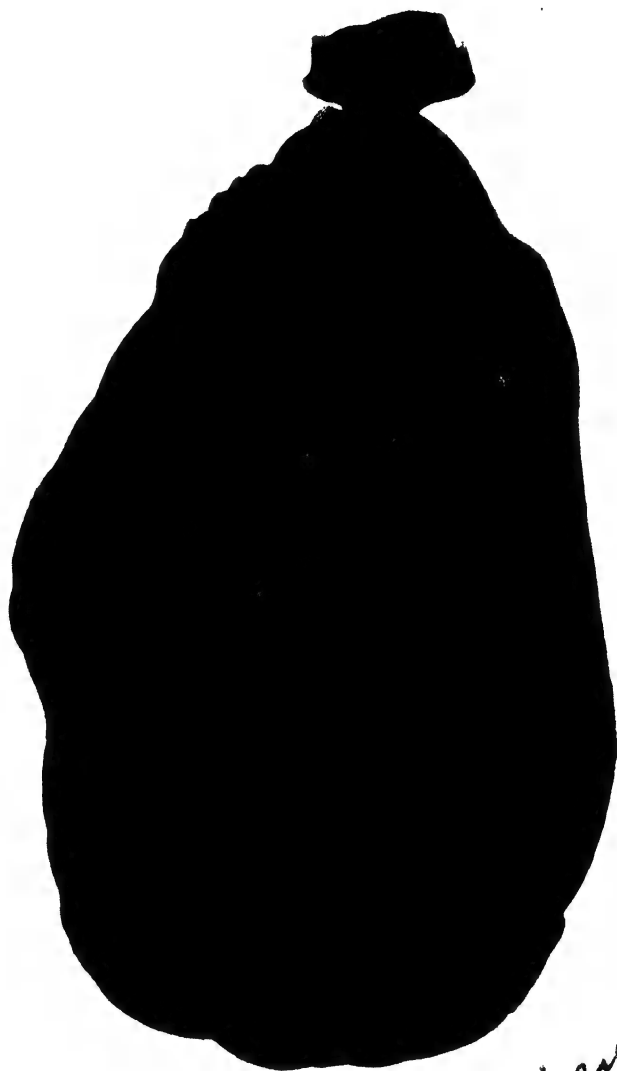
### ACUTE CHOLECYSTITIS

*Etiology.*—The lines of communication along which infection may reach the gall-bladder are the general systemic circulation, the portal circulation, lymph stream and biliary ducts. Rosenow has proved experimentally that streptococci isolated from cases of acute cholecystitis in man produce a similar condition in animals if injected intravenously, and the evidence of many workers upholds the blood-stream theory of infection. In typhoid fever organisms are absorbed by the portal radicle, taken to the liver in large numbers and excreted in the bile, but acute cholecystitis in the acute stage of typhoid is very rare, and the condition cannot be obtained by injecting organisms into the lumen of the gall-bladder. For these reasons it seems unlikely that either the portal circulation or the bile ducts are frequent routes of infection. Lymphatic spread accounts for a certain number of cases.

The causative organisms are usually streptococci, which can be isolated from the wall of the gall-bladder and from the lymphatic glands which drain it. Organisms of the coli group, including the typhoid bacillus, are frequently found in the bile, but are probably a secondary infection and not causative.

Cholecystitis occurs at any age, but is rare in childhood and becomes increasingly more frequent as the years advance. The majority

of cases occur between 40 and 60 years of age, the frequency is greater in women, particularly multiparæ, than men (7 : 1), and the fat, over-



*Anna Linkeisen*

FIG. 344

Acute cholecystitis. An enlarged thick-walled gall-bladder containing mixed stones. The intensely inflamed mucous membrane exhibits many patches of gangrene.

indulgent and lazy are the more prone. The etiological relationship between this condition and gall-stones is fully discussed later (p. 700).

*Pathology.*—Infection starts primarily in the muscle coat and spreads to the mucous membrane. According to the severity and extent of the infection, certain varieties are described, viz., catarrhal, suppurative and gangrenous.

**Acute Catarrhal Cholecystitis** may occur as an independent infection or may accompany catarrhal jaundice. The mucous membrane is red and œdematous, and there is some swelling in the submucosa. It usually subsides without complications, but may lead to some permanent narrowing of the cystic duct, thereby predisposing to chronic cholecystitis.

**Acute Suppurative Cholecystitis** is almost always a complication of gall-stones, the result of a calculus blocking the cystic duct or becoming impacted in Hartmann's pouch. Often the stone is single and large, and the gall-bladder is the seat of chronic cholecystitis. As a result of the obstruction the organisms take on a greatly increased virulence and the gall-bladder becomes acutely inflamed. The whole thickness of its wall is œdematous, red and swollen, and adhesions form around it. Pus collects in the lumen, and unless the stone becomes disimpacted or is removed, the condition will pass into the next stage.

**Acute Gangrenous Cholecystitis** is an advanced stage of the foregoing. The increasing tension within the gall-bladder and the virulence of the infection will lead to thrombosis of the vessels, and gangrene sets in either in patches or of the whole gall-bladder (Fig. 344). The first area to suffer is usually the neck around an impacted stone. Eventually the gall-bladder may rupture and infected bile either bursts through the surrounding adhesions and leads to general peritonitis, or if the adhesions hold firm, an abscess cavity will form locally containing pus, one or more stones and a slough, which represents all that remains of the gall-bladder. In all these severe infections the cystic duct will be obliterated and if the gall-bladder is not removed or destroyed a mucous fistula will result.

**Empyema** of the gall-bladder is a condition in which a stone has become impacted in the cystic duct or Hartmann's pouch, and the virulence of the infection, although too low to produce the gravely acute lesions, is severe enough to cause a purulent inflammation of the mucous membrane. As a result the gall-bladder slowly distends, some omentum becomes adherent and a large tender swelling results.

**Mucocele** results from a similar condition of stone impaction, but in a sterile gall-bladder. As no bile can enter and bile pigments are rapidly absorbed by its wall, which secretes large quantities of mucus, the gall-bladder distends and appears as a large, pale, thin-walled swelling.

*Symptoms and Signs.*—An attack of acute cholecystitis may be the first sign of hepatico-biliary disease, but more commonly there is a history of indigestion typical of chronic cholecystitis. Again, in many patients the condition results from stone impaction, and the story of colic will immediately precede it. The specific symptoms of acute cholecystitis are pain, vomiting, constipation and fever.

1. Pain is the first symptom and is felt in the right upper quadrant of the abdomen. It is persistent and colicky, and grows in intensity until the tension is relieved either by subsidence of the inflammation, rupture of the gall-bladder or operation. The localisation of the pain is at times atypical owing to visceroptosis, when the enlarged and tender gall-bladder may reach the right iliac fossa.

2. Vomiting. Shortly after the onset of the pain there is an initial attack of vomiting and this tends to become more persistent than in most abdominal disorders.

3. Constipation is obstinate and is due to paralysis of the hepatic flexure of the colon, which is in contact with the inflamed gall-bladder. In cases of medium intensity the mistake is frequently made of diagnosing acute intestinal obstruction, with failure to recognise the underlying cholecystitis.

4. Fever will vary in degree according to the virulence of the infection. During the attack in gangrenous cases the temperature may reach  $104^{\circ}$  F., but in empyema it may be only  $101^{\circ}$  F.

On examination there is only one cardinal sign, *i.e.*, local tenderness. Rigidity is not present in mild attacks and does not appear in severe attacks until the peritoneum is involved. A mass may become palpable below the costal margin, consisting either of the gall-bladder itself or of the organ and adherent omentum. The pulse rate is raised and there is a marked leucocytosis. The analogy between acute cholecystitis and acute appendicitis is often very close, the history and the location of the tenderness being the main differences.

*Treatment—A. Expectant.*—A patient suffering from acute cholecystitis is often a poor subject for both anaesthesia and operation. She will be bronchitic and her heart muscle fatty and flabby. In such patients the risk of operation is such that an expectant attitude is adopted and the results are sufficiently encouraging to influence many surgeons to adopt this method as the routine treatment. The patient is confined to bed, kept on a fluid diet and large hot dressings are applied to the abdomen. Morphia in doses of gr.  $\frac{1}{2}$  is given every six hours and a careful watch kept on the pulse and temperature. Appropriate treatment is directed to the relief of pulmonary and cardiac embarrassment.

*B. Operative.*—If the general condition is so good that there are no real contraindications to operation, the method of choice is cholecystectomy. The patient is well rid of an organ, which can never fully recover and which must always remain a source of danger. In the cases of acute gangrenous cholecystitis, which have not been operated on in the early stages, an abscess cavity will form. Its walls are the omentum and neighbouring structures and its contents are one or more gall-stones and a quantity of pus. Little or no trace of the gall-bladder walls may be found. Removal of the stones and drainage will suffice. Cholecystostomy and drainage should never be performed, as the gall-bladder does not recover, and further, if the cystic duct is occluded, a mucous fistula will persist on the abdominal wall.

**CHRONIC CHOLECYSTITIS**

*Pathology.*—Chronic cholecystitis may follow attacks of acute cholecystitis or may be primarily a chronic process. In the milder form the mucous membrane is red, slightly thickened and shaggy, being speckled with little white spots. This has been aptly described as “the strawberry gall-bladder,” the white spots being deposits of cholesterin in the mucous membrane (Fig. 345). Later the walls become thickened and shrunken, the sclerosis affecting all coats. Stones, which are usually present, will be tightly gripped and ulceration may occur around them. Such chronic inflamed gall-bladders may at any time become acutely infected.

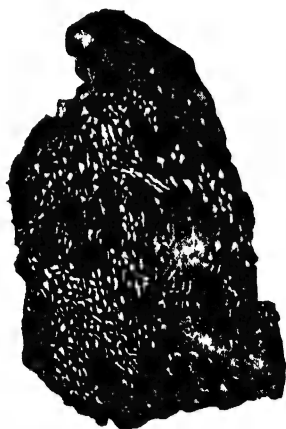


FIG. 345  
A “strawberry  
gall-bladder.”

*Symptoms.*—This condition is described as affecting women of the “fair, fat and forty” type. It is certainly more common in women in the ratio of 7 : 1, and is particularly seen in fat, middle-aged women who have borne children. The syndrome is known as the flatulent

or gall-stone dyspepsia, and the symptoms are due to the chronic cholecystitis rather than to stones. Patients complain of discomfort in the epigastrium and a most distressing flatulence. The discomfort bears no fixed relationship to the taking of food, sometimes occurring immediately after a meal, at other times just before the next. This indefinite time relation is the most important feature in the history, serving to distinguish this condition from peptic ulcer of the stomach or duodenum. The flatulence causes an uneasy feeling of distension, loud and embarrassing internal gurglings and external eructations. Many patients will complain that they cannot eat foods rich in cholesterin, such as eggs, butter and fat. There may be deep tenderness in the right side of the epigastrium.

*Treatment.*—Chronic cholecystitis is the precursor of many serious conditions, amongst them carcinoma of the gall-bladder. For this reason the gall-bladder should always be removed.

**GALL-STONES**

*Etiology.*—The chemical composition of gall-stones varies, being either pure cholesterin, pure bilirubin calcium or most commonly a mixture of the two. They may be single or multiple, the latter sometimes running into many hundreds of small pigment



FIG. 346  
A gall-bladder containing  
multiple faceted stones.



stones. The larger stones, especially in a contracted gall-bladder, bear well-marked facets (Fig. 346).

The factors involved in the production of gall-stones are infection and disorders of metabolism. The *infection* may be in the gall-bladder, the liver, or both. As a result three things may happen: (1) the bile contains a high percentage of proteins and calcium, and numerous centres for simultaneous crystallisation occur; (2) the bile acids are so diminished that cholesterin is precipitated; and (3) fibrosis of the gall-bladder wall leads to an imperfect absorption of cholesterin. *The disorders of metabolism* are chiefly those concerning the fate of cholesterin. Hypercholesterinæmia (*i.e.*, an excess in the blood) may be produced by disease, in response to physiological demand (as in pregnancy), and by the ingestion of too much cholesterin-containing food, *e.g.*, eggs, butter and fats. Such excess in the blood may lead to an excess in the bile, and pure cholesterin stones may be formed. The deposition of pure pigment stones is an example of another type of metabolic disorder.

The exact interrelationship and interaction of these processes is not yet fully appreciated, but it is evident that stagnation of the bile can no longer be considered an etiological factor, nor is the presence of a foreign-body nucleus needed. Infection undoubtedly plays an important rôle, for stones and cholecystitis coexist in over 75 per cent. of cases. The close relationship of pregnancy to gall-stones is due not to stasis caused by the enlarged uterus, but to the frequency of bacillus coli infections during this period and to the hypercholesterinæmia which accompanies it.

Stones may occur at any age; the author has recently removed them from a boy of 9 years and a girl of 8 years, but the commonest period is between 40 and 55 years, and in women of the fat and blonde type who have borne children.

*Clinical Picture.*—The symptoms of gall-stones depend upon their position in the biliary tract and their attempts to migrate. A stone in the gall-bladder will present a completely different picture to one in the common bile duct, and it cannot be too insistently emphasised that no single composite description of the symptoms is possible. In like manner the treatment is different, and for these reasons gall-stones will be described under separate headings, as follows:—

- A. Those in the gall-bladder.
- B. Those attempting to leave the gall-bladder.
- C. Those in the common bile duct (Fig. 347).

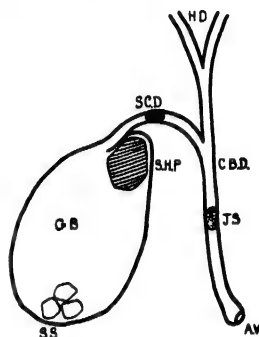


FIG. 347

Diagram showing the various positions of stones in the biliary tract.

G.B., the gall-bladder;  
C.B.D., the common bile duct;  
H.D., hepatic ducts;  
S.S., silent stones;  
S.H.P., stone in Hartmann's pouch;  
S.C.D., stone in cystic duct;  
J.S., stone in the common bile duct or "jaundice stone."  
A.V., ampulla of Vater.

**Stones in the Gall-bladder—Symptoms** — 1. The Silent Stone. Stones may lie quiescent and symptomless in the gall-bladder and be

discovered during operations for other conditions or during a post-mortem examination.

2. The Gall-stone Dyspepsia. In these cases the flatulent dyspepsia is due to chronic cholecystitis, the symptoms of which have been described above (p. 700).

*Treatment.*—If the gall-bladder is only slightly affected by chronic cholecystitis, and if it appears likely that it will resume normal function, then it should be opened at the fundus, the stones removed and drainage established. In all other cases the gall-bladder should be removed. In every case a careful search must be made in the hepatic and common bile ducts to make certain that they contain no stones. Although it is desirable to save the gall-bladder whenever possible, it must be admitted that it is rarely justifiable to leave it.

**Stones Attempting to Leave the Gall-bladder.**—The results may be tabulated thus :—

A. They may enter the cystic duct . . . . .	1. Pass through.	{ Acute cholecystitis. Empyema. Mucocoele.
	2. Impact . . . . .	
	3. Fall back.	
B. They may enter Hartmann's pouch . . . . .	1. Impact . . . . .	{ Acute cholecystitis. Empyema. Mucocoele.
	2. Fall back.	

A small stone may succeed in passing through the cystic duct at the first attempt and will then probably pass down the common bile duct and enter the duodenum. A large stone will block the opening of the cystic duct or more likely become impacted in Hartmann's pouch. Clinically, therefore, there are three types :—

1. The stone which passes through the cystic duct. This may have been preceded by a history of flatulent dyspepsia, and the patient is suddenly seized with violent pain in the right upper quadrant of the abdomen. Often she describes its maximum intensity as extending from the tip of the 9th costal cartilage upwards and inwards to the midline near the xiphisternum, and it is frequently referred through to the back in the region of the inferior angle of the right scapula. The pain is a true colic and usually of the severest intensity, the victim becoming ashen grey, cold, clammy and sweating. As the attack continues the face becomes lined and drawn, and the pulse small, weak and rapid. In the intervals between the more acute spasms, there will be tenderness but no rigidity. So great is the pain, however, that the patient will hardly tolerate examination but will beg for something to relieve the agony. At the beginning of the attack there is vomiting and this may be repeated. Eventually the stone enters the common bile duct (see below).

2. The impacted stone which later falls back free into the gall-bladder. In these cases the picture is indistinguishable from the cystic duct colic, but its termination is brought about by disimpaction of the stone. This usually occurs suddenly, often after an attack of vomiting. A feeling of soreness remains for several days.

3. The permanently impacted stone. The attack of colic, having reached its maximum intensity, decreases in severity and may disappear completely for a few hours, to be followed by a second attack of less severity until the pain finally subsides. The future of the gall-bladder depends upon the presence or absence of infection. If there is no infection, the gall-bladder becomes distended with mucus—mucocoele of the gall-bladder—and a few days later the organ can be felt as a pear-shaped swelling in the abdomen. If the gall-bladder is infected, the picture of acute cholecystitis becomes engrafted on the colic, or, if the virulence of the infection is low, an empyema of the gall-bladder results (Fig. 348).

*Treatment.*—Stones which succeed in passing into the common bile duct will usually enter the duodenum. A small number will be held up in the duct and need appropriate treatment. Every stone which impacts temporarily or permanently requires operative extraction, the gall-bladder being removed.

**Stones in the Common Bile Duct.**—*A.* Those that pass through at the first attempt. These stones cause severe colic. Some patients describe the area of maximum intensity as stretching from the xiphisternum downwards and slightly outwards to a point two-fingers breadth below the transpyloric plane. So severe is the pain that patients have died from the shock and exhaustion it produced. The attack ends abruptly as the stone enters the duodenum. Vomiting may be a prominent feature and afterwards there may be a tinge of jaundice, but it is transient.

*B.* Those that become impacted. These cases are characterised by recurrent attacks of colic at varying intervals, accompanied by high fever at the onset and jaundice afterwards. The temperature, which is due to subacute cholangitis (p. 688), rises abruptly within the first 2 hours of the attack to 104° or 105° F. and falls equally rapidly within 2 days. Jaundice will appear within 24 hours, and its subsequent progress depends upon the frequency of the attacks and the degree of the obstruction. If one stone is present and a long interval occurs between each attack, the jaundice will disappear completely, to recur after each attack. If, however, the intervals are short and several stones are present, causing some permanent partial obstruction, the jaundice becomes deeper after each attack, slowly fading for a time but never entirely disappearing. This variation in the depth of the jaundice is of the greatest diagnostic importance.

An examination will reveal some rigidity during the spasms but none in the intervals, and there will be tenderness over the upper abdomen. In the intervals deep tenderness can be elicited over the



FIG. 348

A gall-bladder opened to show a stone impacted in Hartmann's pouch, with very thickened walls and an intensely inflamed mucous membrane. An example of acute upon chronic cholecystitis.

duct, but not over the gall-bladder, which is impalpable. It is useful to bear in mind Courvoisier's law in these cases, viz., "In jaundice due to stone there is no enlargement of the gall-bladder, but in cases due to carcinoma of the head of the pancreas a large palpable gall-bladder is present" (Fig. 349).

*Treatment.*—Every stone in the common bile duct must be removed at the earliest opportunity to avoid any further damage to the liver. The dilated duct is opened above the duodenum and the stones milked up or down into the opening and removed with forceps or a blunt scoop. A blunt probe is then passed into the duodenum to make sure that no stone has been overlooked. Stones impacted in the ampulla of Vater may be too firmly embedded to be moved. An attempt is made to grasp them with special forceps introduced through the bile-duct incision from above. If this fails the operation known as trans-duodenal choledochotomy is performed, in which the anterior wall of the duodenum is incised opposite the ampulla, this being then dilated from within the duodenum and the stone extracted. The duodenal incision is then sutured. In every case the hepatic and common bile ducts are washed out with saline and a tube stitched into the common bile duct to drain the bile for seven to ten days.

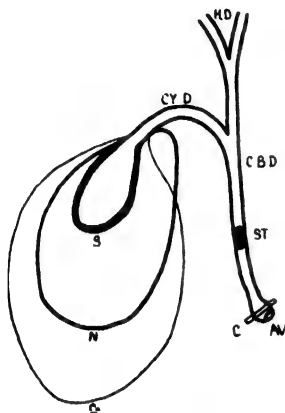


FIG. 349

Diagram illustrating  
Courvoisier's law.

N., normal gall-bladder;  
S., the thickened shrunken  
gall-bladder of chronic  
cholecystitis associated  
with ST., a stone in  
the common bile duct;  
G., a greatly distended gall-  
bladder associated with  
C., a growth in A.V., the  
ampulla of Vater.

*Complications.*—These are acute and chronic cholecystitis, mucocele and empyema of the gall-bladder, local and general peritonitis, carcinoma of the gall-bladder, external and internal biliary fistulæ, acute, subacute and chronic cholangitis, acute and chronic pancreatitis. All these are described under their respective headings, except the fistulæ.

An **External Biliary Fistula** may be either spontaneous or operative. The spontaneous variety is now a surgical curiosity, being due solely to neglect. The inflamed gall-bladder becomes adherent to the anterior abdominal wall at some point between the right costal margin and the umbilicus; the inflammatory process spreads into the abdominal wall and finally bursts through the skin, pus and gall-stones being extruded. No bile will flow, as the cystic duct is certainly occluded. An external fistula will occur after drainage of a gall-bladder whose cystic duct is occluded or obstructed, and a discharge of mucus persists. At times the wound may attempt to heal, and pain due to distension of the gall-bladder occurs until the fistula reopens. The treatment is cholecystectomy, except in the case of old and fragile patients, who may need a small silver drainage tube fitted to prevent the fistula contracting. A fistula-draining bile will result after removal or drainage of the gall-bladder, if an unsuspected obstruction of the common duct is present. Treatment is the relief of the obstruction.

An **Internal Biliary Fistula** is produced by the adhesion of an inflamed gall-bladder to the duodenum or hepatic flexure of the colon. Ulceration of the gall-bladder wall occurs around the stone, the process spreads into the wall of the intestine, and finally a fistula is formed through which the stone passes. In the case of a stone in the small intestine, acute intestinal obstruction may follow.

### GROWTHS OF THE BILIARY SYSTEM

**Carcinoma of the Gall-bladder.**—The great majority (over 80 per cent.) of these growths occur in connection with gall-stones; they are more common in women than men, are seen after the age of 55 years and attack the fundus more frequently than the neck. They are columnar-celled in type. Fortunately it is a rare growth, for its insidious onset usually results in its being inoperable when first seen clinically. Patients in whom there is a long-standing history of chronic cholecystitis become aware of a painless swelling under the right costal margin, or in some people secondary involvement of the liver may provide the first indication. On examination an irregular nodular swelling will be felt in the position of the gall-bladder. Later, pain, jaundice and ascites supervene.

*Treatment* is essentially prophylactic in that every thickened gall-bladder should be removed. The growth will rarely be operable, but if possible the gall-bladder and neighbouring glands should be resected with a wedge of liver.

**Carcinoma of the Common Bile Duct.**—This occurs as a scirrhus ring stricture at any part of the duct, including the ampulla, and rarely in the hepatic ducts. The onset is insidious, jaundice of gradually deepening intensity being the chief symptom. The gall-bladder will be greatly distended and easily palpable. In rare cases it may be possible to resect the stricture, but usually a palliative cholecyst-gastrostomy is all that can be done.

### OPERATIONS ON THE BILIARY SYSTEM

So many different procedures are used in gall-bladder and bile-duct surgery that they are here defined briefly.

*Cholecystostomy* is the opening and drainage of the gall-bladder.

*Cholecystectomy* is the removal of the gall-bladder.

*Cholecystgastrostomy* or enterostomy is the anastomosis of the gall-bladder to the stomach, duodenum or jejunum.

*Choledochotomy* is the opening of the common bile duct for the extraction of stones and for exploration and drainage.

*Transduodenal Choledochotomy* is the approach to the ampulla of Vater by incising the anterior wall of the second part of the duodenum.

*Retroduodenal Choledochotomy* mobilises the duodenum and, turning it forwards and inwards, opens the bile duct from behind.

R. M. HANDFIELD-JONES.

## CHAPTER XXXIV

### THE PANCREAS AND THE SPLEEN

#### THE PANCREAS

**A** *ATOMY*.—The pancreas is a gland of double function (it produces external and internal secretions) lying behind the peritoneum in front of the aorta, vena cava and renal vessels. It consists of a head, a neck, a body and a tail.

The head lies in the concavity of the duodenum. Above it is the pylorus, and behind are the right renal vessels, the inferior vena cava and the common bile duct. In front it is covered by the beginning of the transverse mesocolon.

The neck arises from the anterior surface of the head towards its left edge, and passes upwards and to the left to join the body. Behind it in a groove between it and the head runs the superior mesenteric vein to join the splenic vein, and so form the portal vein.

The body passes across the middle line at the level of the transpyloric plane to reach the tail at the beginning of the lienorenal fold. It is triangular on cross-section, having posterior, anterosuperior and antero-inferior surfaces. The posterior surface has behind it the aorta, left renal vessels, hilum of the left kidney and splenic vein; the anterosuperior is covered with the peritoneum of the lesser sac, and is in relationship with the posterior wall of the stomach; the antero-inferior is covered with the peritoneum of the greater sac and has coils of small intestine in relation to it. Along the anterior border runs the attachment of the transverse mesocolon. The tail lies in the lienorenal fold and reaches the hilum of the spleen.

The pancreatic duct starts in the tail and runs through the whole length of the gland to open into the second part of the duodenum, either separately or having previously joined with the common bile duct just proximal to the ampulla of Vater. One or more accessory ducts may be present opening into the duodenum above the ampulla.

The blood supply is from the superior pancreaticoduodenal branch of the gastroduodenal, the inferior pancreaticoduodenal branch of the superior mesenteric and branches of the splenic artery. The veins join the splenic vein.

*Methods of Examination*.—Clinical examination is difficult owing to the depth of the pancreas within the abdomen, and laboratory tests for detecting its insufficiency are hardly satisfactory. The faeces in pancreatic deficiency are laden with fats and fatty acids (steatorrhœa) and there is an increase in the amount of undigested muscle fibre (azotorrhœa). Diarrhœa is a constant symptom. The best laboratory test is the diastase reaction in the urine, which normally contains 10 to 30 units of diastase and in disease may contain 200 units.

**INJURIES** are very rare, occurring in conjunction with other severe abdominal accidents, and are usually fatal. Mild injuries may be the cause of pseudopancreatic cysts.

### ACUTE PANCREATITIS

The old classification into hæmorrhagic, gangrenous and suppurative is unsound as all three processes are present in severe cases. The clinical picture and the pathological findings depend on the virulence of the process, and the following classification is more satisfactory :—

(1) Fulminating, (2) acute, (3) subacute, and (4) catarrhal.

*Etiology.*—This is one of the most serious of all the abdominal emergencies, and is not common. The author, although having the unique experience of operating on two cases in one evening at St Mary's Hospital, has had only eighteen examples in the past thirteen years ; of these, ten were men and eight women, and in all of them there were present gall-stones of the small multiple variety. Gall-bladder disease with or without stones is present in all cases.

*Pathology.*—Pancreatitis can be produced experimentally by injecting bile into the pancreatic duct. Normal bile produces chronic pancreatitis, while infected bile causes the typical lesions of acute inflammation. It is suggested that, if a small stone is impacted in the sphincter of Oddi, bile from the ampulla is forced back along the pancreatic ducts ; on the other hand, some observers deny that this is possible. At operation, although hundreds of small stones may be present, it is very rare to find one actually blocking the outlet of the ampulla. The question is therefore one of considerable difficulty. Probably some cases are due to a reflux of bile along the pancreatic ducts, but in others the infection is carried from the primary focus in the gall-bladder to the pancreas by lymphatics. The infecting organism is usually a streptococcus.

The naked-eye changes are produced by the conversion of trypsinogen into trypsin, which results in self-digestion of the pancreas and erosion of vessels. At first the pancreas becomes swollen, red or purple and infiltrated with small hæmorrhages. Owing to the escape of ferments, fat is converted into salts of the fatty acids and areas of "fat necrosis" occur in the omentum, mesentery and peritoneum generally. These are small, firm, white areas in the fat beneath the peritoneum which cannot be mistaken for anything else. Later, hæmorrhage becomes more widespread in the gland and infection and auto-digestion cause sloughing of the gland substance. There may be free blood in the greater sac and there will certainly be a blood-stained exudate in the lesser sac of the peritoneum.

In the less acute or subacute varieties the hæmorrhage and the sloughing are less marked and a slower suppurative process is seen. As a result a large mass may be formed of the infected pancreas, gall-bladder, omentum and surrounding structures with an abscess cavity in the centre.

*Symptoms and Signs.*—In the **fulminating** variety the pain is so severe, the collapse so marked and the patient's distress of mind so great that it is probably the most terrible of all abdominal emergencies. The onset is sudden, agonising pain being felt in the epigastrium, going through to the costolumbar angle on both sides. There is an initial attack of vomiting, which may later become a prominent feature



in some patients, and collapse is profound. There will be absolute constipation. The pulse is thin, running and rapid and the temperature subnormal. Cyanosis is often well marked, due to rigidity of the diaphragm. On examination the upper abdomen is very tender and completely rigid, and there is usually tenderness in the right costolumbar angle.

The **acute** variety presents a very similar picture except that the pain and collapse are not so severe and the prognosis not so grave. After subsidence of the acute onset pain and tenderness persist, but rigidity becomes less marked and abdominal distension takes its place, while an indefinite tender swelling becomes palpable deep in the abdomen.

The **subacute** variety is still less severe. The onset is gradual and collapse is not present. The picture resembles an acute cholecystitis rather than an abrupt abdominal disaster. There is a gradual onset of epigastric pain, becoming severe in a few hours, an initial attack of vomiting, no cyanosis, and the temperature is raised to 102° or 103° F. In the early stages there is tenderness and rigidity in the right upper quadrant of the abdomen and later a large ill-defined swelling appears. In some cases this embodies the whole pancreas and can be recognised as such by its shape and position. In others the mass is due to adhesions with all the adjacent structures. In untreated cases operation reveals an abscess with a central slough of the head of the pancreas.

**Catarrhal Pancreatitis** is seen as a complication of some of the infectious fevers, *e.g.*, mumps.

*Diagnosis.*—After forty-eight hours a discoloration is said to appear in the loin on one or both sides below the last rib, and Loewe's adrenalin conjunctival test is positive in acute pancreatitis. Such tests are of academic interest only and if relied upon can lead only to loss of valuable time. In the grave varieties, the condition is usually mistaken for a perforated peptic ulcer, and in the less grave for acute cholecystitis, which may be coexistent. In neither case does the error matter as the patient's condition will obviously demand a laparotomy, and the fat necrosis immediately establishes the diagnosis. Nevertheless there are features in the disease which should lead to more frequent correct diagnosis, and its possibility should always be borne in mind in grave upper abdominal emergencies. In the past conflicting reports cast doubt upon the reliability of the diastase test in the urine. Recent work has shown that the diastase index is always high provided the urine is tested during the acute phase of an attack. It falls to normal very quickly.

*Treatment* consists in adequate drainage of the pancreas and of the common bile duct. The pancreas is fully exposed, the peritoneum over it incised and tubes placed down to it. Care must be taken to drain the body as well as the head and several tubes may be needed. The common bile duct is opened above the duodenum (any contained stones being removed), flushed out with saline and a tube stitched in. If the patient's condition permits, the gall-bladder is opened and stones removed, but this is not essential in the saving of life. The



gall-bladder should be preserved lest a chronic pancreatitis follow recovery and a cholecystgastrostomy be needed.

The results of this grave condition should be improved. The important factors are adequate drainage of the pancreas over a long period (in the author's successful cases the tubes remained in between twenty-eight and thirty-seven days), and drainage of the common bile duct. Of the author's eighteen cases, ten are alive and well. One fulminating case lived for five weeks and died of broncho-pneumonia with resolution of the pancreatic condition as shown at post-mortem, and one died twenty-five days after operation because the left end of the body was inadequately drained, the remainder of the pancreas having cleared up. Recently it has been claimed that, if a correct diagnosis can confidently be made, better results follow expectant treatment. In view of the acknowledged difficulty of diagnosis these claims are not easy to establish.

### CHRONIC PANCREATITIS

This is a medical disease which is sometimes brought to the surgeon for treatment. Chronic infection leads to fibrosis which may be either interlobular, when the gland is shrunken and hard but the islands of Langerhan's escape, or interacinar, as seen in diabetes mellitus and hæmochromatosis. The primary focus is in the gall-bladder or duodenum, and infection is carried to the pancreas by lymphatics. The clinical history is vague and unreliable but the symptoms of the causative factor will be present with steatorrhœa, azotorrhœa and wasting. Jaundice will occur if fibrosis of the head of the pancreas affects the common bile duct or ampulla.

The *treatment* consists in relief of the cause combined with a cholecystgastrostomy.

### PANCREATIC CYSTS

The pathology of pancreatic cysts is still imperfectly understood. They may be classified as follows :—

- |                |   |                                    |
|----------------|---|------------------------------------|
|                |   | (a) Retention cysts.               |
|                |   | (b) Cystadenoma.                   |
| 1. True cysts  |   | (c) Congenital polycystic disease. |
|                |   | (d) Hydatid cysts.                 |
|                |   | (e) Blood and lymph cysts.         |
| 2. Pseudocysts | . | (a) Traumatic.                     |
|                | . | (b) Inflammatory                   |
|                | . | (c) Neoplastic.                    |

**True Cysts** are rare. Retention cysts occur as the result of chronic pancreatitis or of any condition causing blockage of the main ducts. Cystadenomata are exceedingly rare and may be multilocular, or contain intracystic papillomata. Congenital polycystic disease will sometimes be seen in the pancreas as well as in the kidneys.

**Pseudocysts** are usually collections of fluid in the lesser sac of the peritoneum. They are due to injury whereby the peritoneum covering the upper surface of the pancreas is torn, and blood and

pancreatic fluid enter the lesser sac. A mild peritonitis seals off the foramen of Winslow and a closed sac results. The injury is of mild severity, no grave internal trauma results, and within forty-eight hours the patient has recovered. After a few days or weeks a swelling can be palpated in the epigastrium.

The true cysts are lined with epithelium and the pseudocysts with endothelium. They both contain an alkaline, brownish and turbid fluid, with albumen and usually pancreatic ferments, but without bile or urea.

*Symptoms.*—The patient is usually a man past 40 years of age and there may be a history of injury or pancreatic disease. The cyst may grow either into the pancreas and seriously disorganise it, or away from it and press on adjacent structures. The clinical picture therefore varies considerably. There may be slight epigastric pain, trivial indigestion, a little flatulence and constipation. In other cases with pancreatic deficiency there may be rapid wasting, diarrhoea and a sallow complexion. If the bile duct is pressed on, jaundice will develop. All cases present a cystic swelling which is more commonly on the left than the right, though it may be centrally situated. It comes to the surface either (1) between the stomach and the liver; (2) between the stomach and transverse colon; (3) below the transverse mesocolon amongst the coils of small intestine; (4) between the layers of the mesocolon pushing the transverse colon forward; or (5) by passing outwards into the loin. Diagnosis is never easy as they are rare swellings, and many other retroperitoneal cysts occur, but it should never be mistaken for a hydronephrosis, which can be recognised at once by a pyelogram.

*Treatment* presents many difficulties. Every case should be operated on, but the ideal procedure of excision or enucleation is not always practicable. Failing this, the cyst must be drained either by tube, by gauze packing, or by marsupialising it to the abdominal wall. Some surgeons prefer to explore the cyst from the front and obtain drainage through the loin. These cases tend to develop a persistent sinus, which may become a distressing affliction owing to digestion of the wound by pancreatic ferments.

### PANCREATIC CALCULI

These are very rare, are composed of calcium carbonate and magnesium phosphate, and are formed in the pancreatic ducts if these are partially obstructed. They are found very seldom in life, and then only as a surprise in an X-ray film. Their symptoms are varied and not pathognomonic. Occasionally after a severe attack of colic they may be recovered from the stools.

### GROWTHS OF THE PANCREAS

Adenoma, cystadenoma and sarcoma are described, but carcinoma is the only one of importance. Both primary and secondary forms occur, but the primary carcinoma of the head of the pancreas is not so common as would appear, because some of the cases so described are in reality either growths of the lower part of the common bile

duct or chronic pancreatitis. It is a spheroidal-celled carcinoma simplex and is densely scirrhus in type. It gives no symptoms until it presses on or involves the common bile duct. It occurs more often in men than women, usually after the age of 50 years. Jaundice is frequently the first symptom and, having appeared, becomes persistent, varying only by a steady deepening in intensity. According to Courvoisier's law the gall-bladder becomes palpable and distended and there is usually little difficulty in differentiating these cases from those in which a stone is impacted in the common duct. Later, pressure on the portal vein causes ascites and on the inferior vena cava swelling of the legs. The prognosis is hopeless and all that can be done is a palliative cholecystgastrostomy to relieve the jaundice. This is always justified, as it prevents the intolerable itching from which many of these patients suffer.

## THE SPLEEN

*Anatomy.*—The spleen lies in the left hypochondrium beneath the 9th, 10th and 11th ribs. It is surrounded by peritoneum which is reflected from its surface at two places, one fold attaching it to the stomach, the gastro-splenic omentum, and another passing to the left kidney, the lieno-renal ligament. The outer surface is in contact with the diaphragm, while the visceral surface is related to the stomach, the left kidney, the splenic flexure of the colon and the tip of the pancreas. The splenic artery is a branch of the celiac axis; the splenic vein joins the mesenteric veins to form the portal vein; and the lymphatics enter the glands in the hilum, their efferents going to the glands around the celiac axis artery. The anterior border is sharp and contains one or more definite notches, which are preserved in most types of splenic enlargement.

*Function.*—The spleen is a member of the hæmopoietic system. It is the factory of white blood cells, the destroyer of worn-out blood cells of all types and of all foreign constituents in the blood. Its presence is not essential to life, the other members of the system being capable of taking on its functions. After splenectomy there is a slight temporary secondary anæmia, the yellow bone marrow is largely replaced by red marrow, and there is some hyperplasia of the lymphatic tissue in the body.

## ANOMALIES

**Accessory Spleens** are sometimes seen in the gastrosplenic or great omentum in the form of small spherical bodies of splenic tissue. Occasionally the spleen itself is replaced by a bunch of small spleens.

**Movable Spleen** is a condition found occasionally in thin parous women in whom the pedicle is greatly lengthened and the spleen able to move freely within the peritoneal cavity. It may reach to the right iliac fossa and has been found in the sac of an inguinal hernia. If it occurs apart from generalised visceroptosis, it is due either to injury or to some splenic disease. It may be symptomless or a vague dragging pain may be felt. A tumour easily recognised as the spleen is felt within the peritoneum. Many cases can be made comfortable with an abdominal belt, but failing this the spleen should be removed. No useful purpose is served by attempting to stitch it up (splenopexy).

**Torsion** may occur in a movable spleen by axial rotation, so that its vessels become thrombosed. The symptoms are comparable to those of a twisted ovarian cyst, chiefly pain and vomiting. The spleen should be removed.

**Injuries of the Spleen.**—The symptoms of rupture of the spleen are those of intraperitoneal bleeding and are described in Chap. XXVI, p. 532.

### INFECTION, CYSTS AND GROWTHS

**Abscess** of the spleen may be metastatic, infarctive or traumatic. Metastatic includes those occurring in the infective fevers, pyæmia

and septicæmia; infarctive in the breaking-down of infarcts in infective endocarditis; and traumatic when a hæmatoma in the spleen becomes infected. Lastly, abscess may result from the suppuration of a hydatid cyst. It is a rare condition and always a very serious complication of the coexisting disease. It causes a painful and tender enlargement of the spleen with high fever and rigors. The abscess may burst into the peritoneal cavity, any adjacent hollow viscus, the pleural cavity, or through the abdominal wall on to the surface.

The ideal *treatment* is splenectomy, but owing to dense adhesions drainage is usually the only safe procedure.

**Tuberculosis** of the spleen causes a hard and painful enlargement and is rare apart from the miliary form. Syphilitic gumma is also rare.

**Cysts** are usually hydatid in origin, but rare examples are on record of blood, serous and lymphatic cysts.

**Primary Growths** are rare, only angioma and sarcoma occurring, the latter forming a hard nodular and rapidly growing tumour. **Secondary deposits** are also rare, except in connection with malignant lymphomata (Fig. 350).

### THE SURGICAL SPLENOMEGALIES

1. **Splenic Anæmia or Banti's Disease.**—In this disease the spleen is the primary seat of the disease, and only in the later stages is cirrhosis of the liver added to the picture. There is an introductory

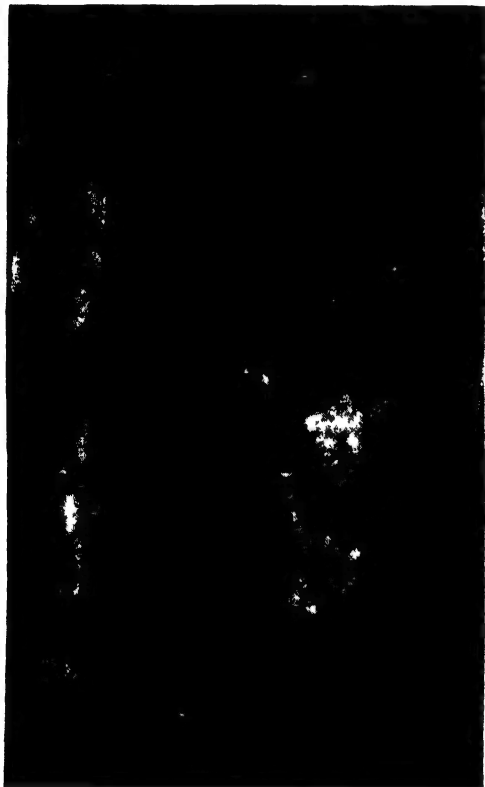


FIG. 350

A spleen showing multiple deposits from a case of malignant lymphoma.

stage of insidious onset, characterised by a mild anæmia, lassitude and possibly a hæmatemesis; then follows the splenomegaly, and later a third stage of hepatic cirrhosis, to which only can the term "Banti's disease" be properly applied. The spleen is hard and enlarged to or beyond the umbilicus. It gives rise to dull, dragging pain due to its weight. The anæmia is of the secondary type with a low colour index and a leucopænia. It may be many years before the liver is affected. Hæmatemesis may be severe in some cases or absent in others. Splenectomy results in cure if performed before hepatic involvement occurs, and even after this some improvement may be expected.

**2. Acholuric Jaundice.**—This disease may be either familial or acquired, and is characterised by a moderate enlargement of the spleen, recurrent attacks of mild jaundice and a mild secondary anæmia. There is no bile in the urine, the stools are normal, and the jaundice is more a dull grey earthy discoloration than the yellow colour of obstructive lesions. In the majority of cases there is an increased fragility of the red blood cells in hypotonic saline. During attacks there is usually mild fever and an increase of the anæmia. Splenectomy cures the anæmia and jaundice but the fragility of the red cells remains. The prognosis is not so good in the acquired type.

**3. Purpura Hæmorrhagica**, especially if an essential thrombocytopenia is present, may be accompanied by a moderate degree of splenic enlargement and a very low platelet count in the blood. Splenectomy has become the established method of treatment for the severe cases, and in spite of the danger of hæmorrhage it is a justifiable risk, as the prognosis without operation is hopeless.

**4. Gaucher's Splenomegaly** is marked by an enormous enlargement of the spleen in childhood. It may be familial and in some respects resembles splenic anæmia, but the anæmia is slight and hæmatemesis does not occur, though there may be bleeding from the nose and gums. Operation is indicated only for distress and discomfort due to the size and weight of the spleen.

**5. Malarial enlargement** of the spleen is a danger to the patient in two ways. First, owing to its friability it is easily ruptured from mild trauma (in the East a recognised method of removing one's enemy is to strike him in the left side, and a special flat, wooden weapon exists for this purpose), and secondly, some parasitologists believe that it is the spleen which harbours the parasite. For these reasons a splenectomy should be performed.

**6. The Spleen** is enlarged in myeloid and lymphatic leukæmia, polycythæmia and lymphadenoma (Fig. 351). In none of these should splenectomy be considered.



FIG. 351

A typical "hard-bake" spleen in lymphadenoma.

## CHAPTER XXXV

### THE KIDNEY AND URETER

**S**URGICAL *Anatomy of the Kidneys.*—The kidneys, two in number, lie behind the peritoneum on either side of the vertebral column. Each measures 4 in. in length,  $2\frac{1}{2}$  in. in breadth and  $1\frac{1}{4}$  in. in thickness. Their position is variable as they move with respiration, but they extend from the level of the upper border of the last dorsal vertebra to the middle of the body of the third lumbar vertebra, the left being slightly higher than the right. Their weight is about  $5\frac{1}{2}$  oz. Each presents an anterior and a posterior border and an upper and lower pole. The surfaces are smooth and glistening, being covered with the renal capsule. The outer border is convex and the inner concave, receiving as it does the renal vessels and the renal pelvis. The upper pole is rather larger and more rounded than the lower. In life the organ is movable and tensile and shows none of the visceral markings described by anatomists.

**Relations.**—(1) *Anteriorly*, the right kidney is in contact with the right suprarenal gland, liver, duodenum, hepatic flexure of the colon and peritoneum, which separates it from the small intestine. The left kidney is in contact with the left suprarenal gland, spleen, stomach, pancreas, splenic flexure of the colon and peritoneum. (2) *Posteriorly*, the relations are similar on the two sides. The inner half rests on the crus of the diaphragm and the psoas magnus muscle, and the outer half on the 12th rib, diaphragm and the quadratus lumborum muscle. The last dorsal nerve with the ilio-inguinal and ilio-hypogastric branches of the 1st lumbar nerve pass posterior to the kidney. The pleura lies behind the kidneys, the diaphragm intervening. (3) *Superiorly*, the suprarenal gland caps each kidney. (4) *Externally*, the kidneys lie against the anterior layer of the transversalis aponeurosis, and at its upper margin the right touches the liver and the left reaches the spleen. (5) *Internally*, the hilum is a longitudinal fissure with thick, rounded lips leading to the renal sinus. It transmits the branches of the renal vein, renal artery and the pelvis in that order from before backwards.

**Perirenal Capsules.**—The kidneys are embedded in a cushion of tough fat which is continuous with the subperitoneal fat. The whole is surrounded by a dense fascia—the fascia of Zuckerkanhl. This, arising from the transversalis aponeurosis in the loin, sends a strong anterior layer in front of the kidney which is continuous with the fascia over the opposite kidney. Its posterior layer passes behind the kidney and is attached to the vertebral bodies. Above it forms a separate compartment for the suprarenal gland; below, the two layers do not fuse with each other.

**The Renal Pelvis** is the funnel-shaped expansion of the ureter, and in the renal sinus it divides into two main sections, from which the calyces spring.

**The Renal Artery** divides into two branches passing one in front and the other behind the pelvis. In this way the kidney is supplied by an anterior and posterior group of arteries, which remain independent. As a result a line of demarcation exists between the two sets of vessels, the so-called “bloodless line of Hyrtl,” parallel to and just behind the outer

border. An additional renal artery is present in about 20 per cent. of people. The lymphatic drainage is into the glands lying in front of and between the aorta and the vena cava at the level of the renal arteries.

**Internal Structure.**—The cut surface shows two layers—cortex and medulla. The medulla surrounds the renal sinus and forms the pyramids. The apex of each of these ends by coalescing with several of its neighbours to form a papilla, which projects into a minor calyx. The number of these varies from six to fourteen. The cortex is thinner and of a different pattern and covers the medulla, sending columns between the pyramids. Several minor calyces unite to form a short, broad major calyx, of which there are usually two, or rarely three. These unite to form the renal pelvis.

**Surgical Anatomy of the Ureter.**—The ureter passes downwards and slightly inwards on the posterior abdominal wall behind the peritoneum, to which it is so closely attached that if the latter is dissected up the ureter will remain adherent to it. It lies on the psoas muscle, is crossed anteriorly by the spermatic or ovarian vessels, while the genito-crural nerve passes behind it. It enters the pelvis by crossing obliquely over the bifurcation of the common iliac artery, the right ureter being here crossed by the root of the mesentery and the left by the mesosigmoid. In the pelvis it passes round the outer wall beneath the peritoneum, crossing the obturator vessels and nerve and the obliterated hypogastric artery. In the male it is crossed by the vas deferens, and in the female by the base of the broad ligament and the uterine artery. It passes over the lateral fornix of the vagina in close relation to the cervix uteri. It enters the bladder by traversing the wall obliquely. The ureter has three narrowings in its lumen, viz., at the ureteropelvic junction, at the pelvic brim and at its entrance into the bladder. It measures 12 in. in length, its intramural vesical part being  $\frac{3}{4}$  in. long and the two orifices between  $\frac{3}{4}$  and 1 in. apart.

**Examination of the Urinary Tract.**—Modern methods have transformed the diagnosis of urinary diseases to an exact science; the following sets out the technique of a complete urological investigation.

1. **Inspection.**—The stigmata of failing renal function are easily recognisable. The tongue is dry, red and glazed, and later brown and furred; the face has the earthy colour of toxæmia rather than the pallor of anæmia; the skin is dry and coarse; the hair is dry, lustreless and fragile; the pulse is rapid, full and bounding and increased in tension. Patients complain of thirst, headaches, loss of appetite and nausea. Locally only very large tumours are apparent in the loin.

2. **Palpation.**—A normal kidney cannot be felt in normal people, unless they are very thin, but if it is enlarged or unduly mobile it is easily examined except in very fat subjects. The patient lies on the back, with the thighs flexed, and the surgeon sits by the affected side (for purposes of description, the right side). His left hand is placed behind the patient with the little finger along the iliac crest and the index finger along the lower border of the last rib, the finger-tips resting against the outer edge of the erector spinæ muscle. The right hand is placed on the front of the abdomen along the costal margin with the finger-tips reaching the edge of the rectus abdominis muscle. The kidney can thus be examined bimanually. The characteristics of a renal swelling are:

- (a) It moves on respiration.
- (b) It can be felt bimanually.
- (c) It comes from under the costal arch, fills out the loin and spreads downward to the iliac fossa of the same side and never diagonally toward the umbilicus.



- (d) It can be reduced beneath the costal arch and if not very large slips from between the hands in a characteristic way.
- (e) Renal "ballottement" can be obtained by pressing the tumour backwards with the anterior hand.
- (f) It may or it may not have a vertical band of resonance across it, but it never has a resonant area outside it.

Renal swellings have to be distinguished from :

- A. On the right side : An enlarged gall-bladder, a Reidel's lobe of the liver, other liver swellings, a mass in the colon, duodenum, pylorus, pancreas or suprarenal gland.
- B. On the left side : An enlarged spleen, a mass in the left lobe of the liver, stomach, colon, pancreas or suprarenal gland.

**3. Renal Pain.**—In kidney disease there are four types of pain : (a) *Local renal pain* is situated deep in the loin, is somewhat diffuse, and is described by patients as in the back, in the abdomen or deep in the side. The centre of the area is the costo-lumbar angle behind (*i.e.*, where the last rib meets the erector spinæ) and the tip of the 9th costal cartilage in front. (b) *Referred pain* is felt over the whole or part of the area supplied by the last dorsal nerve and the ilio-inguinal and ilio-hypogastric branches of the 1st lumbar nerve ; that is, down the inguinal canal to the scrotum or labium majus, over Poupart's ligament to Scarpa's triangle and over the iliac crest to the upper part of the buttock. It is never referred upwards to the shoulder or scapula. (c) *Renal colic* is an agonising pain with periodic exacerbations felt in the kidney area and along the line of the ureter. (d) *Pain in the opposite kidney* is not reflex pain but is produced by tension within the kidney due to its unyielding capsule. When the functional activity of one kidney is impaired, additional strain falls upon the other, which has to increase its normal work. In time, in young people, it will hypertrophy to twice its normal size, but until then an intense hyperæmia is produced, which will lead to increased renal tension. These four types of pain in renal disease will be referred to repeatedly later, but will not again be described in full.

*Examination of the Urine.*—The urine is examined for changes in its specific gravity, in its twenty-four hour quantity, and for any abnormal constituents. In surgical practice the urine will be tested in ALL cases, not only in those in which the disease is urinary in origin. In women only a catheter specimen provides true evidence.

1. **Polyuria**, when continuous, is seen in many conditions in which there is a gradual destruction of renal tissue, and in these, while the actual quantity is increased, the constituents are diminished. In nervous polyuria no change in renal function is found.

2. **Oliguria** is a diminution, and **Anuria** a total cessation of the secretion of urine. Thompson Walker classifies anuria as follows : (a) hysterical anuria ; (b) anuria due to changes in blood pressure ; (c) reflex anuria ; (d) infective anuria, the infection being either blood borne or ascending from the bladder ; (e) tension anuria due either to obstruction or to its too-sudden relief.

3. **Albuminuria.**—Albumen alone is rarely a sign of surgical renal disease, but it may have a marked influence on surgical conditions. If found in a patient before operation, a general overhaul is necessary to ascertain its cause. This may lead to the abandonment of a non-urgent operation or to the adoption of special technique in an emergency.

4. **Glycosuria.**—Sugar is present in the urine in certain surgical conditions *e.g.*, carbuncles, diabetic gangrene, certain intracranial lesions, etc. It will



demand consideration in the planning of surgical treatment, including a decision as to the use of insulin.

5. **Hæmaturia** is a common symptom and one of the utmost importance. No time must be lost in tracing it to its source. If bleeding is occurring at the time of examination the cystoscope will show definitely the origin of the hæmorrhage; otherwise reliance must be placed in pyelograms. The causes of hæmaturia will be discussed under the various diseases, but one condition is most conveniently dealt with here.

*Essential renal hæmaturia* is the name given to a condition in which symptomless bleeding can be traced to one kidney, but on exploration the kidney appears normal and microscopy reveals only a slight glomerular nephritis or an angiomatous condition of a papilla. In persistent cases a nephrotomy will probably be required and this is sometimes sufficient to cure the bleeding, but in other cases a nephrectomy will have to be considered.

In *hæmoglobinuria* the urine appears to contain blood, but on examination hæmoglobin will be found without any blood cells.

6. **Pyuria** is evidence of infection and demands a thorough urinary investigation.

7. **Bacilluria**.—Large numbers of bacteria may be passed in the urine without any sign of infection and without any other abnormal constituent. In over 80 per cent. of cases the *B. coli* is the organism present. Treatment is directed toward the cause, and to the clearing of the urine lest an inflammatory process be set up.

8. **Pneumaturia** or gas in the urine is due either to an infection of the urinary tract with gas-forming organisms or to a vesico-intestinal fistula. Diagnosis will be established by cystoscopy and radiography of the colon, which latter will show the presence of diverticulitis or growth.

*Cystoscopy and Ureteric Catheterisation*.—Examination of the bladder by cystoscopy is the most important method of investigation, but it is to be employed by the expert only. Briefly, a cystoscope is a metal tube carrying a light at its end and a series of prisms and lenses which allow direct vision of the bladder; by its use pathological lesions of the bladder may be seen and catheters passed up the ureters, from each of which the urine can thus be collected separately.

*X-ray Photography*.—In good negatives the outline of the kidney is plainly visible and its size, shape and position accurately determined. The presence of stones and foreign bodies is made clear. The renal pelvis and its calycal systems can be visualised in two ways. **Excretion Urography** consists in the intravenous injection of uroselectan, which is secreted in such concentration by the kidneys that it is well seen in X-ray films. Exposures are taken 3, 7, 15 and 30 minutes after injection, and the first appearance of the shadow, its intensity and any deformity in its outline can be recognised. This method has now been brought to a high degree of efficiency. **Retrograde Pyelography** consists in the injection through a ureteric catheter of a 25 per cent. solution of sodium iodide up one ureter. The pelvis is distended by this means and a denser shadow obtained, but its use is now restricted to those cases in which intravenous urography fails to give entirely satisfactory evidence (cf. Figs. 86 and 87, p. 227).

*Estimation of Renal Efficiency*.—Tests for renal efficiency have become so numerous that it is wise to consider what information is demanded of them. In surgical work these tests are employed to guide prognosis and treatment in two main groups of patients. Firstly, if the removal of one kidney is planned, it is imperative to discover the efficiency of the other; secondly, in lesions of the lower urinary tract, an enlarged prostate for

example, it is important to know the total efficiency of both kidneys, in order that the correct treatment may be determined. In both groups a serious operation is contemplated which will throw a great additional strain on renal function. It is evident, therefore, that the importance of these tests lies not in their indication of the work done by a kidney or kidneys under normal conditions, but in their expression of renal adaptability to sudden strain. By such a criterion should all tests be judged.

The tests are :—

- A. Tests of retention, *i.e.*, of substances in the blood.
- B. Tests of excretion, *i.e.*, of substances normally in the urine or of a foreign drug specifically administered.
- C. A combination of the two.

**A. Tests of Retention.**—The urea concentration in the blood is the best of the retention tests. The normal amount varies between 15 and 40 mg. of urea in 100 c.c. of blood. Sixty milligrams indicate a negligible retention, but beyond that figure a danger signal has been shown.

**B. Tests of Excretion.**—Tests of the urine require that, if the function of one kidney only is being tested, its urine must be collected separately by ureteric catheter. The concentration of urea in the urine provides an excellent test, but is misleading unless properly carried out. The technique is as follows : In the evening of the day before the test the patient is given a light meal at 6.30 P.M., after which no food is allowed until the test is completed. The next morning at 7 A.M. 15 grm. of urea are given by mouth with a draught of water. At 8 A.M. the bladder is emptied and this urine is discarded (because it is the result of the diuretic effect of the urea); and then specimens are collected at 9 and at 10 A.M. and examined for the amount of urea. In this way the kidneys have been subjected to a sudden overload and an indication is given of their capabilities under stress. In normal urine there is about 2 per cent. urea, but under the conditions of the test healthy people will secrete 4 per cent. or even more. A reading under 3 per cent. is a danger signal.

Dye tests have been popular, methylene blue, indigo carmine, rosanilin, phloridzin and phenolsulphonephthalein having been extensively used. They are injected intravenously, and the times of their first appearance, their maximum intensity of secretion and their total excretion are noted. They may serve some minor function in urology, but they must be regarded as inadequate tests of surgical renal efficiency.

The method of choice lies in the combination of the urea concentration tests in both blood and urine. They have been grouped together under the term "the urea clearance test," and this affords the most reliable guide. But a general clinical examination of the patient must always remain the determining factor in the making of a difficult decision.

### CONGENITAL ANOMALIES OF THE KIDNEY

These may be :—

- A. In number—either excess or deficiency.
- B. In shape—either foetal lobulation or fusion variations.
- C. In position—either abdominal or pelvic.
- D. In mobility.
- E. In blood supply.

An **additional kidney** is very rarely seen, and most supposed cases are deformities of development of one kidney. **Total absence** of both

kidneys means the production of a dead monster. **Absence of one kidney** is rare, and a more common anomaly is the presence of an imperfect kidney with maldeveloped vessels and ureter. Such a kidney may be represented by a mere mass of fibrofatty tissue, or some renal tissue without glomeruli may be present; the ureter may exist but not be patent or it may be but a small remnant attached to the bladder. A solitary kidney is hypertrophied and may have a double ureter opening into the same side of the bladder. Such examples illustrate the need for a complete urological investigation before planning a nephrectomy.

**Foetal lobulation** is an arrest of development just short of perfection, the kidney substance and function being normal. **Fusion variations** imply union between the two kidneys, a condition known as "horse-shoe kidney." The connecting band of renal tissue is usually between the lower poles (Fig. 352), occasionally at the upper poles and rarely at the middle. There are two distinct pelves with ureters crossing in front of the connecting link. The band of tissue passes in front of the aorta and vena cava. The renal function will be normal but the fusion causes some difficulty in the technique of dealing with disease on one side.

**Misplaced kidneys** to be truly a congenital anomaly must have their vascular supply derived from an unusual origin, otherwise they are merely abnormally mobile. In abdominal misplacements they are probably normal in shape and size but placed too high, too low or too near the midline. In the pelvic type they are spherical not reniform, and usually lie between the common iliac arteries, from one of which arises the renal artery.

Abnormal branches of the renal artery are common, the best-known example being that which leaves the main trunk early and passes downwards and outwards to the lower pole; in doing so it may kink the ureter and lead to hydronephrosis.

Coexisting malformations are often seen in the genital tract of both sexes, *e.g.*, imperfect descent or abdominal retention of the testis, anomalies of the uterus and vagina, or absence of the ovary and tube on the same side.

### ANOMALIES OF THE URETER

Double ureters are common. They may be separate in their whole length with two openings into the bladder and two separate pelves;

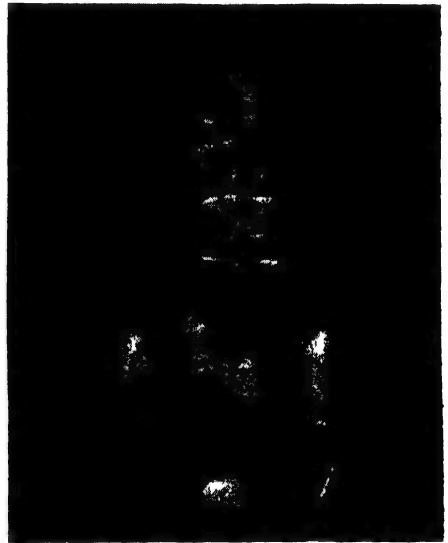


FIG. 352

The convex border of the horse-shoe connection between the two kidneys can be seen. A stone is present low down in the right ureter.

they may arise from two distinct pelves and fuse at once or they may



FIG. 353

A kidney having a double pelvis and double ureter. This specimen shows an early degree of hydronephrosis with obliteration of the pyramidal cups.

unite at any part of their course (Fig. 353). Occasionally a ureter of one side will cross the middle line and enter the opposite side of the bladder. It may open extravasically into a seminal vesicle or ejaculatory duct. Errors in canalisation of the original solid tube may lead to congenital strictures or to valve-like folds of mucous membrane, which may later produce hydronephrosis.

*Surgical Significance.*—Many of these anomalies pass undetected to the grave but, rare as they are, they occasionally produce surgical tragedies. So exact are the methods of renal investigation that there can be no acceptable excuse for the removal of a solitary kidney. It has also been said that the abnormally developed kidney is more prone to disease than the normal. No proof of such statement can be produced.

### MOVABLE KIDNEY OR NEPHROPTOSIS

The kidneys have a normal respiratory movement of 2 in. Beyond this they are abnormally mobile, and Glénard has described four grades of mobility.

1. When the lower pole
  2. When the greater part
  3. When the whole
  4. When the kidney is palpable without respiratory aid. This last degree is also called a "*floating kidney*."
- } of the kidney is palpable on inspiration.

The kidneys move with respiration in the fascial sac of Zuckerkandl, which is open below. The perirenal fat moves with the kidney, and such small fibres as do run from the perirenal capsule to the kidney are quite inadequate to maintain the latter in position; nor has the peritoneum any influence. There is thus no anatomical structure which can have a direct action in holding up the kidney. The chief factors are the abdominal musculature, the maintenance of normal fat content in the retroperitoneal space and a normal body form.

*Etiology.*—Movable kidney is more common in women (10 to 1), on the right side (15 to 1), and occurs most often between the ages of 25 and 50. It may be congenital in rare instances, but is usually acquired. The causes are not known but there are many predisposing

factors. There are well-recognised types of body form, each with its varying details of anatomical structure of thorax and abdomen. Among these the typical visceroptotic form combined with an atonic abdominal musculature will allow prolapse of the kidney. Scoliosis, child birth, a sedentary life, rapid wasting from disease, and the removal of large tumours all predispose to a "dropped" kidney. There is no evidence that injury has any etiological significance.

*Pathology.*—The kidney is normal in shape and size unless it has become hydronephrotic. The vascular pedicle is lengthened, but as it must retain its attachment to the aorta and vena cava, the moving kidney has to travel in the arc of a circle, and some axial rotation will occur. In some cases, the ureter may be so securely attached to the peritoneum that kinking occurs and obstruction follows. In no instance does the suprarenal gland follow the kidney in its movements.

*Symptoms.*—The extent of the mobility has no bearing on the symptoms, which in the great majority of cases are absent. When present they are renal, gastro-intestinal and nervous. The patient complains of indefinite pain or a sense of dragging in the loin, which may be increased by standing, exertion or during menstruation. If the ureter becomes kinked, severe attacks of pain occur, which are known as *Dietl's Crises*. The pain is sudden in onset, very acute, colicky in nature and accompanied by nausea or vomiting. The attacks cease as abruptly as they started. The gastro-intestinal symptoms are those of visceroptosis in general and of the underlying neurasthenia. In the nervous disturbances cause and effect are indistinguishable and the knowledge that a kidney is unduly movable is certain to accentuate the symptoms.

*Treatment* is directed towards the removal of any predisposing factor. Exercises to develop the weak muscles or remedy a scoliosis, and careful dieting to increase the weight are useful. The first essential is to allay any fears as to the presence of cancer. The diagnosis should never be mentioned, but the cause of the underlying neurasthenia must be investigated. Belts are to be avoided completely. Operation should be reserved for those patients in whom a pyelogram shows a definite hydronephrosis. In these cases operation (nephropexy) is necessary to preserve the function of the kidney, but apart from this surgery is not justified.

## INJURIES OF THE KIDNEY

*Etiology.*—Injuries are uncommon, over 90 per cent. occurring in men. The right side is affected more frequently than the left. Bilateral injuries are very rare indeed. They are produced by direct violence such as blows or kicks in the loin, by crushing in "run-over," or compression in "buffer" accidents and by indirect violence in severe muscular contractions.

*Pathology.*—The degrees of damage are :—

1. Tears of the fatty capsule with a perirenal hæmatoma ;
2. Subcapsular contusions, in which the blood is later absorbed ;

3. Interstitial rupture of the kidney parenchyma with or without tears of the capsule. Such tears radiate from the capsule and are chiefly on the anterior surface ;
4. Such tears may extend into the pelvis with resulting extravasation of blood and urine ;
5. Complete fragmentation of the kidney and
6. Associated tear of the peritoneum, which is very rare and more often seen in children.

Spontaneous healing will occur in the first two groups and in some cases in the third, but early surgical intervention will be needed in the others. Infection may follow in all types.

*Symptoms* are shock, pain, the formation of a hæmatoma, hæmaturia and other changes in the urine. Shock is severe, but depends more upon the general effects of a major injury than upon the actual renal damage. Pain is both renal and referred, whilst there is also associated pain due to local bruising of the abdominal and thoracic walls and possibly to other lesions such as fractured ribs. Renal colic may follow from the passage of clots. Hæmaturia is almost constant but varies greatly in amount. A renal hæmatoma may be felt in the loin within two hours of a serious rupture or it may take several days to become palpable. The tumour so formed will not move on respiration. Later, bruising of the skin may be seen at the external abdominal ring and in the scrotum. In some cases the bleeding may be so severe that the symptoms and signs of internal hæmorrhage rapidly appear and death may result. Oliguria follows most severe renal injuries, and should there be only one functioning kidney anuria is likely to result. During healing a polyuria succeeds the oliguria.

*Treatment.*—Many renal injuries will recover spontaneously, while others will die unless operated upon immediately. The shock will demand appropriate treatment at the beginning and after forty-eight hours the degree of injury should be clear. Slight cases should be watched for a few days. All cases of hæmaturia without hæmatoma may be safely treated by rest in bed and careful nursing for fourteen days after the bleeding has ceased. A hæmatoma large enough to be felt should be operated upon, because even if the renal injury is slight the danger of infection is too serious to risk.

Severe retroperitoneal hæmorrhage, intraperitoneal bleeding and extravasation of urine demand immediate operation, as the prognosis rapidly becomes hopeless.

Procedure at operation will depend upon the degree of injury, and in many cases a nephrectomy will be the only proper course ; whenever possible the kidney should be saved and tears in its substance, capsule or pelvis sutured.

**Injuries with External Wound.**—Such injuries are rare even in war. They may be due to stabs with swords, bayonets or knives, and wounds by bullets or shell fragments. There are usually coexisting injuries of other organs, and in large wounds the kidney may completely or partially prolapse. Two factors are of importance, viz., the external leakage of urine and the high percentage of infected wounds. The

symptoms are similar to those in the subparietal lesions, except that there will be a resultant urinary fistula in some cases. Perirenal collections of urine and blood are unlikely unless the track is narrow and tortuous.

The uninfected wound without urinary leakage will probably heal spontaneously. In general the treatment will be that of the wound itself, removal of any foreign body, and suture of the torn kidney and pelvis or removal of the organ if irretrievably injured. If prolapsed, it should be sutured in position before the wound is closed.

### HYDRONEPHROSIS

An incomplete obstruction, which is gradual or intermittent, to the outflow of urine from one kidney produces a distension of the pelvis and calyces, known as a hydronephrosis. Sudden complete blockage causes atrophy of the kidney with little distension.

*Etiology.*—It may be seen at any age, being more common in women and on the right side. If the obstruction is confined to one ureter the hydronephrosis is unilateral, but both kidneys are affected when either the prostate or urethra is the seat of obstruction. A partial hydronephrosis results from the blockage of one major calyx (Fig. 370, p. 743).

**Causes of Unilateral Hydronephrosis** are congenital and acquired. CONGENITAL LESIONS are chiefly due to errors of development at the ureteropelvic junction, leading to a stricture or to the formation of a fold of mucous membrane, which acts as a valve. An “aberrant” renal artery, which arises from the main vessel just after its origin and enters the lower pole of the kidney, may kink the ureter and obstruct the outflow of urine (Fig. 354).

THE ACQUIRED VARIETY has many causes. The lumen of the ureter may be blocked by a stone, a growth of the renal pelvis, foreign bodies or blood clot. Its wall may be narrowed by a stricture of infective, traumatic or neoplastic origin, and it may be compressed by tumours from without such as the pregnant uterus and carcinoma of the uterus or rectum. The abnormally movable kidney is an occasional cause of kinking of the ureter. A certain number of well-developed hydronephroses have no apparent cause, and it is believed that they are due to a defect in autonomic

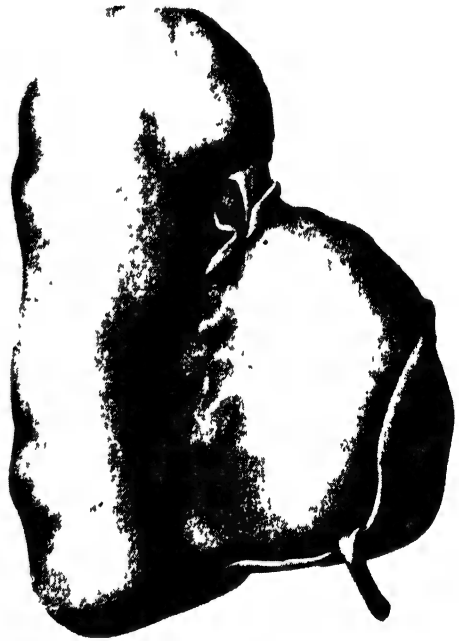


FIG. 354

A pelvic hydronephrosis due to an aberrant renal artery.



neuromuscular control leading to intermittent spasm of the uretero-pelvic junction.

**Causes of Bilateral Hydronephrosis** are found in the bladder and urethra and include vesical calculi, growths of the bladder, benign and malignant enlargements of the prostate, stricture of the urethra, carcinoma of the penis, phimosis and "pin-hole meatus." Very rarely valvular deformities or stricture of the urethra of developmental origin are responsible.

**Pathology.**—The earliest change is obliteration of the protrusion of the pyramid into the calyx, which instead of being cupped becomes rounded or clubbed; next there is a dilatation of the calyces and later a gradual distension of the pelvis, which is first shown by a flattening out of the normal

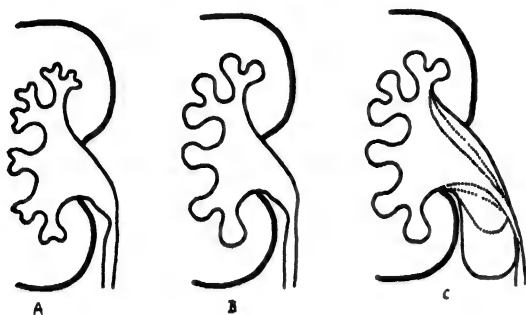


FIG. 355

A diagram illustrating the progressive stages in the formation of a hydronephrosis.

A, is the normal appearance, each calyx being cupped; B, shows the obliteration of the cups; and in C the dotted lines indicate the progressive bulging of the pelvis itself.

regular curve made by the outer wall of the ureter and the lowest calyx (Fig. 355). This stage is called a *pelvic* hydronephrosis. Eventually the renal parenchyma atrophies and the whole kidney distends and becomes lobulated, this being a *renal* hydronephrosis (Fig. 356). In moderate degrees of distension either of these two varieties may predominate.

**Symptoms.**—There are two clinical types, viz., the closed and the open or intermittent. The **Closed** may have no symptoms, or at most a dull aching pain in the loin, until a cystic tumour of renal origin can be recognised.

The **Intermittent** type is the more common. It is important that the condition should be recognised before the renal parenchyma has suffered any damage, so that the function of the kidney may be preserved.

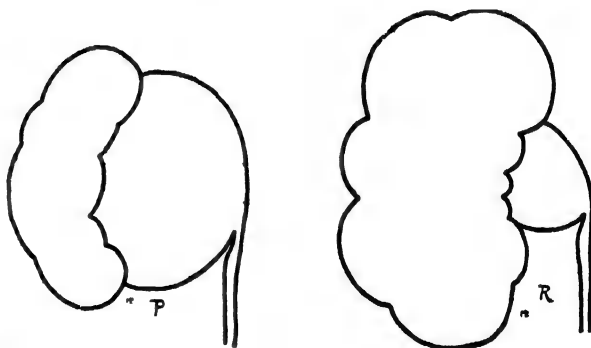


FIG. 356

A drawing illustrating the two types of hydronephrosis: the pelvic P, and the renal R.

*In the pre-Tumour Stage* the picture may be confused by a history attributable to the cause of the condition. The only symptom of the hydronephrosis is pain of renal type, which comes on in periodic attacks. A physical examination is negative and the urine is normal. Every patient who has renal pain and a normal urine should be



suspected of having an early hydronephrosis, and a pyelogram taken (Figs. 357-360).



FIG. 357

A bilateral ascending pyelogram illustrating a congenital hydronephrosis of the right kidney due to a developmental defect at the uretero-pelvic junction.



FIG. 358

A pyelogram of left kidney showing a hydronephrosis due to aberrant renal artery. Failure to recognise its presence led to persistence of a *B. coli* pyelitis in spite of treatment.

*The Tumour Stage* is characterised by recurrent attacks of pain, during which the patient is conscious of the gradual appearance of a tumour in the loin and of a diminished output of urine. The pain ceases, and after a time there is a copious polyuria accompanied by subsidence of the tumour. A pyelogram will confirm the diagnosis.

*Treatment.*—The underlying cause must be dealt with and in the early cases nothing else is required. In later stages plastic operations on the large flabby pelvis should be done to prevent retention of urine within it. A nephrectomy should not be performed unless the kidney is so destroyed that its function has become of no value. Those rare examples of sympathetico-renal tonus (Harris) are treated by stripping the renal pedicle, thereby severing the sympathetic nerves.

#### PRIMARY RENAL ARTERIAL HYPERTENSION

Recent work has proved that a small number of cases of hyper-



FIG. 359

An intravenous urography showing the appearance of a renal type of hydronephrosis of the left kidney.

tension are due to damage to one kidney; such trauma is usually to the vessels. It is believed that the devitalised renal tissue develops a "nephrotoxin" which leads to a high blood pressure. A recent case of the author's illustrates this condition. A young man suffering from hydronephrosis was operated upon and an aberrant vessel going to the lower pole divided. All symptoms due to the hydronephrosis disappeared, but within a few weeks the blood pressure was found to be over 200.

Although such cases are rare, ligature of branches of the renal artery must be abandoned in future and suitable plastic operations substituted. The only treatment for the established condition is nephrectomy.

## ACUTE INFECTIONS OF THE KIDNEY

### ACUTE PYELITIS

**Acute Primary Hæmatogeneous Renal Infection** is one of the commonest diseases met with in practice and at the same time one misdiagnosed more frequently than most.

*Etiology.* — Renal infections are very common in both sexes, a fact which should cause little surprise when it is recalled that the kidney is an organ of excretion, and organisms circulating in the blood will pass through the renal tubules. The condition is seen in patients of all ages from infancy upwards. In the first and second decades it is

equally distributed between the sexes, but after the twentieth year it affects females more than males. The greater liability of the female to constipation and the contiguity of her external genital organs to the anal region suffice to explain this incidence. The right kidney is more often affected than the left, but in many patients the infection is bilateral.

The infecting organisms are chiefly aerobic, being in order of frequency members of the coli group, staphylococcus aureus and albus, bacillus proteus, streptococci of various strains, occasionally other intestinal bacteria and very rarely pneumococcus and gonococcus. The paths of invasion are either by the blood stream, from the bladder

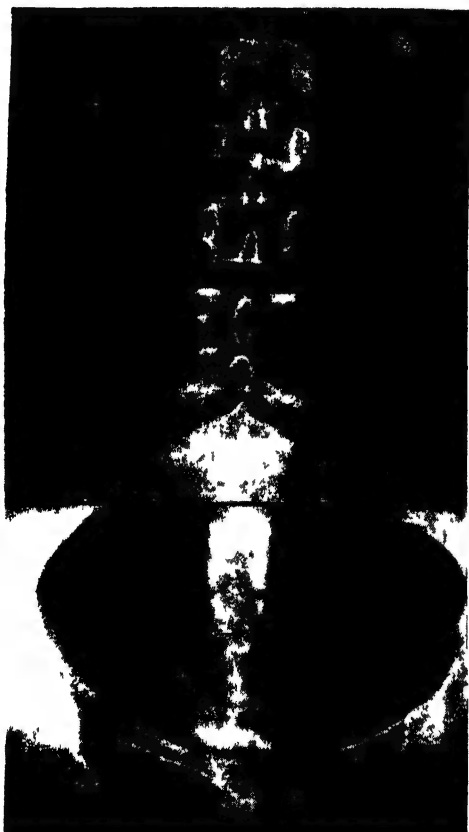


FIG. 360

A composite picture showing the great distension of the right ureter with a minor degree of pelvic hydronephrosis due to a ureterovaginal fistula.

below or via the lymphatics. The portals of entry are often impossible to define. Constipation, appendicitis, gastro-enteritis, cholecystitis and in women tears of the hymen, perineum and cervix may provide an opportunity for the absorption of coli organisms. Staphylococcal infections are usually derived from comparatively trivial skin lesions such as boils and carbuncles. The ascending type of infection from the lower urinary tract may give rise to pyelitis on one or both sides, but it is unlikely to occur unless there is some obstruction to the ureters or urethra. It is possible, though unlikely, that the right kidney may be infected by lymphatic spread from the cæcum.

Certain *accessory factors* play a considerable part in both the onset and duration of renal infections. The danger of an ascending pyelonephritis in cases of enlargement of the prostate and of stricture is well known, but an early unilateral hydronephrosis may be unsuspected at the onset of an acute pyelitis and be the cause of an unexpected failure in treatment. Any of the many causes of hydronephrosis may be found at operation. The slight degree of compression of the ureters by the gravid uterus explains the liability of pregnant women to pyelitis.

*Pathology.*—Acute infections of the kidney are invariably referred to as “acute pyelitis,” a term firmly established by long usage. It is, however, somewhat misleading, for the inflammatory reaction is not confined to the pelvis and there is a round-celled infiltration in the renal parenchyma and later minute areas of focal necrosis.

*Symptoms.*—There is likely to be an initial stage of weariness and lassitude, of which the patient thinks nothing. It will last not more than thirty-six hours. The classical symptoms are: (1) pain, (2) vomiting, (3) rigors, (4) pyuria, (5) high fever, (6) frequent and painful micturition, (7) headache.

Pain is renal in type; it is felt deep in the loin and is described as being in the back at the costo-lumbar angle and in the front of the abdomen below the costal margin. It may also be referred to the usual areas, *i.e.*, downwards and forwards into the groin and scrotum or labium majus and over Poupart's ligament into Scarpa's triangle. Vomiting usually occurs as an initial attack with the onset of the pain and is rarely continued beyond the first two hours. Rigors are so constant a feature that they are to be regarded as pathognomonic. Pus is always present in the urine, but it must be clearly understood that during the first twenty-four or forty-eight hours the quantity may be so small that it is difficult to demonstrate by chemical tests, and a microscopic examination will be needed in most cases.

High fever is characteristic, the temperature rising abruptly to 105° F. in severe cases. Frequent and painful micturition does not appear till the second or third day and is never a really prominent symptom.

Headache is present in every patient in a more or less mild degree, but occasionally it becomes so severe as to be the predominant feature. The clinical picture varies considerably according to the severity of the infection and three types are described.

1. **Acute fulminating pyelitis** has so severe and sudden an onset that it may be mistaken for the perforation of a peptic ulcer. The pain is excessive, the temperature rises to  $105^{\circ}$  F. and there will be one or more rigors. Bimanual palpation of the renal area reveals tenderness and rigidity of the muscles of the loin.

2. **Acute pyelitis** is of less intensity and of more gradual onset and is liable to be mistaken for acute appendicitis. The pain is less severe, the temperature rises to  $103^{\circ}$  F. and rigors are present. There is tenderness in the loin but little rigidity.

3. **Subacute pyelitis** gives mild pain, no vomiting, no rigidity and a temperature of  $101^{\circ}$  F. Rigors are slight and transient and pus is found in the urine in considerable quantity from the beginning.

*Diagnosis.*—(A) Right-sided pyelitis has to be distinguished from the perforation of a peptic ulcer which is characterised by low temperature, slow pulse and more widespread tenderness and rigidity; from acute appendicitis in which the pain begins centrally around the umbilicus and later travels to the right side, the temperature rarely reaches the height of pyelitis and there is never a rigor; from acute cholecystitis and gall-stone colic in which the previous history and the physical signs are quite different. Other lesions of the right kidney such as calculus, hydronephrosis and Dietl's crisis may exhibit equally severe pain but these conditions are not inflammatory. (B) Left-sided pyelitis gives rise to less confusion, acute diverticulitis, intestinal obstruction, torsion of an ovarian cyst and other diseases of the left kidney being possible sources of error.

*Treatment.*—The treatment of urinary infections has been entirely revolutionised by chemotherapy.

Sulphanilamide is given in the following doses :—

1st day .	.	.	.	four tablets of 7.5 gr. three times.
2nd „ .	.	.	.	two „ 7.5 „ „ „
3rd „ .	.	.	.	„ „ 7.5 „ „ „
4th „ .	.	.	.	„ „ 7.5 „ twice.
5th „ .	.	.	.	„ „ 7.5 „ „
6th „ .	.	.	.	„ „ 7.5 „ once.
7th „ .	.	.	.	„ „ 7.5 „ „

It has at least one advantage that it is active in both alkaline and acid urine; it is also less toxic than sulphapyridine.

Some patients cannot tolerate this group of drugs and for them treatment by ketosis obtained either by a ketogenic diet or mandelic acid will prove equally successful. The latter gives many people nausea and the former is not easy to prepare, nevertheless these two methods must be thoroughly understood and used in all patients who cannot tolerate sulphanilamide or who are not improving on it. Details of both can be found in textbooks of medicine.

**The Acute Pyelitis of Pregnancy** differs in no way from the condition described above. In spite of the pressure of the uterus on the ureters, treatment relieves the symptoms if not the bacilluria.

**The Acute Pyelitis of Children** is seen in both sexes and there is often a history of recent gastro-intestinal disturbance. The attack is

usually severe and follows the typical course as described above. It is emphasised here as its existence in children is not sufficiently realised and the diagnosis may be delayed on that account. Children respond well to the ketogenic diet, which will be preferable to mandelic acid in their case if the facilities exist for its provision. The pH may fall even to 4.8 in children under ten years of age.

**Acute Pyelonephritis** (acute secondary ascending renal infection) is usually bilateral. It is secondary to disease in or distal to the bladder, e.g., pelvic and puerperal infections in women and enlarged prostate and urethral stricture in men. The infecting organisms are usually staphylococci or streptococci. It is therefore a complication of pre-existing disease and only too frequently marks the closing scene of life.

*Symptoms.*—During the course of some other disease, either spontaneously or after instrumentation or operation, the patient suddenly has a rigor and the temperature rises steeply to 104° or 105° F. There is pain in both loins, nausea and drowsiness. The tongue is dry and furred and the abdomen distended. The secretion of urine is diminished or abolished. In the worst cases delirium and coma rapidly usher in a fatal issue, while in others, after some days of extreme anxiety the flow of urine is re-established and the condition subsides, leaving permanently damaged kidneys.

*Treatment* should be prophylactic and no surgical interference carried out until the renal function has been estimated. Active drainage must be obtained by suprapubic cystotomy done under a local anaesthetic, and a constant intravenous drip of 5 per cent. solution of sodium sulphate and 10 per cent. glucose in normal saline given.

**Catheter Fever or Urethral Chill.**—Sometimes after the passage of a urethral bougie or catheter, patients have a rigor and a rise of temperature. The time which elapses after instrumentation and the severity of the attack vary greatly. This condition is due to a temporary recrudescence of a renal infection as a result of urethral interference. Such attacks, when first experienced, are an indication for a routine urological investigation; for mild recurrent cases, instrumentation should be followed by the administration of quinine sulphate, gr. v, by the mouth.

**Pyonephrosis.**—A distension of the pelvis and kidney with pus may be due to infection of a previously existing hydronephrosis, the result of a slowly progressing pyelonephritis, or the terminal stage of renal tuberculosis.

*Pathology.*—The infecting organisms are *Bacillus coli*, staphylococci or streptococci. Superimposed on the antecedent condition there is a distension of the kidney and pelvis with pus, and the perinephric fat is adherent, infiltrated and oedematous. The lining of the cavity is in the later stages a thick-walled pyogenic membrane. A partial pyonephrosis may occur when one calyx only is distended and infected.

*Symptoms.*—Pain is constant, severe and renal in type. The urine contains pus in varying amounts and in some of the more

chronic cases there will be attacks of retention in the affected kidney when the urine will be clear and free from pus. The patient may be aware of a swelling in the loin and this can always be felt on palpation. The general condition is poor and there is a febrile swinging temperature. Cystoscopy will reveal a purulent efflux from the affected ureter, unless the pyonephrosis be closed, in which case massage of the loin may produce a discharge of thick creamy pus, resembling the appearance of tooth-paste from a collapsible tube.

*Treatment.*—Nephrotomy and drainage are the first essentials, and if the cause of the obstruction is easily reached, it should be dealt with, *e.g.*, the removal of a calculus. In many severe cases a rapid nephrotomy alone is possible, and more radical measures are delayed until the general condition has improved. Later nephrectomy may be needed if the kidney fails to recover.

**Perinephritis.**—The sclerosing type of fibrolipomatous perinephritis is seen in a mild degree in every case of renal infection, whatsoever the cause. The thickened, fibrous fat is adherent to the kidney and gives no symptoms. In some patients, notably those with a long-standing calculous pyonephrosis, a large tumour is formed.

**Perinephric Abscess.**—*Etiology.*—There are two forms: primary and secondary. **Primary** may result from infection of a hæmatoma following injury to the kidney, but is more often seen in connection with trivial skin infection, *e.g.*, boils, and is invariably staphylococcal. It is more frequent in men, on the right side and in the active years of adult life. The infection is carried by the blood stream and deposited in the fat, or more probably the glomeruli of the kidney filter off the organisms, and a small subcapsular abscess develops. This bursts through the renal capsule and pus enters the perinephric fat.

**Secondary** abscesses occur as complications of other inflammatory diseases, such as appendicitis, cholecystitis, salpingitis, liver abscess and empyema. A chronic tuberculous perinephric abscess may result from disease of the vertebral column, ribs or pelvis.

The abscess may be above the kidney, in which case the appearance of a swelling will be long delayed; it may be postero-inferior, when it presents in the back and loin.

*Symptoms.*—In the secondary variety the symptoms merely complicate the previously existing disease.

In the primary form the onset is usually misleadingly vague. There is a history of recurrent boils, during which time the patient is a little run down. Then there may be an interval after the last boil, and later a distinct decline in the general condition with fever and great weariness. Soon a swinging septic temperature is established, with some pain in the back, often thought to be lumbago. Occasionally the onset is abrupt, with severe pain and a rigor. The local signs may be absent or trivial, and include some deep tenderness over the kidney, pain and limitation of movement in the hip joint on the affected side. Later the loin will be filled up with a swelling that bulges in the back but does not spread forward to any extent. It does not move on respiration, nor on palpation. In the suprarenal

type there may be no palpable swelling. Pus in the urine is a variable sign, but in the early stages staphylococci will almost always be present.

*Diagnosis* may be unusually difficult. The history and the general condition seem to point so obviously to a perinephric abscess, and yet the long delay in the appearance of a swelling may be most misleading. An X-ray photograph may assist in obscure cases by showing a raised or immobile diaphragm on the affected side.

*Prognosis* in primary cases is excellent, but in secondary cases depends upon the original condition.

*Treatment.*—Incision and drainage are needed as soon as a palpable swelling is present. If a pyonephrosis coexists it must be drained also and any causative lesion will need appropriate treatment. It is wise to withhold operative interference until localising signs are evident. Perinephritis without abscess formation should be treated in a manner similar to tuberculosis; in these cases incision can do no good and may do harm.

**Carbuncle of the Kidney.**—Metastatic staphylococcal infection of the cortex does not always follow the sequence of events described under perinephric abscess. Instead of a small subcapsular abscess a more chronic indurated swelling is formed; this spreads slowly and shows little tendency to form pus. In time a considerable area of the kidney is invaded by this chronic indurated lesion, to which the name "carbuncle of the kidney" has been applied (Fig. 361).

The clinical picture is indistinguishable from that of perinephric abscess in its early stages before an external swelling has appeared, but a pyelogram may provide the diagnosis. Many weeks of pyrexial illness are to be expected.

These patients are treated in bed in the open air in a manner similar to cases of tuberculosis. Sulphathiazole may bring about a rapid improvement. In resistant cases a nephrectomy may have to be considered if the kidney is extensively destroyed.



FIG. 361

Carbuncle of kidney.

## CHRONIC INFECTIONS OF THE KIDNEY

### CHRONIC PYELITIS

Chronic pyelitis, once a common condition, is rapidly becoming an index of missed diagnosis or imperfect treatment, and in time its very existence will be a reproach. The introduction of the ketogenic diet was

accompanied by the return—in large numbers—of old patients with chronic pyelitis to see if at long last a cure had been found. Chronic pyelitis follows an unresolved acute attack and is characterised by the presence of pus and bacteria in the urine and slight symptoms of general loss of condition. These patients are liable to constant exacerbations of the infection and are suffering from a definite, if slight, diminution of renal function. Each attack takes away a little more of their reserves and their vitality. The infection in such people is not limited to the renal pelvis, but is a true pyelonephritis, and all forms of treatment such as alkaline and acid diuretics, vaccines and pelvic lavage are equally futile.

*Treatment.*—At the present time a patient with chronic pyelonephritis must be subjected to a complete urological overhaul. The excretion from each kidney must be investigated for the presence of organisms, for any abnormal constituent and for the efficiency of that kidney. If the findings are compatible with the diagnosis of a simple urinary infection on one or both sides, treatment by sulphanilamide is started forthwith. If this fails to sterilise the urine it must be discontinued. In long-standing cases, in which the infection is confined to one kidney, the other being uninfected and efficient, and if a pyelogram of the affected side shows appearances suggestive of renal damage, then it will be wise to consider removal of the infected kidney.

#### SYPHILIS OF THE KIDNEY

Bilateral subacute parenchymatous nephritis occurs in the secondary stage and is accompanied by a slight albuminuria and oedema of the feet. It is usually transient, but a few cases are on record of death from renal failure. Tertiary syphilis gives rise to chronic interstitial nephritis and to gummata, which latter are usually mistaken for neoplasms. These manifestations are uncommon.

#### BILHARZIOSIS OF THE KIDNEY

This is very rare, the kidney being in a state of chronic nephritis and the pelvis inflamed and ulcerated. Ova may be found in the pelvis, in the kidney and in the subcapsular cysts.

#### ACTINOMYCOSIS

This is likewise very rare and in appearance differs in no way from its general characteristics elsewhere.

#### RENAL FISTULA

The great majority of these fistulæ follow operations, but three types are described. Perirenal fistulæ unconnected with the urinary tract are due to perinephric abscess, appendicitis, empyema or disease of the ribs or spine. Spontaneous renal fistulæ are rare, but may be



seen in neglected cases of pyonephrosis, in which the pus tracks towards the skin and finally bursts through. Post-operative fistulæ imply either that the ureter is still obstructed, that imperfect drainage has been established or that the infection is tuberculous. Appropriate treatment of the cause should suffice to cure the condition, but nephrectomy may be required.

### RENAL TUBERCULOSIS

Renal tuberculosis occurs in an acute miliary form, which is usually rapidly fatal and has no surgical interest, and also as a chronic surgical infection. This type is never the primary focus in the body, but is secondary to other lesions, *e.g.*, in the lungs, the cervical or mediastinal lymph glands or the epididymis.

Tuberculosis is one of the more frequent surgical lesions of the kidney, occurring most commonly between the ages of 20 and 40 years and being very rare in infancy and old age. The right side is slightly more frequently affected than the left, and women than men. In its early stages it is rarely bilateral, but in untreated cases over 60 per cent. of patients die with both kidneys infected.

*Pathology.*—The tubercle bacillus reaches the kidney in one of three ways: either via the renal artery from a distant focus, by the lymphatics around the ureter carrying the infection from the bladder or by direct extension from neighbouring structures, *e.g.*, the ribs, spine or bowel. Three stages in the progress of the infection may be described:—

1. **Pyramidal Origin.**—The earliest lesion is seen at the base of a pyramid and not at its apex, as was previously taught (Fig. 362). Spreading centrifugally it will destroy the pyramidal tissue and soon reach the mucous membrane of the calyx. Erosion of the renal parenchyma continues and is now accompanied by spread along the calyx in the submucous layer. By this means the infection passes into the neighbouring calyces and the pelvis.

2. **Ultero-cavernous Stage.**—Ulceration of the calyx and erosion of the renal parenchyma are proceeding simultaneously. Each calycal system tends to remain isolated from its neighbours by fibrous tissue, and there may be seen four to seven separate areas of caseation in varying stages of progress, spreading slowly through the kidney cortex.



FIG. 362

A left kidney with three distinct lesions illustrating the stages of tuberculous infection. In the centre is a small early lesion which demonstrates the position where a surgical tuberculous lesion starts. At the upper pole a moderately advanced lesion is seen, and at the lower pole complete caseation has occurred.

3. **Tuberculous Pyonephrosis.**—Finally, when each main centre has reached the capsule complete destruction of the kidney occurs and all that remains is a multilocular cavity containing caseous pus (Fig. 363.)

**Involvement of the Ureter.**—The infection having reached the pelvis, involvement of the ureter is certain. The disease spreads in the submucous coat, whence ulceration of the mucous membrane follows together with an infiltration and thickening of the whole ureteric wall. This process, starting at the ureteropelvic junction, travels down the ureter and finally reaches the bladder, in which the lesions first appear in the mucous membrane above and external to the orifice over the course of the intramural ureter.



FIG. 363

A tuberculous pyonephrosis, the lower half of which retains the caseous material.

**Symptoms.**—Frequency of micturition is the earliest symptom and is noticed during the day, but as the disease progresses it will be present throughout the twenty-four hours. In the later stages it becomes most distressing, urine being voided every quarter of an hour, and finally the bladder becomes so contracted that incontinence is established. Polyuria is a constant and an early symptom and can be shown to be localised to the affected side.

The urine shows certain definite changes. Pus will be found intimately mixed with the urine, but on standing it will settle, leaving perfectly clear urine above it. Moreover, even on prolonged

standing the urine remains clear, which it fails to do in any other infection. Tubercle bacilli will always be found if examined for by correct methods. It is useless to rely upon the examination of a specimen containing but a few ounces of urine. A full twenty-four hours' collection must be examined. This will not fail to show tubercle bacilli if the centrifuged deposit be examined with care. Blood is a variable constituent; in some patients a brisk hæmaturia is the initial symptom and in most cases red blood cells will be found in the deposit. Albumen may come from the affected kidney or from the other side, due to a toxic nephritis. Its presence alone is not a sign of tuberculosis, but if unaccompanied by any other abnormal constituent it should lead to the examination of a twenty-four hours' specimen.

Pain is conspicuous by its absence, and even when present is frequently in the unaffected kidney as a result of compensatory hypertrophy.

*Localisation of the Infection.*—In a patient, whose urine contains tubercle bacilli, two questions have to be answered : first, is the infection localised to one kidney ? second, can the other kidney be proved free of infection and also efficient ? Intravenous urography (Figs. 364 and 365) will demonstrate the side of the lesion in over 95 per cent. of patients, and ureteric catheterisation of the opposite side will provide the answer to the second question. Cystoscopy will reveal the extent, if any, of bladder involvement and the specimen collected from the unaffected ureter will be tested for bacilli and for the percentage of urea. If it is

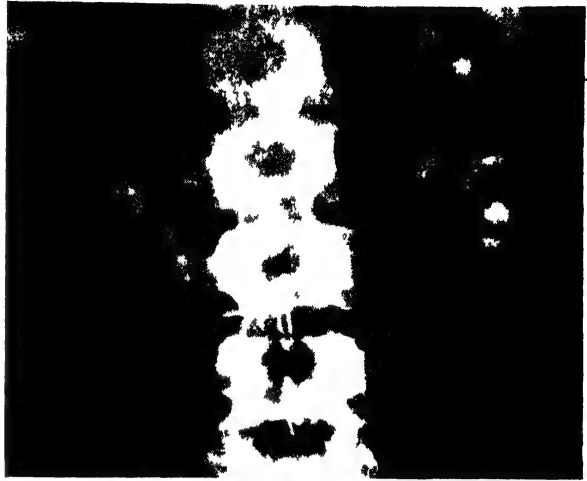


FIG. 364

Ultero-cavernous tuberculosis of upper pole of left kidney.

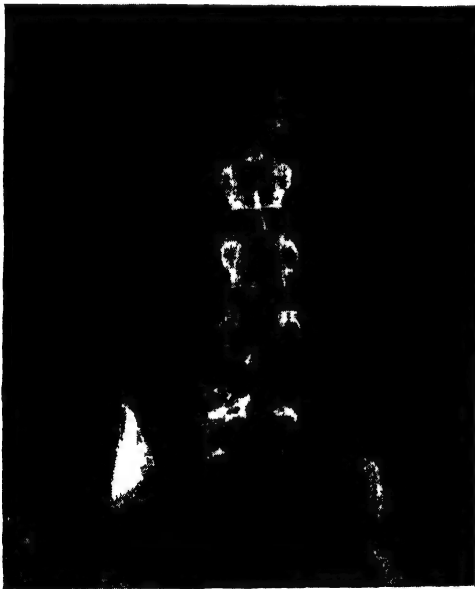


FIG. 365

An intravenous urography showing complete absence of secretion by the right kidney which can nevertheless easily be seen with mottled areas of calcification throughout it. This is the kidney shown in Fig. 363. The left kidney is seen to have a double pelvis and ureter.

a round hole, which projects into the bladder lumen and so stands out on an eminence. As the congestion becomes more intense, the efflux is

found impossible to arrange an intravenous urography, the localisation must be done by cystoscopy alone. Should the bladder be normal, catheters are passed up both ureters and the urine of each examined for bacilli, the negative specimen being estimated quantitatively for urea. Tuberculous patients do not tolerate instrumentation well and all the information required must be obtained during one cystoscopy. The changes which occur around the ureteric orifice are well defined and usually go through the following stages. First, there is hyperæmia with capillary congestion surrounding the orifice so that the typical appearance of the mucous membrane gives place to a dull red suffused zone with the orifice in the middle of it. Later the thickening of the ureter converts its opening into

sluggish and prolonged. Later still, along the line of the intramural ureter, small tubercles form which later develop into grey ulcers. In the stage of healing the contraction of the ureter leads to the drawing up of the orifice in a funnel-shaped manner.

*Treatment.*—A tuberculous kidney must be removed, provided no contraindications are present. These are: (1) the presence of tuberculous infection in the other kidney; (2) active tuberculous lesions elsewhere, *e.g.*, in the lungs, joints, peritoneum and other parts of the genito-urinary tract (the bladder excepted); (3) the absence or inefficiency of the other kidney; and (4) serious general illness such as would preclude any operation. The operation should include the removal of the ureter as far as the pelvic brim. The *after-treatment* is as important as the operation. Patients should be sent to convalesce in the open air, but there is no need to insist upon the full rigour of sanatorium treatment. In six months time they are examined for any evidence of urinary tuberculosis, and as long as tubercle bacilli persist in the urine they must continue to live an open-air life. No patient may return to an indoor occupation until three months have elapsed since the urine has been proved free of bacilli. The treatment of those patients, who are not suitable for nephrectomy, should be a full institutional régime.

## RENAL CALCULI

### GENERAL CONSIDERATIONS OF STONE FORMATION IN THE URINARY TRACT

The principles of stone formation are the same in every part of the urinary tract. As yet there is no completely acceptable explanation of them. Changes in the reaction of the urine, though affecting the constitution of a stone, have nothing to do with their initial formation. The following facts are known:—

- 1 Crystals must be present in the urine owing to supersaturation with one of its normal constituents, but this alone is not sufficient for stone formation, as they may be passed as such in large numbers (see "Oxaluria," p. 742).

2. Certain colloids are present in normal urine, but it is believed that an abnormal colloid, of an irreversible type, is needed to act as a cement substance to weld the crystals together into a stone nucleus.

3. A nucleus having formed, neither of these two factors need continue, for such a nucleus will continue to grow if it remains in contact with constantly changing normal urine.

4. Neither an adventitious nucleus nor an infection is necessary for stone formation in the urinary tract.

5. Prolonged recumbency during treatment of such diseases as anterior poliomyelitis and

6. Alterations in calcium metabolism, both favour stone formation.

Reversal of reaction of the urine causes a difference in the constitution of the stone, and mixed calculi can occur. For example, if a stone of calcium oxalate is developing in an acid urine and a staphylococcal infection occurs, making the urine alkaline, then oxalate



FIG. 366

A mixed stone which has almost completely destroyed the kidney. The centre is composed of calcium oxalate, followed by a zone of indigo, a narrow strip of oxalate, a wide area of uric acid and finally phosphates.

deposition ceases and phosphates are laid down. If the infection is now cured by treatment and the acidity restored, further oxalates will be added to the calculus (Fig. 366).

The relative frequency of the different types is impossible to assess, as statistics vary so much throughout the world. The stone may be single or multiple, round or branched. Irregularities of diet predispose to supersaturation and the heat of tropical climates tends to produce a highly concentrated urine. Both these factors may therefore contribute to stone formation. Heredity plays an important part in cystin lithiasis, but is of no import in the other types.

### COMPOSITION

The various substances which may form urinary calculi are :

- (a) Those in an acid urine—uric acid, ammonium or sodium urate, calcium oxalate, cystin, xanthin and indigo. (b) Those in an alkaline urine—calcium phosphate, magnesium-ammonium phosphate and cholesterin. Their physical characteristics are set out on the following page.



FIG. 367

Multiple calculi with a renal hydronephrosis and atrophy of the renal parenchyma.

**Renal Calculi** are bilateral in nearly 50 per cent. of cases. When unilateral they affect either kidney equally. They are more common in men, and the years between 20 and 50 provide 73 per cent. of the total.

The effects on the kidney vary considerably. Some stones cause little or no damage, but usually some renal deterioration ensues. Chronic interstitial nephritis is the most insidious complication ; hydronephrosis may follow im-

packtion of a stone in the ureter ; atrophy results when a stone is tightly gripped in a calyx ; when the stones are multiple (Fig. 367) a gradual fibro-fatty replacement of the whole kidney parenchyma may follow ; and superadded infection increases the renal damage by producing pyelonephritis and pyonephrosis.

**Symptoms.**—Position, size and mobility of the stone influence the history, and three groups can be described. The **Silent Stone**, which either forms and is embedded in the cortex, or is sufficiently large to be tightly gripped in a calyx, will cause no symptoms, being discovered only in a routine examination or at post-mortem. Generally speaking, also, the larger the stone the fewer the symptoms. The **Mobile Stone**, which moves about in a calyx or in the pelvis, gives rise to pain and hæmaturia, both of which are made worse by exercise or jolting and are improved

TABLE SHOWING THE PHYSICAL CHARACTERISTICS OF URINARY CALCULI

	REACTION OF URINE.	CONSISTENCE.	COLOUR.	OUTER SURFACE.	CUT SURFACE.	CRYSTALS.
Uric Acid .	Acid.	Hard.	Brown or dirty yellow.	Smooth and regular or smooth and lobulated.	Regular concentric laminae.	Polymorphous.
Urates .	Acid.	Soft and friable.	White.	Smooth and later dendritic.	Homogeneous.	Amorphous.
Oxalates .	Acid.	Very hard.	Dirty grey.	Mulberry or jagged crystals.	Wavy concentric laminae.	(a) Octahedrons (envelopes). (b) Dumb-bells (short bar).
Phosphates .	Alkaline.	Soft and friable.	White.	Smooth and dendritic.	Homogeneous.	Calcium, stellar. Triple, knife rests and coffin lids.
Cystin .	Acid.	Neither hard nor friable.	Honey turning greenish blue.	Smooth and soapy.	Homogeneous radiating fissures.	Flat hexagonal plates.
Xanthin .	Acid.	Hard.	Red or cinnamon.	Smooth.	Homogeneous.	Whetstone.
Indigo .	Acid.	Firm.	Blue-black.	Polished.	Homogeneous.	
Cholesterin .	Alkaline.	Firm.	Honey coloured.	Smooth.	Homogeneous radiating fissures.	Rhomboids with an angle missing.

by rest. The pain is both renal and referred. The **Migratory Stone** attempts to leave the pelvis and enter the ureter. It may succeed in passing to the bladder at the first attempt; it may progress some distance at the first effort, but reach the bladder only after several attacks of colic; it may remain impacted in the ureter and need removal or it may be held at the ureteropelvic junction during the attack and then fall back free into the pelvis. The migratory stone causes very severe attacks of renal colic, which can be controlled only by morphia. Vomiting at the time of onset is usual and sometimes persists throughout the attack, which will be terminated

by the release of the stone, if it again becomes free in the pelvis. Should it succeed in entering the ureter the colic continues and its symptoms will be described later.

*Physical Signs.*—

1. Examination of the patient will reveal little. Some deep tenderness in the loin or at the costolumbar angle may be elicited. Heavy percussion over the renal area behind may produce sharp stabs of pain, but this test should not be employed. A typical zone of renal hyperæsthesia is frequently found.

2. Examination of the urine reveals abnormal constituents in between 70 and 80 per cent. of cases in the form of albumen, pus, blood,

crystals or bacilli. A trace of blood and albumen is common, but pus comes only after a superadded infection. A non-catheter specimen, especially in women, is valueless, whereas a few pus cells or blood corpuscles found in a catheter specimen may be of great importance.

3. X-ray examination shows the great majority of stones. Doubtful shadows can be identified by intravenous urography or ascending pyelography (Figs. 368 and 369).

*Diagnosis.*—Any of the surgical diseases of the kidney may cause some difficulty, but a complete urinary investigation leads to certain differentiation. The following non-renal lesions may cause considerable doubt—cholecystitis, gall-stones, peptic ulceration, appendicitis and mesenteric adenitis—but renal and gastro-intestinal radiography

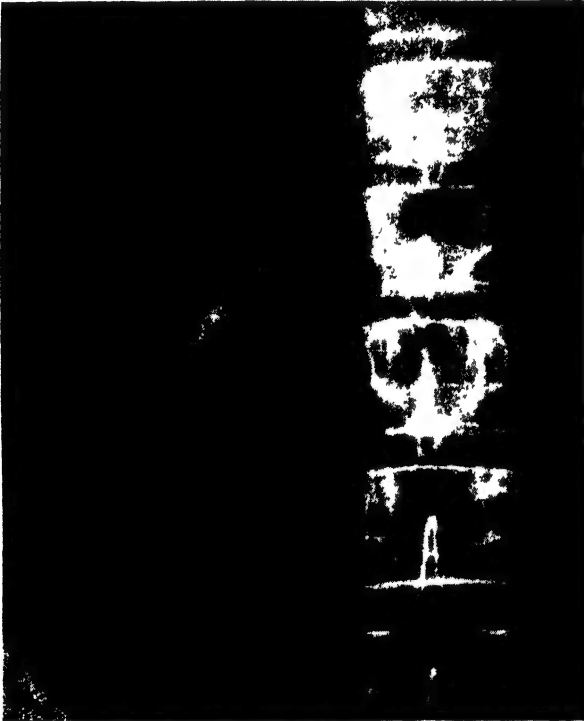


FIG. 368

X-ray showing a dendritic calculus filling both pelvis and calyces.



should solve the difficulty. Subluxation of the sacro-iliac joints, osteo-arthritis of the spine and the various types of cerebrospinal syphilis with tabetic crises should be considered in difficult cases.

**Treatment—(A) Unilateral Calculi.**—The silent stone can safely be left alone and its rate of growth checked by X-rays every six months. In a sterile urine and in the absence of growth it may be left indefinitely.

Stones in the calyces and pelvis should be removed. The absence of infection is no reason for leaving them, but its presence makes operation essential. Similarly, those stones which give rise to colic by intermittent impaction at the ureteropelvic junction must be removed. The urgency lies in the fact that such stones damage the integrity of the kidney and on this account their removal should not be delayed.

**(B) Bilateral Calculi.**—Cabot describes the treatment under four headings: (a) large calculi in both kidneys; (b) large calculus in one kidney, small one in the other; (c) moderate-sized calculi in both kidneys; (d) small calculi on both sides. A patient with large calculi in both kidneys is in less danger than those in group (b). Large bilateral calculi imply that they have formed more or less silently over a long period. The kidneys are damaged but fairly well compensated, and in the absence of infection there is some difficulty in coming to the right decision. The age and general condition of the patient will decide the issue, the younger and sounder patient giving the best results. If operation is considered, the kidney which shows the better renal function should be dealt with first.

In the case of a large calculus on one side and a small one on the other the latter should be removed at once, so that any further damage to that kidney may be prevented and at a later date appropriate measures taken to deal with the other kidney. In the last two groups the patient is unknowingly faced with the very serious complication of calculous anuria, and for this reason the stones should be removed as early as possible, the more efficient kidney being dealt with first.

**Pyelotomy** is the operation of choice for all stones in the renal pelvis and for most in the calyces except in the presence of an advanced infection when a tube through the renal cortex is desirable for drainage. Pyelotomy does no injury to the renal cortex and the incision can easily



FIG. 369

An ascending pyelography illustrating a filling defect of the right renal pelvis caused by a non radio-opaque calculus

be sutured, healing readily without the danger of a urinary fistula. After the removal of a stone a bougie must be passed down the ureter to prove that there is no obstruction. Nephrotomy should be confined to those cases in which either a branched stone is too large and irregular to extract through an opening in the pelvis, or a stone is more or less shut off in one calyx. In that it damages renal tissue this operation should be used only in especially difficult cases.

Nephrectomy is reserved for those cases of calculous pyonephrosis in which the kidney is practically destroyed. It should not be performed unless the surgeon is certain that no hope of any recovery in the function of the kidney remains. If there is any doubt, a nephrotomy with drainage is the proper procedure.

*Complications.*—The following complications may be seen : infection, obstruction, fibrosis and atrophy, development of carcinoma and calculous anuria. Infection follows stone in a large proportion of cases and leads to pyelitis, pyelonephritis, pyonephrosis and later possibly to a perinephric abscess. Obstruction leads to hydronephrosis (Fig. 370), and if this becomes infected a pyonephrosis results. A carcinomatous ulcer of the renal pelvis is a rare occurrence. Calculous anuria will be described at length as a complication of stone in the ureter (p. 752).

#### OXALURIA

Crystals of all its constituents may be found in the urine, but only those of calcium oxalate present a definite clinical condition known as oxaluria. This is usually associated with a nervous dyspeptic temperament, and the urine is found to contain a cloud of mucus with numbers of calcium oxalate crystals. It is believed that most, if not all, of the calcium oxalate in the urine is derived from ingested food, and certain articles of diet are known to have a high oxalate content. Generally, therefore, this condition is of more interest to the physician than to the surgeon, but occasionally it presents itself to the latter with an attack of colic, caused by the sharp edges and angles of the crystals when present in very large numbers ; the colic is as severe as that due to calculus, and blood will be found in the urine. Such a picture provides a difficulty in diagnosis, and unless oxaluria is remembered as a possible cause of renal colic, other more serious conditions may be suggested.

#### CYSTS OF THE KIDNEY

The cystic conditions seen in the kidney are :—

1. Congenital polycystic disease.
2. Single cysts.
3. Multiple cysts in chronic interstitial nephritis.
4. Cyst formation in new growths.
5. Parasitic cysts.

The multiple cysts in chronic nephritis are of no clinical interest. Cyst formation in new growths is due to degeneration in a hypernephroma or is seen as an essential process in the teratoid tumours, and as such are merely incidental in the history of the tumour.

### POLYCYSTIC DISEASE

This is a condition in which both kidneys are slowly destroyed by the formation of cysts throughout the renal substance. It is seen



FIG. 371

Congenital polycystic kidney.

in two periods of life. The infantile type appears very early, sometimes being the cause of an obstructed labour or of a dead foetus, and in other cases resulting in death before 6 years of age. The adult type rarely gives symptoms before 40 and is commonest between 45 and 55 years. Women are more frequently affected than men and there is some evidence of an hereditary factor.

*Pathology.*—The condition is always bilateral, but one kidney is often larger than the other. The swelling retains the shape of the kidney even when it reaches a large size. The surface is irregular with thin-walled cysts projecting from it, and the whole organ is riddled with spaces of varying size and colour, between which the renal tissue is flattened, atrophic and unrecognisable. The pelvis itself and the connections between it and the calyces are elongated and narrow, but the latter

are normal in contour (Fig. 371). The fluid in the cysts contains urinary constituents. The condition may be associated with a similar change in the liver, pancreas and spleen, and with other congenital anomalies such as cleft palate, imperforate anus or club foot. Microscopically, the cysts are lined by flattened or cubical epithelium standing on a thin wall.

The origin of this condition is developmental. The kidney develops from two separate tissues. The ureter, pelvis and straight collecting tubules are derived from the ureteric bud of the Wolffian duct, whereas the glomeruli and the convoluted tubules arise from the metanephric cap. These two sets of tubules must unite before a functioning kidney is completed. Presumably in the infantile type there is widespread failure of union, and in the adult group a sufficient number have joined to carry on the function of secretion until the kidney is later being subjected to toxic and metabolic disorders.



*Symptoms.*—In the infantile group, if the child is born alive, it is an ailing weakly baby and dies of renal failure in its early months. In the adult type the symptoms are certain indefinite changes in the output of urine, the presence of a tumour, hæmaturia and pain. Many of these patients have a most vague history; some discover a tumour in the loin quite by chance, in others hæmaturia may be the initial symptom, whilst others notice that they have alternating periods of oliguria and polyuria. The mass of the tumour may cause a dull dragging pain. Sooner or later the second kidney becomes palpable. In general, patients remain well for a long time with little evidence of renal failure and the final picture of anuria is abrupt in onset and short in duration.

The diagnosis in the stage when one kidney only is palpably enlarged is apt to be confusing, especially if there has been hæmaturia, but the appearances on pyelography are so typical that a neoplasm can be excluded with confidence. The prognosis is difficult and no prophecy as to time can be made with any safety. These cases vary widely in the interval that elapses between the recognition of the tumour and death, but the prognosis is more favourable when the tumour is first seen in later life.

*Treatment.*—No surgical treatment is of any avail. If the kidney has been exposed under a mistaken diagnosis, Rovsing's procedure may be adopted. This consists in puncturing all the cysts visible, but its results do not justify its adoption as a set operation. General medical care should be directed to relieving the strain on the threatened renal function.

#### SINGLE CYSTS

Such a cyst may occupy as much as one-quarter of the kidney. They are also a developmental anomaly and will only rarely be seen clinically, when they are present as a cystic swelling in the loin. They will probably be mistaken for a hydronephrosis until a pyelogram has been taken. Treatment is not necessary, but if they are of large size or are giving pain they may be removed without fear of a fistula resulting.

#### PARASITIC CYSTS

Hydatid disease is seen in the kidney, where it may produce no symptoms or signs save a tumour and a dragging pain in the loin, until complications supervene. Rupture may occur into the pelvis with the most severe colic, hæmaturia and collapse. The treatment is nephrectomy.

#### GROWTHS OF THE KIDNEY

The classification of growths of the kidney is not so straightforward as in many other organs. The following is believed to be as near pathological truth as possible.

*Primary Growths of the Kidney*

	<i>Epithelial.</i>	<i>Connective Tissue.</i>	<i>Mixed.</i>
Benign . . .	Adenoma	<div style="display: inline-block; vertical-align: middle;"> <div style="font-size: 2em; vertical-align: middle;">{</div> <div style="display: inline-block; vertical-align: middle;"> Lipoma Fibroma Angioma </div> </div>	
Malignant . . .	<div style="display: inline-block; vertical-align: middle;"> <div style="font-size: 2em; vertical-align: middle;">{</div> <div style="display: inline-block; vertical-align: middle;"> Hypernephroma Carcinoma </div> </div>	Sarcoma	Teratoblastoma

*Secondary Growths of the Kidney*

Carcinoma	Sarcoma
-----------	---------

*Growths of the Renal Pelvis*

Benign . . .	Papilloma	Angioma
Malignant . . .	Carcinoma	

THE BENIGN GROWTHS are rare. The adenoma is a pathological curiosity discovered usually at post-mortem examinations. It is an encapsuled tumour beneath the renal capsule and as it is often seen in kidneys, which are affected with chronic interstitial nephritis, it may be in the nature of a regeneration nodule. The connective tissue growths are too rare and of too little significance to merit description.

## THE MALIGNANT GROWTHS

Malignant growths are not common, constituting only between 0.5 and 2 per cent. of the total cancer admissions to large hospitals. The mixed tumours are most frequently seen in children under 6 years of age and the remainder in adults between the ages of 35 and 65 years, the maximum number occurring between 45 and 50 years. Men are affected in the proportion of 3 : 2 and the two kidneys are equally attacked. No predisposing factors are known, and although calculi are sometimes coexistent there is little evidence to support them as an etiological factor.

*Pathology*—(A) **Hypernephroma** (Grawitz' Tumour).—This tumour may arise from any part of the renal cortex, but usually springs from one or other pole and but rarely from the central area. Despite the size to which it may grow, it tends to leave one pole surprisingly free. Its external surface is smooth though lobulated, its colour varies from yellow to brown or red. In consistence it is firm except over areas of necrosis. The cut surface is absolutely typical. The tumour gives the appearance of firm encapsulation in places, but elsewhere outlying nodules are present. It is golden yellow in colour with areas of greyish necrosis and dark patches of hæmorrhage scattered throughout its substance. There is an indefinite division into lobules by strands of bluish semitranslucent fibrous tissue. The renal pelvis may be distorted or invaded (Fig. 372).

Microscopically, a frozen section stained with Sudan-III. and hæmatoxylin shows a brilliant red picture, the tumour cells being laden with lipoid. In paraffin sections the cells are large, polygonal and vacuolated, with a pale scanty protoplasm and well-defined nucleus.

They are arranged in columns along the capillaries, forming an alveolar type of tumour, and in some cases there is a definite papillary grouping. The appearances are strongly reminiscent of the zona fasciculata of the adrenal gland (*cf.* Fig. 40, p. 109).

It spreads by embolism, by direct permeation along the renal vein and by lymphatics to the juxta-aortic glands. Vascular emboli lead to secondary deposits in the lungs. In addition these growths show a selective affinity for the bones, and many cases are recorded in which the bony deposit was the first sign of the disease. Infiltration of the perirenal fat may also occur.

Grawitz suggested that these tumours arose from misplaced adrenal cells, which had been included in the developing kidney. Such "rests" are known to occur and the tumour does bear a marked resemblance to the adrenal cortex. Stoerk submitted that their papillary structure precluded their adrenal origin. Wilson and Willis believed they were of Wolffian derivation. The view held to-day is that they are primary growths of the kidney cells of a specialised type.

(B) **Carcinomata** are indistinguishable from the sarcomata and the mixed tumours. They are whitish grey homogeneous growths, which spread rapidly through the kidney and grow to moderate size. They vary greatly in their microscopic appearance, some mimicking the renal tubules and forming an adenocarcinoma, while others consist of a mass of undifferentiated cells—a carcinoma simplex.

(C) **Sarcomata** are the rarest of all renal tumours. Modern pathological diagnosis tends to place most of the so-called sarcomata among the teratoid tumours. They are whitish grey growths which rapidly destroy the kidney and metastasise by the blood stream.

(D) **Teratoblastomata** (Mixed Tumours of Infants).—These growths may arise anywhere in the cortex and not necessarily exclusively from the hilum as Bland Sutton suggested. They grow rapidly to a great size and destroy the kidney. They are grey or ivory white, homogeneous in appearance and have little tendency to hæmorrhage, necrosis or cyst formation. They spread by vascular embolism and by direct growth along the renal vein (Fig. 373). Microscopically, they consist of tissue resembling a spindle-celled sarcoma, and the other

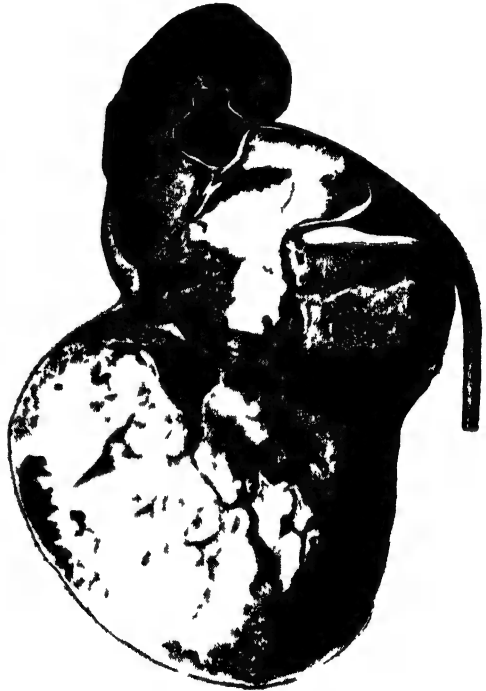


FIG 372

A Grawitz tumour. The apparent encapsulation of the growth is well seen, the upper pole appears quite normal but the renal pelvis is full of growth.

elements may not be easy to find, but careful search will always reveal both striated and non-striated muscle fibres and tall columnar epithelial cells arranged in tubules. Cartilage, bone and the derivatives of the skin are very rarely found. In the past a great many names have been applied to this tumour, but the two given here are both pathologically correct and clinically descriptive, whereas all others are misleading.

*Symptoms of all Renal Tumours* are hæmaturia, the discovery of a tumour and mild dragging pain. Hæmaturia is present in over



FIG. 373

A kidney completely replaced by a teratoblastoma. Both artery and vein are shown full of growth.

80 per cent. of cases, being most regularly found in the hypernephromata and least commonly in the teratoblastomata. It is not of great severity at first, but recurs in attacks of greater frequency and quantity. It is often the only symptom, and may appear in the form of worm-like clots, which are formed in the ureter. A palpable tumour is likely to be of late occurrence, particularly when the growth is in the upper pole. In infants it may be the only sign and the tumour may attain great size. Pain is not constant and is usually a dull ache in the loin, although referred pain may be present. A sudden exacerbation is usually due to hæmorrhage occurring in the tumour. Too much stress has been laid on the presence of a varicocele as a symptom of renal growth. It is seen in a small percentage of cases and is of no significance except on the right side. Here it may provide an additional piece of evidence, but its absence means nothing.

*Diagnosis* is made by intravenous urography, which will show

a deformity in the shadow of one or more calyces. If any doubt exists an ascending pyelography will usually settle the question (Fig. 374). The prognosis is poor; in children the mixed tumour is usually fatal; in adults carcinoma has a very high mortality rate and only in the hypernephromata is there any real hope of a lasting cure. The outlook would be improved if EVERY case of hæmaturia, however slight, was submitted to a full urological investigation at its first occurrence.

*Treatment.*—In the absence of secondary deposits, and if the opposite kidney is efficient, the affected kidney must be removed. The operation may be difficult because of adhesions and enlarged



glands After nephrectomy radon seeds should be implanted around the pedicle among the glands.

### GROWTHS OF THE RENAL PELVIS

**Papillomata** are rare. They are more frequent in men than in women, and in the right kidney. It is believed that workers in the aniline-dye industry are prone to them. They are transitional-celled tumours of the villous and pedunculated type. The border-line between the benign and malignant papillomata is ill-defined, and apparently benign examples have been known to seed out in the ureter, bladder or even in an operation wound. They give rise to symptomless hæmaturia and to hydronephrosis if the tumour blocks the ureter. Owing to their doubtful status these tumours must be treated by nephrectomy if the other kidney is efficient.

**An Angioma** is occasionally seen in a calyx at the apex of a pyramid and may be the cause of "essential renal hæmaturia."

**A Carcinoma** is either a malignant papilloma or a squamous-celled ulcer. The latter is very rare and may be due to the irritation of a calculus. It produces pain, hæmaturia and possibly hydronephrosis and its treatment is nephrectomy.

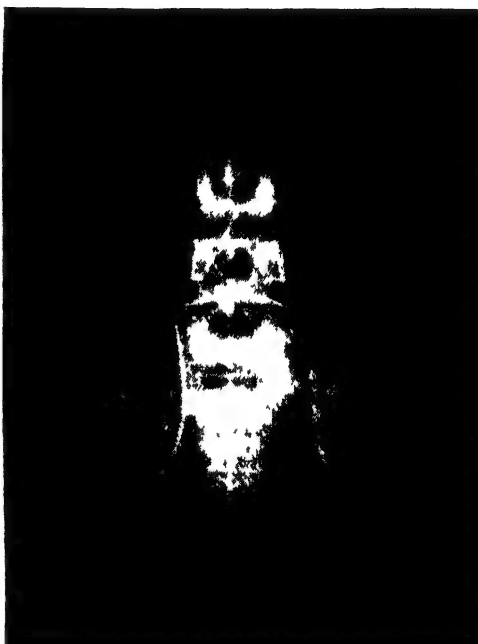


FIG. 374

A bilateral ascending pyelography showing a distorted shadow on the left side due to a Grawitz tumour.

### THE URETER

The ureter can be felt by abdominal palpation in thin patients and its termination is accessible to a finger in the rectum or vagina. Its efflux may be studied by cystoscopy and its course defined in an X-ray film by the passage of an opaque bougie. In this way it can be seen crossing the tips of the 3rd, 4th and 5th lumbar transverse processes, passing just internal to the sacro-iliac joint and the ischial spine. It then curves outwards and again inwards behind the shadow of the horizontal ramus of the pubis.

### INJURIES

**Subcutaneous Injuries** are rare and give no immediate symptoms apart from a slight hæmaturia. As the urine slowly leaks through a tumour forms, which after several days will become infected. If

injury is suspected, intravenous urography will provide the diagnosis, and if it is a complication of an open wound the external leakage of urine will quickly reveal damage to the ureter. Treatment consists in exposure and suture of the tear with drainage.

**Surgical Injuries.**—The ureter is liable to injury in many surgical and gynæcological operations, *e.g.*, during the removal of carcinoma of the rectum or uterus, ovarian cysts, cervical fibroids, etc., and it may be crushed during difficult forceps deliveries. It may be divided cleanly, lacerated or included in a ligature; or again its blood supply may be cut off and sloughing of the wall occur later with fistula formation. If recognised at the time suture over a ureteric catheter will give admirable results. One of the commonest late sequelæ is the uretero-vaginal fistula, a source of the greatest discomfort and distress to the patient. These and similar lesions should be treated by the implantation of the ureter into the bladder, or, failing this, into the rectosigmoid junction. Other fistulæ are known but are rare, and they will heal spontaneously provided that there is no obstruction to the ureter.

**CYSTS OF THE URETER** are rare. They are seen in the intramural part and cause a prolapse of the ureteric orifice into the bladder. The condition is either congenital (Fig. 375) or due to inflammatory cicatrization. The symptoms are those of hydronephrosis or renal infection and occasionally a calculus will form in the cyst.

*Treatment* aims at the removal of any cause and slitting the orifice so as to leave a wide opening.

### URETERIC CALCULI

A primary ureteric stone forming around a ligature or other foreign body is extremely rare, the vast majority reaching the ureter from the renal pelvis. It might be imagined that the fact that a stone has entered the ureter would be a guarantee of its passing through to the bladder without difficulty, but the ureter narrows at the pelvic brim and at its entrance to the bladder, and at either of these points a stone may be arrested (Figs. 352 and 375).

*Symptoms* are pain and some changes in the urine. The pain varies in type and severity. The contractions of the ureter give colic, which is maximal at a point over the position of the stone. There may be pain in the kidney area and referred pain in typical zones. Some patients do not complain of severe colic, but have persistent aching pain accompanied by a feeling of great tiredness. When the stone reaches the intramural portion of the ureter frequency of micturition, strangury and penile irritation will be added to the picture. The changes in the urine are those already described in the section on Renal Calculi (p. 740).

The sequence of events in the passage of a ureteric stone depends upon its fate: either it passes through to the bladder at the first attempt, in which case the attack of colic terminates abruptly as soon as the stone falls free into the bladder, or it is held up temporarily or permanently with intermittent attacks of colic. The intervals

vary between hours and many weeks, and in the latter patients there is a danger of hydronephrosis.

*Diagnosis* is made by X-rays, which will not only demonstrate the stone but will permit of its exact localisation by orientation with the transverse processes of the lumbar vertebræ and the various bony points of the pelvis and sacrum. In this way the progress made after each attack can be verified. Intravenous urography will demonstrate the early signs of hydronephrosis. Cystoscopy will show pouting of the orifice and possibly also bullous œdema when the stone is in the intramural ureter.

The differential diagnosis rests between renal and other forms of colic, *i.e.*, biliary and intestinal. The type and distribution of the pain, the urinary symptoms and the X-ray pictures will solve any difficulty.

*Treatment.*—The patient will assuredly be seen during an attack of colic. This is controlled by a hypodermic injection of morphia gr.  $\frac{1}{4}$  and atropine gr.  $\frac{1}{100}$ , the patient being put to bed and kept warm with hot-water bottles. If the stone is small and is going to pass in one attack, this treatment will suffice and it will be voided within a few hours.

**Stones not Passing at the First Attempt.**—The general principles are that (1) a stone making definite progress after each attack may be left to pass, provided it is not taking too long, and (2) every stone

making no advance must be removed as soon as possible. A patient with a stone impacted in the ureter is in danger not only of a slow deterioration of kidney function from back pressure, but also of a sudden calculous anuria. The management of these patients is clear; after the first attack the stone is localised by radiography and after the second attack its progress is noted. If the stone has not appeared after two months an intravenous urograph is taken, and if there is the least evidence of early hydronephrosis, operation must be considered. Many of the stones, however, can be induced to pass by an injection of one of the Acetylcholine group of drugs, *e.g.*, trasentin.

*Palliative Treatment.*—During the attack the morphia and atropine injection is given and six hours later the following medicine administered by mouth and repeated every four hours until the pain has ceased :—

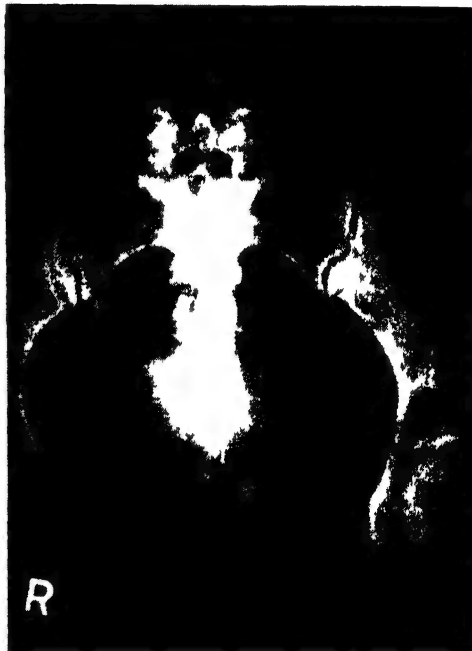


FIG. 375

An intravenous urography showing on the right side a congenital ureterocoele. On the left side a stone may be seen in a similar cystic dilatation of the lower end of the ureter.

Tinct. opii . . . . .	℥xv
Tinct. bellad. . . . .	℥vii
Tinct. hyosc. . . . .	℥ss
Syrupus aurantii . . . . .	℥ss
Aquam . . . . .	ad ℥i

During the intervals patients should be placed on a carefully regulated medical régime, and at least 6 pints of bland fluids must be taken daily.

*Operative Measures.*—Cabot reports that of the stones which become impacted 15 per cent. do so in the upper part of the ureter and 75 per cent. in the pelvic portion. The high ones are removed by the same approach as renal stones, while those in the pelvis can be reached through either a midline or a lateral muscle-splitting incision with extraperitoneal dissection by the side of the bladder. Those in the intramural ureter can sometimes be coaxed down by various means, such as passing two or three catheters up past the stone and injecting sterile paraffin, when the stone may become engaged in the catheters and withdrawn with them. Stones firmly impacted in the orifice should be removed by suprapubic cystotomy, though the expert cystoscopist may feel justified in dividing the rim of the opening with a diathermy electrode.

### CALCULOUS ANURIA

This condition, the possibility of which adds a heavy burden of anxiety to those in charge of a patient with a ureteric calculus, consists in a sudden failure of secretion of urine. It may be due to the following combination of circumstances :—

1. Simultaneous blocking of both ureters by stones.
2. The blocking of one ureter, the other kidney being absent either congenitally, after operation or having been destroyed by disease.
3. The blocking of one ureter, the other kidney being diseased either grossly or by the early stages of chronic interstitial nephritis.

*Symptoms* are pain and the cessation of the secretion of urine. There may have been previous attacks of pain, or the anuria may be ushered in with pain, suggestive of a ureteric stone. But it is often slight, and indeed examples are on record when there has been no pain at all. The disease falls into two stages :—

(A) **Stage of Tolerance.**—From the start of the anuria there is a period lasting from six to twelve days, in which the patient feels perfectly well. No urine, or at most 1 or 2 oz. in twenty-four hours, is passed. The absence of symptoms may mislead both the patient and the doctor, neither of whom may appreciate the gravity of the condition.

(B) **Stage of Toxæmia.**—The well-being of the stage of tolerance ends abruptly and drowsiness, headache and delirium appear. Nerve reflexes are absent or diminished, movements of the limbs are sluggish, the pulse and respiration are slow and irregular and finally Cheyne-Stokes breathing occurs. Edema is usually absent, but abdominal

distension, vomiting, constipation and hiccough are common. Death occurs within four days of the onset of this stage.

*Diagnosis.*—There should be no doubt as to the diagnosis, but the difficulty lies in the localisation of the stone. Certain facts must be appreciated.

1. No matter how well the patient may appear, anuria calls for urgent investigation.

2. The period of tolerance gives ample time for investigation if patients present themselves early enough, and the number of days of anuria give an idea of the margin of safety left. Under no circumstances whatever should treatment be left until the onset of toxic signs.

3. An X-ray will usually, but not always, define the position of the stone, and in skilled hands the wax-tipped ureteric bougie will give more sure information.

4. Certain patients will present grave difficulties. The pain may be non-existent or bilateral, it may not be possible to localise the stone by any means or the patient may be seen for the first time at the beginning of the stage of toxæmia. The procedure in these cases is fortunately clearly defined.

*Treatment.*—The obstruction must be removed without an hour's delay. The stone, or stones, should be removed from one or both ureters. In those difficult cases where localisation has failed, both kidneys must be drained by lumbar nephrostomy. This allows free drainage and the re-establishment of the secretion of urine, and after the patient has come safely out of danger, further steps can be taken to define and remove the stone.

R. M. HANDFIELD-JONES.

## CHAPTER XXXVI

### THE BLADDER, PROSTATE AND VESICLES

**A***NATOMY*.—The bladder when empty or moderately distended is entirely a pelvic organ, but as the result of obstruction it may reach as high as, or higher than, the umbilicus. Its normal capacity is between 10 and 15 oz. The superior surface is concave when empty, convex when distended, and is covered by peritoneum, which separates it from the small intestine and the sigmoid colon. As it distends it strips the peritoneum off the anterior abdominal wall, and so becomes a contact relation with the abdominal muscles. In front a space filled with fat and areolar tissue—the space of Retzius—separates it from the symphysis pubis. Its apex, directed upwards and forwards is attached by a fibrous cord, the urachus, to the umbilicus. Behind in the male, the seminal vesicles and the vasa deferentia lie between it and the rectum, and below it rests on the prostate and the puboprostatic ligaments. In the female, the anterior wall of the vagina and the uterus lie behind and below it. The neck of the bladder, where it becomes continuous with the urethra, is 2 in. behind the mid-point of the symphysis pubis. The trigone is a triangular area bounded by the interureteric bar above and lines joining the ureteric openings to the urethral orifice. It is a distinct part of the bladder, its muscles being derived from that of the ureters.

The bladder is supplied with blood by the superior and inferior vesical branches of the internal iliac artery, and its venous drainage is into the internal iliac vein. The lymphatics pass to the glands on the external iliac artery and at the bifurcation of the aorta. The nerve supply is from the 3rd, 4th and 5th sacral, together with sympathetic twigs from the hypogastric and hæmorrhoidal plexuses.

*Examination*.—An over-distended bladder forms a visibly prominent swelling, having a central position in the lower abdomen. It rises out of the pelvis as a spherical smooth tumour, which is dull on percussion. The base can be palpated digitally from the rectum or vagina. Cystoscopy allows a complete visual examination, and after filling the bladder with sodium bromide, an X-ray picture will define its limits. This is known as cystography.

### CONGENITAL ANOMALIES

#### PATENT URACHUS

This is a rare condition, and the degree of patency varies, thus :—

1. Complete, *i.e.*, a vesico-umbilical fistula.
2. Incomplete . . . 

{	Vesico-urachal sinus.
{	Umbilico-urachal sinus.
{	Intermediate, <i>viz.</i> , a urachal cyst.

The complete fistula is most commonly noticed because of the discomfort from leakage of urine at the umbilicus, but it is probable that a large number of bladders have some degree of symptomless vesico-urachal sinus either as a minute channel or a wide-mouthed diverticulum. The complete fistula is made obvious either at birth or in infancy by urinary leakage, or may be seen in later life in men when prostatic or urethral obstruction forces open the persistent passage and urine appears at the umbilicus, the patient not having previously been aware of the defect. The umbilico-urachal sinus varies from a small tumour at the umbilicus to a minute sinus, which gets blocked and so gives rise to recurrent attacks of pain, tenderness and swelling. The urachal cysts result from closure of the duct above and below with persistence in the middle. They form elongated rounded swellings in the midline between the umbilicus and pubis.

*Treatment* consists in removing any cause of obstruction, *e.g.*, phimosis in infants or enlarged prostate in adults, when, if the fistula persists, the whole urachus should be removed. Urachal remnants at the umbilicus should be removed, owing to the possibility of recurrent infections, while urachal cysts need removal for fear of malignant change. No operation should be performed in infants until they reach the age of nine months or a year.

ABSENCE OF THE BLADDER is a very rare condition, and is accompanied by widespread abnormalities of the external genital organs. The ureters open into some unusual structure, *e.g.*, vagina, bowel or skin.

#### ECTOPIA VESICÆ

This is happily also a rare anomaly, for it is difficult to picture anything so distressing. It consists in a failure of development of the bladder, save for a small basal area which includes the ureteric openings, and also in failure of growth of the skin of the anterior abdominal wall in the midline below the umbilicus. There is a small area of posterior bladder wall therefore, whose edges are firmly adherent to the margins of the skin defect. The recti muscles are present but widely separated, and intra-abdominal pressure forces the bladder forward until it is flush with the surface. The urine trickles away and spouts out on coughing or straining. Associated with the bladder defect is a rudimentary penis in a state of epispadias (p. 780) the urethra being represented by a groove. The testes are retained in the abdomen, the pubic bones do not meet in the midline, and the pelvis is generally malformed, leading to a waddling gait. Although there is no developmental defect in the brain, untreated cases show a poor mentality as they grow up and are backward in every way.

*Treatment.*—Attempts to reconstruct the bladder have always failed, and the only successful procedure is the transplantation of the ureters into the rectum or pelvic colon. Stiles' operation is done in two stages, the ureters being transplanted at different times, six weeks being allowed to elapse between the two operations, at the second of which the small amount of bladder wall is completely removed and the defect in the abdominal wall repaired.

### DIVERTICULUM OF THE BLADDER

A diverticulum is a sac lined with vesical mucous membrane protruding from the bladder into the surrounding fat, and opening into it by a narrow orifice. It is more frequently recognised since diagnostic methods have improved and is not a rare condition.

*Etiology.*—Diverticula may be single or multiple and vary in size from a peanut to a cavity larger than the normal bladder. They are found on the lateral and posterior walls near the ureteric orifices or at the apex. They are very rare in women, but in males may be seen either in early childhood or in the later years after middle life, when they follow those conditions causing urinary obstruction. The question of their origin remains hotly debated, some writers insisting that all are congenital and others that all are acquired. Similarly, the presence or absence of muscle in their walls is disputed. There seems little doubt that they may be either congenital or acquired, and many of them have muscle fibres irregularly arranged in their walls.

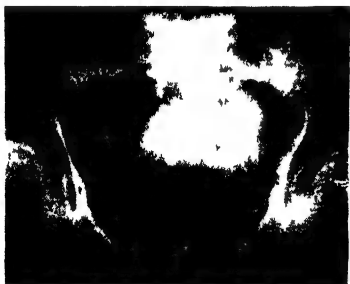


FIG. 376

Multiple diverticula of bladder revealed by cystography.

*Symptoms.*—They are symptomless until infection occurs. Stagnation of urine in them makes them prone to this, and stone formation is likely; but even so, the symptoms are vague and varied. There may be unexplained attacks of frequency or of dysuria. Some patients complain that, after a normal emptying of the bladder of clear urine, they pass a copious amount of thick cloudy urine a few

minutes later. Some can produce the second emptying by pressure from above, while others describe a feeling of the tumour moving downwards when the bladder is emptying. It is safe to adopt the attitude in all atypical bladder cases that a diverticulum is a likely explanation.

*Diagnosis.*—Cystoscopy and cystography will show the number, size and position of the diverticula, but the photographs must be taken in several planes otherwise some shadows will be masked by that of the bladder itself.

*Treatment.*—Owing to the impossibility of dealing adequately with the infection in a diverticulum and because of the danger of malignant change occurring in it, its removal is advisable. Appropriate treatment must be undertaken for the cause of any obstruction which may be present. The diverticulum may be removed either from within the bladder or from without. The ideal method combines the two avenues of approach. The bladder is opened suprapubically and the diverticulum packed with ribbon gauze to make it stand out firmly for subsequent recognition. Extravesical dissection then readily isolates the diverticulum which is removed by division of its neck, the defect in the bladder being closed by sutures in layers. Drainage is maintained by an indwelling catheter for five days.



## ANOMALIES OF FUNCTION

The anomalies of function may be classified thus :—

(Modified from Thomson Walker.)

- |  |  |  |
|--|--|--|
| A. Incontinence                        | $\cdot \left\{ \begin{array}{l} \text{False} \\ \text{True} \end{array} \right\} \left\{ \begin{array}{l} \text{Passive} \\ \text{Active} \end{array} \right\}$  | $\text{due to} \left\{ \begin{array}{l} \text{Mechanical causes.} \\ \text{Nervous disease.} \\ \text{Bladder spasm.} \\ \text{Nocturnal enuresis.} \end{array} \right.$ |
| B. Acute retention                     | $\left\{ \begin{array}{l} \text{With obstruction.} \\ \text{In spinal cord lesions.} \\ \text{In acute and chronic intoxications.} \\ \text{From inhibition or spasm.} \\ \text{From achalasia of sphincter.} \end{array} \right.$ |  |
| C. Chronic retention from obstruction. |  |  |

## INCONTINENCE OF URINE

**False Incontinence** or distension with overflow is a condition in which the bladder is distended as a result of mechanical obstruction or of disease of the spinal cord, when, after the limit of its capacity is reached, the urine simply dribbles away.

**True Incontinence** implies an empty bladder. In the *passive* type, the internal sphincter is paralysed, and urine flows straight from the ureters to the urethra. In *active* incontinence the sphincter is functioning, but so inefficiently that the bladder contractions overcome it and the urine is expelled into the urethra.

**Mechanical Incontinence** is seen in women particularly after childbirth, and in its mild forms is present only on straining, coughing or sneezing. Uterine prolapse with a cystocele is a common cause.

**Nervous Disease** may cause either distension with overflow or true incontinence, and symptomless anomalies of bladder function should always lead to an examination of the central nervous system and of the spine to exclude spina bifida occulta.

**Bladder Spasm** may be so severe in acute and chronic cystitis and tuberculous disease that incontinence results.

**Nocturnal Enuresis** of children may be simply a delay in the establishment of voluntary control over the act of micturition, which in the first twelve months is automatic, and some children learn control later than others. In some cases children between the ages of 4 and 8 years, who have had perfect control, develop the habit of wetting their beds. Some coexisting disease can usually be found to account for this bladder irritability, *e.g.*, intestinal worms, vulvo-vaginitis, phimosis, cystitis or oxaluria; in the remainder the child will be highly excitable and nervous. Control can almost always be taught, and only in exceptional cases does the condition survive puberty. The treatment is the removal of the cause. Late hours and mental excitement must be avoided, and no fluids given after tea-time, those which tend to act as diuretics being excluded entirely. The child must empty the bladder immediately before getting into bed, and is

awakened after two hours sleep to empty it again. Tincture of belladonna in suitable doses may be needed, but is a double-edged weapon owing to its thirst-producing qualities. During the day the child is encouraged to hold the water over increasing periods, so teaching the bladder to become accustomed to distension. Above all, patience and kindness will work wonders, whereas scolding and punishment will aggravate the trouble. Resistant cases sometimes respond to full dilatation of the urethra by bougies; those which resist all other forms of treatment may be completely relieved by Millin's corpus spongiosum plication technique.

### RETENTION OF URINE

#### A. Acute.

##### 1. Retention with obstruction is due to lesions in :

- A. The Penis—phimosis, paraphimosis, encircling rings or string and growths.
- B. The Urethra—stricture, rupture, stone, acute urethritis and pressure from without.
- C. The Prostate—benign enlargement, carcinoma and acute prostatitis with or without abscess.
- D. The Neck of the Bladder—growth or an impacted stone.

*Diagnosis.*—It is essential to distinguish at once between retention and anuria, and then to ascertain the cause of the retention. In anuria the bladder is empty or contains a small quantity of very concentrated urine; in retention the tense dull suprapubic swelling is characteristic.

*Treatment*—1. Retention due to Obstruction.—The patient is placed in a hot bath after a suppository of morphia and belladonna has been introduced into the rectum. If this fails a catheter must be passed, and a note of warning is necessary. It is dangerous to empty the bladder completely at the first catheterisation, as death from renal hæmorrhage or anuria may occur. Two methods of gradual emptying are in use. In the first a catheter is tied into the bladder and a piece of rubber tubing is attached to it leading to a collecting bottle. An adjustable tap is included in the circuit and the flow of urine so adjusted that the bladder is completely emptied within seventy-two hours. A second method entails the passage of a catheter every six hours. On the first occasion it is removed when the bladder can no longer be palpated above the pubis, and at the succeeding catheterisations more and more urine is evacuated till the bladder is empty in about seventy-two hours. Many patients will regain the power of voluntary micturition after one catheterisation.

2. Retention due to Spasm or Inhibition occurs in hysteria and after recent and vaginal operations and is treated by the application of large hot fomentations over the lower abdomen and the hypodermic injection of "Doryl" (in 0.1 grm. doses). This drug is so powerful in its action that catheterisation is rarely necessary.

3. Retention due to Spinal Cord Lesions.—Those diseases and injuries which produce retention of urine raise a grave problem—that

of the "cord bladder." No matter how carefully catheterisation is performed, urinary infection is not only inevitable but usually fulminating. There are two methods of treatment, viz., suprapubic cystostomy and tidal drainage. Whichever is chosen it must be adopted at once and not after one or two days' intermittent catheterisation. The former controls infection and prevents many deaths from renal failure, the latter has two great advantages. By rhythmically filling and emptying the bladder its muscles are kept in training for the day when automatic

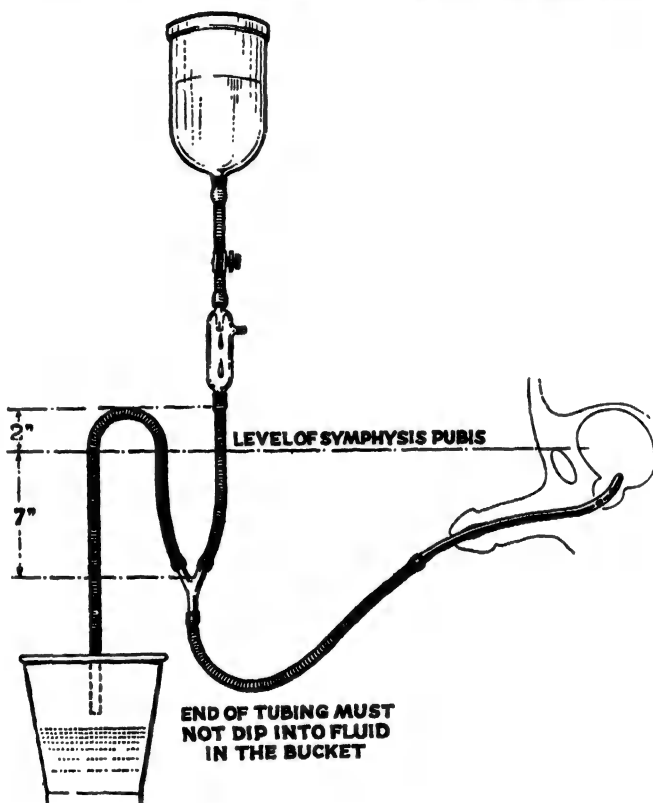


FIG. 377

Tidal drainage apparatus. (*Surgery of Modern Warfare.*)

bladder control is taken over by the spinal centres. Further the fluid used for irrigation is a mild antiseptic which assists in controlling infection. The apparatus is shown in Fig. 377.

4. Retention due to Achalasia of the Sphincter.—Retention, either complete or with a bladder up to the umbilicus and a regular passage of about 4 oz. of urine at frequent intervals, is very occasionally due to imbalance of the two sides of the sympathetic system. Although the detrusor muscle is able and willing to empty the bladder, the sphincter refuses to give way. Treatment is a pre-sacral neurectomy.

**B. Chronic** retention is a condition in which there is a long-standing partial obstruction without actual stoppage. It is seen in all cases of enlarged prostate and urethral stricture. The pathological results are of the utmost importance. In the order of their

occurrence they are dilatation of the bladder, hypertrophy of its muscle with trabeculation and sacculation, dilatation of the ureters and the renal pelvis, producing hydronephrosis. Sooner or later infection supervenes, giving rise to cystitis, ureteritis, pyelitis and pyonephrosis. The lesson to be learned is that benign enlargements of the prostate and urethral strictures are intrinsically of little importance, but take on the gravest significance because of their far-reaching and destructive effects on renal function.

### INJURIES OF THE BLADDER

**Contusions.**—The mucous membrane may be bruised without an actual tear in the bladder wall. The result depends on the severity of the bleeding and the presence of infection. Slight cases present a transient hæmaturia which clears up rapidly, while in others clot retention has been recorded, and in the presence of an infected urine, severe sepsis may result.

**Rupture.**—This is more common in men (9 : 1), and between the ages of 20 and 40 years. It occurs when the bladder is full and often during alcoholic intoxication. The cause is a kick, a blow, or some crushing accident, and a previously diseased bladder will always be more vulnerable. Spontaneous rupture is seen in carcinoma of the bladder or ulcerative cystitis. Fractures of the pelvis may lead to penetration of the bladder wall by fragments of bone. The tear may be on the postero-superior surface when the peritoneal cavity will be opened, or on the lateral and anterior aspects when the extravasation is extraperitoneal.

**Symptoms—Intraperitoneal.**—There are two stages in the clinical picture, the first or pre-peritonitis stage lasting from twelve to twenty hours, and that of peritonitis which becomes gradually established later. Owing to shock and intoxication a reliable account of the accident or of the patient's condition is difficult to obtain.

**First Stage**—The injury is followed by severe shock and hypogastric pain, with an intense desire to pass urine, which results in only a few blood-stained drops. Although no urine has been passed for hours, no bladder dullness can be elicited, but shifting dullness in the flank may be present. If a catheter is passed, a few drops of blood-stained urine will come away, but if by gentle manipulation the catheter passes through the tear into the peritoneal cavity, a large quantity of urine is obtained, and this may be increased by having the patient propped up in bed.

**Second Stage.**—Sterile urine produces an aseptic toxic peritonitis of gradual onset characterised by abdominal pain and tenderness, with signs of commencing paralytic ileus, viz., distension, absence of bowel action, and vomiting. If the urine is infected the resulting septic peritonitis is more rapid in onset and more severe in both local and general manifestations.

**Extraperitoneal.**—The introductory story is similar to the above, but urine extravasates into the pelvic cellular tissues and slowly a diffuse swelling appears above one or both inguinal ligaments. A grave and widespread pelvic cellulitis results and early drainage is imperative.

**Diagnosis.**—The picture may be so typical that the diagnosis is easy, but many cases cause anxious doubt. A catheter may be passed once or twice, but its use is to be deprecated owing to the grave risk of introducing sepsis. The practice of introducing a known quantity of sterile fluid to see if all or only a portion is returned is permissible only if it is regarded as a preliminary to immediate operation. So disastrous is it to overlook a ruptured bladder that it is wise to adopt the policy of exploring the bladder suprapubically in every case of doubt.

**Treatment** is immediate operation. In the intraperitoneal rupture, the peritoneal cavity is opened by a midline subumbilical incision, and a thorough toilet carried out. The rent is then identified and closed by sutures, and the bladder drained by an indwelling catheter. If the rent is inaccessible from the peritoneal cavity, the bladder is opened, the rent sutured from within, and both peritoneum and bladder drained.

In extraperitoneal cases a suprapubic cystotomy is performed, and the tear sutured. The bladder is drained, and tubes inserted wherever urine has extravasated and collected.

**Wounds** in civilian practice are very rare, but comparatively common in war, and the latter are frequently complicated by wounds of neighbouring structures. Injury may occur during surgical operations, *e.g.*, on a hernia, the vagina or uterus. The treatment consists in suturing the tear in the bladder and drainage.

### CYSTITIS

Inflammation of the bladder as a primary condition is rare, and is usually an expression of, or a complication of, infection in other parts of the genito-urinary system in both sexes.

**Cystitis** may be classified as follows :—

- |                  |   |   |   |  |
|------------------|---|---|---|--|
| 1. Non-bacterial | . | . | . | { Mechanical.<br>{ Chemical.<br>{ Acute.<br>{ Chronic.<br>{ Tuberculous. |
| 2. Bacterial     | . | . | . |  |
| 3. Parasitic.    |   |   |   |  |

**NON-BACTERIAL CYSTITIS** is recorded as an acute cystitis due to irritation from strong chemicals, taken either by the mouth or instilled per urethram; and to the mechanical effects of foreign bodies, stones or uterine displacements. The condition is transient, and no treatment is needed save the removal of the cause.

**BACTERIAL CYSTITIS.**—The bacteria flourishing in an acid urine are *B. coli*, gonococcus and *B. tuberculosis*, and those in an alkaline medium are staphylococcus, streptococcus, *B. proteus*, and *B. typhosus*, while mixed infections are relatively common. When ammoniacal decomposition occurs, as it will in an alkaline urine, an additional chemical factor is at work. The normal bladder resists infection, even in the presence of a heavily infected urine from the kidneys.

**Predisposing Factors.**—1. Trauma acts by devitalising the mucous

membrane. This may result from rough urethral instrumentation, fractures of the pelvis, complicated obstetric deliveries, and operations for carcinoma of the rectum, uterine fibroids or prolapse.

2. Irritation by stones, foreign bodies or growths.

3. Chronic retention is the commonest cause, as seen in cases of enlarged prostate, urethral stricture, diverticulum of the bladder, retroverted gravid uterus and cystocele.

4. Neuropathic atony and trophic changes. Certain spinal cord lesions cause imperfect emptying combined with a trophic malnutrition of the bladder wall. In such cases no precautions, however exact, can save the patient from cystitis if the use of a catheter becomes necessary.

5. Congestion of the bladder by cold, excretion of chemical irritants, alcohol, sexual excess, and the spread of a neighbouring inflammatory process, *e.g.*, diverticulitis of the colon.

**Paths of Infection.**—*A.* Descending from the kidney. This is more important than was previously thought, and in stubborn cases ureteric catheter specimens should be examined.

*B.* Ascending from the urethra. The posterior urethra may fairly be regarded as the central point of the genito-urinary tract; from it there is ready access to the prostate, seminal vesicles, vasa deferentia, anterior urethra, bladder and ureteric orifices. Infection may spread from it to the trigone of the bladder, either spontaneously or carried by bougies and catheters. In women the short urethra offers no barrier to the passage of infection from the vulva and vagina.

*C.* Contiguity. Infections of neighbouring organs may spread to the bladder, *e.g.*, appendix abscess, diverticulitis of the colon or salpingitis.

#### ACUTE CYSTITIS

The mucous membrane and submucosa alone are affected, the changes being either generalised or confined to the trigone. The membrane is red, oedematous and plicated. Fibrin may be deposited on the surface, and necrosis occur with localised sloughing. Rare cases are reported in which complete casts of the bladder have been shed. The muscle is not infected, but may become hypertrophied from over-activity.

**Cystoscopic Appearances.**—The essential change is the replacement of the normal delicate branching vascular network by a diffuse bright red capillary congestion. Later, oedema causes the mucous membrane to be thrown into ridges, on which are small erosions with attached flakes of fibrin. Small greyish ulcers in an angry red background appear, and vesicles develop—the so-called bullous oedema.

In encrusted cystitis the slough on an ulcer becomes coated with phosphates, and a dead white plaque is seen which can be detached only with difficulty.

**Symptoms.**—These are: (1) frequency, (2) urgency, (3) strangury, (4) pain, (5) abnormal constituents in the urine and (6) rapid disturbance of the general condition.

**Frequency** is the earliest symptom and is more marked in the

daytime. It varies in intensity and may become so severe that micturition occurs every fifteen minutes. The **urgency** may be such that involuntary micturition occurs unless relief is ready at hand, and in very bad cases, the urgent desire to micturate remains unappeased even immediately after the bladder has been emptied, *i.e.*, **strangury**. Pain varies in degree, being exceptionally severe in bad cases. It is constant, felt as a burning sensation deep in the pelvis, and referred to the perineum and tip of the penis. It is increased by movement, in the upright position and at the end of micturition.

**Changes in the Urine** include the presence of pus, blood, bacteria, fibrin and flakes of necrosed mucous membrane. In alkaline cystitis a very foul-smelling urine may result. In rare cases bleeding may be severe, and clot retention has occurred in the author's practice.

The **general condition** undergoes an alarming deterioration in cases of even moderate severity, the key to which is distress and loss of sleep due to pain and frequency. The temperature and toxicity are not unusual, but added to the exhaustion is a mental distress which makes bad cases quite pitiful.

*Diagnosis and Prognosis.*—There can be no doubt as to the existence of acute cystitis, but it is essential to discover its cause, the presence of any predisposing factor and its complications. In the acute stage no cystoscopy is permissible, but a complete urinary investigation must be done as soon as the acute symptoms have subsided. Uncomplicated cases will clear up in six weeks, those with coexisting disease will not improve till the causative factor is removed, while cystitis in a paralysed bladder is wellnigh incurable.

*Treatment—A. Acute Stage.*—Patients must be nursed in bed, placed on a milk diet and given very large quantities of bland fluids as in the treatment of the acute febrile stage of pyelitis. Massive linseed poultices over the lower abdomen will to some extent relieve the pain, frequency and strangury, or patients may be lifted into really hot hip baths. Sleep is essential, and morphia and atropine will be needed at first. In severe cases, where sleep is impossible, and the general condition is deteriorating rapidly, a suprapubic cystotomy should be performed. During the acute stage, no attempt at cystoscopy or lavage is justifiable. Many patients will improve rapidly on sulphanilamide.

*B. Post-febrile Stage.*—Investigations are now directed towards the causative or predisposing factor, which must receive appropriate treatment. The cystitis and bacilluria should be treated with sulphanilamide, which will effect a permanent cure in most cases. Intractable cases will require bladder lavage with silver nitrate (1 : 6,000) or mercury oxycyanide (1 : 5,000). Patients must remain in bed for ten days after the temperature has become normal.

### CHRONIC CYSTITIS

The changes in chronic cystitis are very varied, but essentially the inflammation spreads into the muscle coats with the result that the bladder is no longer capable of normal distension, and in advanced



cases can hold only 1 or 2 oz. of urine. The mucous membrane shows a variety of appearances. *Granular cystitis* denotes a thickened and plicated mucous membrane, *vegetative cystitis* a more advanced condition, and *cystitis cystica* the proliferation of the epithelium into small cyst-like areas. *Pseudomembranous cystitis* is a variety in which sloughs are formed and slowly shed, and *gangrenous cystitis* a more advanced stage, with widespread necrosis of the bladder wall. In the early stages the muscle is hypertrophied but still capable of re-education to normal extensibility; later, fibrosis occurs, making diminished capacity permanent.

*Symptoms* are those of acute cystitis in a mild degree, frequency being pronounced and periodic exacerbations common. In the slight cases they may be hardly noticeable. The urine will contain pus or mucus, epithelial debris and organisms.

*Treatment*.—Any coexisting disease must be treated, as it is otherwise useless to attempt to cure the cystitis. Bladder washouts are of great service and continuous irrigation through a two-way catheter the most efficient method. The bladder must be gradually distended to normal capacity by daily clamping of the catheter. The urine will become sterile with sulphanilamide. Cystitis due to *B. proteus* will not yield to chemotherapy, but responds to silver nitrate irrigations.

#### TUBERCULOUS CYSTITIS

Tuberculosis of the bladder is invariably secondary to infection in other parts of the genito-urinary system. Primary cases are reported, but the evidence is not conclusive. It is more common in men between 20 and 40 years of age.

*Method of Infection*.—When secondary to a renal infection, the disease spreads into the bladder wall from the intramural ureter, and so reaches the bladder just above and external to its orifice. Tuberculosis of the male genital tract reaches the bladder either from the prostatic urethra or from the seminal vesicles.

*Morbid Anatomy and Cystoscopic Appearance* have been described under renal tuberculosis (p. 735). In protracted cases the bladder wall becomes hypertrophied and contracted, and in the presence of a mixed infection undergoes the changes typical of chronic cystitis.

*Symptoms* are those of chronic cystitis, viz., frequency and pain, and the urine contains pus, blood and tubercle bacilli.

*Treatment*.—If possible the primary cause must be eliminated, and it is well recognised that the vesical lesions respond favourably to treatment after nephrectomy and to a lesser extent after orchidectomy. General constitutional treatment in a suitable climate with injections of tuberculin or sanocrysin will hasten progress. Urinary antiseptics are of no value except in mixed infections, and local treatment to the bladder is to be avoided.

Bilharzial cystitis has been described in Chap. IV, p. 52.

#### FISTULA

**Suprapubic Vesical Fistula**.—After a suprapubic drainage of the bladder a fistula may persist, usually in the lower part of the



wound. Failure of closure may be due to an unrelieved urethral obstruction, severe sepsis, tuberculous or carcinomatous invasion of the track, adherence of the bladder to the wound edge, prolapse of an atonic bladder wall, or rarely to trophic changes connected with disease of the central nervous system. The fistula may partially heal and then recur, and it may transmit all the urine or only part of it.

The *treatment* is the removal of the cause, except in those cases in which a permanent fistula has been made as a method of treatment for inoperable urethral obstruction.

**Vesico-intestinal Fistula.**—This may be traumatic or pathological. The latter is due either to inflammation or new growth of the bladder or neighbouring parts of the intestine. Examples of inflammatory fistula are those due to chronic cystitis with pericystitis, appendicitis with abscess formation, pelvic peritonitis, tuberculosis of the cæcum or diverticulitis of the colon; whilst the malignant fistulæ are due to growths of the bladder, colon or cæcum.

The symptoms are those of a slowly progressive chronic cystitis which, when the fistula is complete, become more marked, bubbles of gas being passed with the urine.

*Treatment.*—Some inflammatory fistulæ heal spontaneously, but in others a colostomy must be performed to remove as far as possible the source of infection and radical excision of the diseased areas must be considered. (*Vide* Diverticulitis of the Colon.)

**Vesico-vaginal Fistula.**—This type rarely exists except as a result of surgical interference or of the injuries of childbirth. The fistula opens high up on the anterior wall of the vagina. The symptoms are distressing both mentally and from the excoriation of the skin of the vulva and thighs. The fistula must be closed, after careful preliminary preparation to clear up the infection of vagina and bladder. The bladder and vagina are separated by a combined suprapubic and vaginal approach, and each is carefully repaired. An indwelling catheter is left *in situ* for ten days.

### VESICAL CALCULI

*Etiology.*—Our present knowledge of the etiology of urinary calculi has already been dealt with (p. 736). It remains to add certain points concerning the bladder. Generally, conditions are more favourable to alkaline decomposition, so that phosphatic stones are apt to predominate, especially during the later years of life in men, when obstruction is common. In children vesical calculi are more frequently seen than renal. The number may vary from one to as many as four hundred, and when multiple they are faceted. A single stone is spherical unless forming in a diverticulum, when it will be dumb-bell in shape. Spontaneous fracture occurs occasionally.

*Symptoms.*—**A. Silent Stones.**—Stones may form without symptoms, and will always do so if they are prevented from coming into contact with the trigone. Such conditions are present when a stone grows in a diverticulum or in a retroprostatic pouch.

**B. Stones Irritating the Trigone.**—There is no characteristic picture produced exclusively by a vesical calculus. Any pathological condition

causing irritation of the base of the bladder gives a typical syndrome. The symptoms are :—

- (a) Pain is absent in a full bladder with the patient at rest, but is felt on movement and particularly on emptying the bladder. At the end of micturition the contracting bladder presses the stone against the trigone and the pain is greatly increased. Referred pain will be felt along the urethra, at the tip of the penis, rarely in the testes, and occasionally in the back, foot or heel. Children may be brought for advice as to a prolapsed rectum, the result of straining caused by bladder irritability.
- (b) Sudden stoppage of flow during micturition used to be regarded as one of the cardinal symptoms, but is not always present, and may be found in other conditions.
- (c) Frequency is usually a symptom.
- (d) Abnormal constituents vary from a few red blood cells with an occasional shed epithelial cell to a foul stinking urine loaded with pus.
- (e) Priapism, more common in the young, which may lead to masturbation.



FIG. 378

X-ray photograph of stone in the bladder later successfully treated by litholapaxy.

*Diagnosis.*—This is certain as stones can so readily be seen through a cystoscope; but it is necessary to ensure that no coexisting lesion is overlooked. For this reason, though an X-ray photograph will show the stone (Fig. 378), a cystoscopic examination is essential before treatment can be planned.

*Treatment.*—The solution of a vesical calculus by drugs administered orally remains entirely mythical. Coexisting lesions may require medical treatment but the stone itself must be removed, either by litholapaxy or by suprapubic lithotomy.

**Litholapaxy** is the operation of crushing the stone into small fragments by an instrument named a lithotrite, after which the pieces are removed by a Bigelow's evacuator. In this way no incision is made, and the patient needs only twenty-four to forty-eight hours to recover from the anæsthetic, and can return to full activity at once. There are certain definite contraindications to crushing, viz. :—

1. Too small a urethra ;
2. A severe cystitis ;

3. The presence of coexisting conditions, the treatment of which demands a suprapubic approach ;
4. Stones in unduly large numbers ;
5. Stones larger than 2 in. in diameter. No stone is too hard ;
6. Lack of skill in the operator.

**Lithotomy** is the removal of the stone by opening the bladder. This requires a suprapubic approach, which is easy to perform and certain in result, but means at least fourteen days and often longer in bed, with resultant weakening of the patient's general condition.

The contraindications to litholapaxy must be strictly observed, but any patient with a stone suitable for crushing should never be submitted to a suprapubic operation. There can be no conceivable justification for a surgeon performing the latter, simply because he does not possess the requisite skill to undertake litholapaxy. It is not to be forgotten that the best interests of the patient alone decide the issue.

### FOREIGN BODIES IN THE BLADDER

Foreign bodies may reach the bladder either by introduction along the urethra, gunshot wounds, injuries or operations. The first group is by far the commonest and many curious articles have been introduced into the bladder, particularly in the female, *e.g.*, hairpins, safety pins, small toys, beads, straws and catheters either whole or in part. In view of an intelligible reluctance, it is often some time before such patients ask advice. The symptoms are then those of mild cystitis with calculus. The true nature is revealed by radiography and cystoscopy. Many foreign bodies can safely be removed by an operating cystoscope ; others will require a suprapubic cystotomy.

### GROWTHS OF THE BLADDER

These can be classified as follows :—

	<i>Epithelial.</i>	<i>Connective Tissue.</i>
Benign . . . . .	{ Adenoma. Papilloma.	{ Fibroma. Fibro-angioma. Myoma. Angioma.
Malignant . . . . .	Carcinoma.	Sarcoma.

The benign growths are all rare except the papilloma ; an adenoma may be seen at the base of the bladder arising from the glandular elements in the deepest layers of the mucosa. A fibroma occurs as a submucous tumour projecting into the bladder cavity, a rhabdomyoma is occasionally seen in children, and an angioma may give rise to vesical bleeding.

### PAPILLOMATA

They are the commonest bladder tumours and over 50 per cent. are found close to the ureteric orifices. They are more frequent in men (3 : 1) and 74 per cent. occur between 30 and 60 years ;

they may be either single or multiple, and are said to have a high incidence in aniline dye workers.

*Pathology.*—Their appearance varies widely, and it is impossible to distinguish with certainty the benign from the malignant. Two groups are described. The villous type consists of delicate filaments of varying length with a fine connective tissue core, arising from a circumscribed base with a short broad pedicle. The processes can only be appreciated when floating in fluid, as on removal they collapse and the tumour appears as a soft spongy mass. They are frequently multiple and are capable of seeding out in the bladder wall and in an operation wound. The second type is coarser with short club-like processes and is rarely multiple. The pedicle may vary in length and thickness (Fig. 379).



FIG. 379

Papilloma of the bladder.

The processes consist of a loose vascular core and a covering of transitional epithelium (Fig. 380). They are a precancerous condition, and the microscope alone can reveal their true nature. Invasion of the bladder muscle is not essential to malignancy, for in the periphery, at the apex of a process, cells may be found erupting through the basement membrane.

*Symptoms.*—A symptomless, spasmodic, and often severe hæmaturia is seen. The attacks are transient, rarely lasting over five

days, and the free intervals may be months. The hæmorrhage is profuse in most cases, but in others trivial. No satisfactory explanation is forthcoming for this periodicity. There may be slight pain on micturition, and obstruction to the ureteric and urethral orifices producing symptoms of hydronephrosis or difficulty of micturition.

*Diagnosis and Prognosis.*—A cystoscopy will reveal the lesion. So difficult is the differentiation between benign and early malignant papillomata that only an expert cystoscopist can hope to attain a high percentage of correct diagnoses. Destruction of a benign papilloma leads to a lasting cure, but their multiplicity, ability to seed out and precancerous proclivities make prognosis a difficult matter, and a guarded opinion is always wise.

*Treatment—A. Endovesical.*—These tumours are easily destroyed by “fulguration” with the diathermy current applied through a catheterising cystoscope. No anæsthetic is necessary. Every benign papilloma is suitable for this method, size and multiplicity being no contraindication, the tolerance of the patient alone dictating the length of each application.

**B. Suprapubic Removal** is sometimes advised for very large or for multiple tumours. If a diathermy instrument is available, the endovesical method is to be preferred, unless the patient is unable to tolerate repeated treatments.

### CARCINOMA

It is rare before 40, most frequent between 50 and 70 years of age, and is commoner in men. The majority are malignant papillomata.



FIG. 380

Microscopic drawing of a papilloma of the bladder.

*Pathology.*—1. **Papillary Carcinoma** or malignant papilloma starts as a benign growth, and infiltration of the stroma of the processes, pedicle or base occurs. The processes become thickened and tend to fuse, an indurated area surrounds the base, and the mucous membrane becomes fixed to the underlying muscle. Ulceration, phosphatic incrustation and necrosis may occur on the surface.

2. **Scirrhous Carcinoma** is occasionally seen as a hard nodular mass with a broad base. The mucosa may be fissured and ulcerated, but this growth spreads in the submucous layer, and the mucosa is often little affected.

3. **Malignant Ulcer** is very rare and has the typical appearance of a squamous-celled carcinoma.

4. **Adenocarcinoma** is even rarer, occurs at the base and tends to spread outside the bladder.

The malignant papilloma retains its papillary character while it is still in the processes or pedicle, but on infiltrating the bladder wall it assumes a carcinoma simplex form. The scirrhus growth is a carcinoma simplex with a large amount of fibrous tissue. The ulcer is a squamous-celled growth, and the adenocarcinoma shows gland spaces lined by cubical or columnar cells.

All types of papilloma seed out locally. The malignant tumours are slow to spread outside the bladder wall and to metastasise at a distance. The growth so often obstructs the ureteric orifices that death from renal failure is more common than from general spread.

*Symptoms.*—In the malignant papilloma, the hæmaturia becomes less periodic and finally is persistent in varying amounts. Pain and frequency may follow later. The scirrhus growth will be symptomless for a time and then there occur pain, frequency and bleeding. The ulcerating growth gives pain and symptoms of chronic cystitis. Adenocarcinoma remains silent until bladder ulceration or extravescical spread has occurred.

*Treatment*—**A. Endovesical.**—1. Fulguration and radium combined. A malignant papilloma does not react to fulguration alone, but gives brilliant results from combined treatment (Geraghty). 1,000 mg. of radium are applied for one hour direct to the tumour by a special applicator, and fulguration is carried out immediately afterwards.

2. Radium alone. Fulguration does harm to any carcinoma infiltrating the bladder wall, and must never be used. Radium applied direct followed by intensive deep X-ray therapy holds some hope for the future.

**B. Suprapubic.**—Partial or complete cystectomy is advised, but according to Swift Joly the results are singularly disappointing. Small growths may be removed with success, the ureter being reimplanted if necessary.

**C. Palliative.**—Pain, hæmaturia and frequency may become so unbearable that a suprapubic cystotomy, or double nephrostomy may be needed. Repeated electrical cauterisations may temporarily stem the bleeding and pre-sacral neurectomy allay the pain.

**SARCOMA** is a rare tumour occurring either in the young or in later life. In the young grape-like masses project into the bladder cavity, while in the older type the tumour resembles a scirrhus plaque.

## THE PROSTATE AND VESICLES

*Anatomy.*—The prostate is chestnut-shaped and measures  $1\frac{1}{2}$  in. by  $1\frac{1}{4}$  in. by  $\frac{3}{4}$  in. in its transverse, vertical and anteroposterior diameters. The base is concave and surrounds the neck of the bladder, being pierced by the urethra and the ejaculatory ducts. The convex anterior surface is separated from the symphysis pubis by a pad of fat and the puboprostatic ligaments. The apex rests on the triangular ligament, and the posterior surface is flat and applied to the anterior surface of the rectum. The rounded lateral surfaces are surrounded by the anterior fibres of the levatores ani. It is enclosed in a fibrous capsule and around its lateral and anterior surfaces

is a plexus of veins (the prostatic plexus of Santorini), while outside this is a reflection of the rectovesical layer of the pelvic fascia.

The gland is described as having a middle and two lateral lobes, the former lying behind the urethra between the ejaculatory ducts and the base of the bladder. The urethra is crescentic on section, the concavity being filled by the verumontanum which projects from the posterior wall of the urethra. On its surface is the opening of the utricle in the midline, on either side of which are the orifices of the ejaculatory ducts. The prostatic ducts open into the sinus on either side of the ridge.

The prostate is supplied by branches of the inferior vesical and middle hæmorrhoidal arteries; the venous plexus drains into the vesical veins and so into the internal iliac veins. The lymphatics pass to the iliac and sacral glands.

*Anatomy of the Seminal Vesicles.*—The vesicles are a pair of glandular reservoirs lying behind the bladder, between it and the rectum and above the prostate. On surface view they have a corrugated appearance similar to a bunch of varicose veins. They converge towards the middle line, where their ducts join the vasa deferentia to form the ejaculatory ducts. The lumen is a convoluted channel with many small diverticula. The blood supply is from the middle hæmorrhoidal and inferior vesical arteries, and the lymphatics drain into the iliac glands.

*Method of Examination.*—

*A. Rectal.* With the patient in the knee-elbow position, the posterior surface can be explored by a finger in the rectum. The lateral lobes are separated by a shallow vertical groove, and on either side the finger may be pushed forward to examine the lateral surfaces. Above the prostate a transverse groove separates it from the seminal vesicles. The finger can define the extent of any enlargement, the persistence or obliteration of the vertical and transverse grooves, and alterations in consistency and sensation. Changes in the vesicles can be recognised as they are just within reach.

*B. Cystoscopy* will reveal any intravesical enlargement, and the presence of other pathological conditions in the bladder. It will assist in the differentiation of benign and malignant enlargements.

**Prostatitis.**—Acute prostatitis occurs chiefly in connection with gonorrhœa. It is rarely found in other conditions. The author has seen examples of a prostatic and a perinephric abscess of staphylococcal origin in the same patient. The symptoms and treatment are described elsewhere (p. 58).

Chronic prostatitis is a very common residual complication of gonorrhœa and is described under that heading.

Tuberculous prostatitis is dealt with under genital tuberculosis (p. 798).

**Prostatic Calculi.**—Prostatic calculi are by no means uncommon.



FIG. 381

Prostatic calculi.

They are said to arise by deposition of oxalate and carbonate of lime in the corpora amylaceæ, and are always small and multiple. Many give no symptoms, others give perineal pain and disturbances of micturition, while a few ulcerate into the urethra and thereby produce agonising pain especially upon micturition. Diagnosis can often be made by rectal palpation and confirmed by radiography (Fig. 381). They are best removed by a perineal exposure without opening the urethra.

### SENILE ENLARGEMENT OF THE PROSTATE

Prostatic enlargement occurs after the age of 55 years, and is one of the most common surgical diseases of men in later life. There are



FIG. 382

The microscopic appearance of senile hypertrophy of the prostate.

some grounds to believe that an endocrine imbalance is responsible for it.

*Pathology.*—The enlargement may affect the lateral lobes and remain entirely below the bladder, it may affect the middle lobe and produce an endovesical tumour, or more commonly both types of enlargement coexist. The change may be localised to one lateral lobe, but the anterior part of the gland in front of the urethra rarely suffers to any extent.

The enlargement of the middle lobe follows the path of least resistance, viz., upwards beneath the base of the bladder. The part affected is the small area behind the urethra and between the ejaculatory ducts. The growth pushes upwards beneath the vesical mucous membrane, displaces the internal sphincter outwards and



projects into the bladder. An endovesical tumour is then formed in the trigone covered with mucous membrane, and behind it lies the postprostatic pouch, in the floor of which the ureters open. The bladder shows hypertrophy, trabeculation and possibly sacculcation.

The microscopic appearances are those of irregularly shaped alveoli lined by columnar or cubical epithelium, standing on a deeper single layer of flattened cells with well-marked nuclei. The acini may contain typical corpora amylaceæ (Fig. 382). Although single small encapsuled adenomata occur, the great majority of these senile



FIG. 383

A bladder opened from the front to show the effects of obstruction by an enlarged prostate. The hypertrophy and trabeculation of the bladder wall are well shown. An unusually large diverticulum is present.

enlargements are in the nature of a diffuse hypertrophy in which all the elements of the prostate gland take part.

*Its Effects on the Genito-urinary System.*—(1) The urethra is increased in length, particularly along its posterior wall, and its crescentic shape on cross-section is converted into an anteroposterior slit. This increase in length has an important clinical bearing, in that an ordinary rubber catheter may fail to enter the bladder, although encountering no difficulty. A coudé or bi-coudé catheter will therefore be used. (2) The bladder shows an endovesical growth, a postprostatic pouch and all the signs of obstruction. (3) The ureters are dilated and the kidneys are in a condition of hydronephrosis. (4) Pressure on the ejaculatory ducts may obstruct them completely or partially. (5) Compression of the venous plexus leads to engorgement of the veins in the

bladder, and in some cases a condition of varicosity appears in the middle lobe and may be a source of severe bleeding. (6) Infection may be long delayed, but eventually cystitis, ureteritis, pyelitis and pyelonephritis occur with a failing renal function (Fig. 383).

*Symptoms.*—An enlarged prostate is of *no intrinsic* importance, its great significance being based on the severe damage it may do to renal efficiency. The early symptoms are frequency and difficulty of micturition.

1. **Frequency** is first noticed at night. The early hours of deep sleep are undisturbed, but afterwards the patient is awakened two or three times before rising. As time progresses the nocturnal frequency becomes more severe, and the day is also affected. It is not due to cystitis as was previously taught, for many patients have an enlarged prostate for years without infection, whereas frequency is always the first symptom. It is due to a disturbance of the neuromuscular control brought about by changes at the bladder base.

2. **Difficulty** is in every stage of the act. A minute or more may elapse before the stream starts, and straining makes it more difficult. When started, the stream has fair volume but little power, and there is a little dribbling at the finish.

Other symptoms may be hæmaturia, acute retention and sexual excitation.

3. **Hæmaturia** may be present and is not necessarily an indication of malignancy. In rare cases, severe hæmorrhage occurs in the bladder, which fills with clot leading to retention. The source of the bleeding is a ruptured vein on the middle lobe.

4. **Acute Retention** must be the fate of almost all untreated cases, and is the symptom which brings many patients to seek advice. Sudden retention is brought about by acute congestion of the mucous membrane at the bladder neck and is precipitated by excesses of food, alcohol, sexual excitement or severe chills.

5. **Sexual Excitation.**—There are grounds to believe that prostatic enlargements may lead to increased sexual excitation and account for some cases of sexual perversions. The condition may thus assume medico-legal importance.

*Diagnosis.*—Every man over 55 years of age complaining of frequency and difficulty of micturition will be suspected of having an enlarged prostate. A rectal examination reveals the enlarged lateral lobes, the cystoscope shows a middle lobe in the bladder and establishes the presence or absence of any other condition. The amount of *residual* urine will be tested. This is the amount withdrawn by catheter from the bladder immediately after voluntary micturition.

*Prognosis.*—As far as the prognosis is affected by the urinary tract condition, this can be adequately estimated by renal efficiency tests. Other coexisting lesions will need to be appraised at their own value. Generally speaking, the prognosis is good in the absence of renal impairment.

*Treatment.*—A. **Expectant.** Patients who have (1) an early and a slight enlargement, (2) a residual urine of less than 4 oz., (3) a sterile urine, (4) a normal renal efficiency, and (5) a frequency not

sufficiently severe to impair their general condition by lack of sleep, can safely be watched. They must be warned against chills and intemperance of all kinds and be re-examined every three months to check their residual urine, renal efficiency and general condition.

**B. Operative.** Patients who have (1) a residual urine exceeding 4 oz., (2) infected urine, (3) had acute retention, and (4) damaged renal efficiency with failing general health, should be advised operation. Those with acute retention will be treated by graduated catheterisation (p. 758), the bladder being completely emptied within seventy-two hours and thereafter kept empty until a decision as to operating is reached.

**Types of Operation.**—1. Enucleation with suprapubic drainage (Freyer's operation). The bladder is opened by a suprapubic incision and the prostate is enucleated with the right index finger. A large drainage tube is inserted into the bladder and the wound sewn up around it. The disadvantages of this procedure are the absence of control of hæmorrhage and the persistence of a suprapubic urinary fistula for periods varying from fourteen to twenty-eight days or even more.

2. Enucleation with closure of the bladder (Harris's operation). This revolutionary change in technique is due to Harris of Australia. The prostate having been enucleated, its bed is exposed by the use of illuminated bladder retractors and the bleeding points are ligatured. The prostatic bed is obliterated with sutures and a catheter passed per urethram is fixed in the bladder which is then carefully sutured. Constant drainage is maintained for seven days, by which time the wound has healed and normal micturition is established as soon as the catheter is removed. Patients need be confined to bed for ten days only.

3. Transurethral resection. MacCarthy's resectoscope comprises an illuminated optical system giving direct vision into the bladder and a platinum loop activated by a current of high oscillation, the whole being enclosed in a bakelite sheath. The intravesical projection and that part of each lateral lobe proximal to the ejaculatory duct are removed piecemeal by a series of cuts with the loop, the object being to remove sufficient tissue to restore the calibre of the normal urethra. An indwelling catheter remains *in situ* for seven days, after which normal micturition should be re-established. Some urologists prefer to use a cold cutting blade instead of the electrified loop.

It must be clearly understood that no removal of the prostate should be attempted until the urea clearance tests have shown a satisfactory renal function. The blood urea must not be above 70 mg. per 100 c.c. of blood. If these tests are not satisfactory the bladder must be drained by an indwelling catheter and irrigated for several days with silver nitrate (1 : 6000) or oxycyanide of mercury (1 : 7500). No resection should be attempted in the presence of a rise in temperature.

Harris's operation and transurethral resection have obviated the use of suprapubic drainage and thereby reduced the complications of prostatectomy. There is no doubt that the prognosis has been greatly

improved by these methods. The selection of operation will depend upon the age of the patient, his general condition, the presence of sepsis and the size of the prostate. Under favourable conditions it would be better to remove the whole prostate, and resection therefore is to be preferred in those men who are not really suitable for the major procedure.

**PROSTATIC OBSTRUCTION WITHOUT ENLARGEMENT.**—There are two conditions which simulate an enlarged prostate. **Young's Median Prostatic Bar** consists in a submucous fibrosis in the base of the bladder just at the entrance of the urethra. It arises in connection with the middle lobe or the subtrigonal glands and is not associated with previous prostatitis. **Fibrosis** of the prostate due to chronic prostatitis may also deform the urethral opening. These two conditions cause symptoms similar to those of senile enlargement, but they can be readily diagnosed with the cystoscope. They are treated by trans-urethral resection.

### CARCINOMA OF THE PROSTATE

This occurs during the latter part of life, from the age of 45 years onwards, being of somewhat earlier incidence than the senile enlargement.

*Pathology.*—The tumour is of the hard scirrhus type and only very rarely is a soft growth seen. It spreads by extension to the bladder, rectum and pelvic cellular tissues, by lymphatics to the iliac glands and by metastases to the liver, lungs and especially the bones. Microscopically it is usually spheroidal-celled, with much fibrosis, but occasionally rapidly growing columnar-celled tumours occur.

*Symptoms* are pain, frequency and difficulty of micturition, hæmaturia and rectal ulceration. Pain is felt in the perineum, hypogastrium and down the back of the thighs. It is persistent and is not relieved by micturition; frequency is not so pronounced as in the senile hypertrophy; difficulty may be a prominent feature and will lead eventually to retention; hæmaturia is not a frequent symptom but may become persistent; rectal involvement will cause pain and tenesmus, with the passage of blood and mucus in the stools, and later, if the sphincter is invaded, a colostomy may become necessary to relieve intestinal obstruction. These growths have a curious tendency to spread either forwards to involve the urethra and bladder base or backwards into the rectum. The symptoms which predominate therefore will be either urinary or rectal, and not until the later stages will both bowel and bladder be involved. Bone metastases may be the first sign and these may take place in the bones of the pelvis by direct spread or in any bone at a distance by vascular embolism.

Rectal examination reveals a very hard, nodular and irregular enlargement of one lateral lobe of the prostate which is fixed. The rectal mucosa does not move over it and an extension may be felt spreading upwards and outwards to the lateral pelvic wall. An important sign is the obliteration of the median groove.

*Prognosis.*—Many of these growths progress very slowly and

patients may live for many months or years. Any expression of opinion should be most guarded.

*Treatment.*—Deep X-ray therapy has given many wonderful results and is the treatment of choice. As soon as difficulty of micturition occurs, the obstruction can be widely removed by transurethral resection. Radical surgery and radium are of little, if any, value.

**Sarcoma of the Prostate** is a very rare condition, seen occasionally in children and proves rapidly fatal.

**The Vesiculæ Seminales.**—Infection of the vesicles is either gonococcal or tuberculous, and these are dealt with in the chapters on venereal infection (p. 59) and genital tuberculosis (p. 798).

R. M. HANDFIELD-JONES.

## CHAPTER XXXVII

### THE PENIS AND URETHRA

**S**URGICAL ANATOMY.—The Penis serves a double purpose, genital and urinary. It is composed of three cylindrical bodies of erectile tissue, one of which transmits the urethra. These are bound together by a sheath of fibro-elastic tissue, named Buck's fascia, and are covered by skin and subcutaneous tissue. The two corpora cavernosa arise in the crura of the penis from the ascending rami of the ischium, and meet below and in front of the symphysis. Thence they run parallel and fixed together to meet the glans. The corpus spongiosum fits into the groove on their ventral surface and has two expanded extremities. The anterior forms the glans which caps the ends of the corpora cavernosa, and the posterior expansion is the bulb, which lies between the crura. The skin moves freely over the whole organ, and at its extremity is prolonged over the glans to form the prepuce, at the edge of which it is reflected back to unite with the mucous membrane of the glans at the coronal sulcus. The two layers are separate and allow of retraction and uncovering of the glans. The prepuce is connected to the under surface of the glans by a fold of mucosa, named the frenum, containing the frenal artery. **The Urethra** is divided into prostatic, membranous and bulbo-penile parts. The prostatic urethra lies within the prostate, is  $1\frac{1}{4}$  in. long, and is directed downwards and slightly forwards. It is spindle-shaped, and along its posterior wall runs an eminence, the verumontanum, on the surface of which open the utricle and ejaculatory ducts. On cross-section it has a reniform shape. The membranous part lies between the layers of the triangular ligament and is surrounded by the compressor urethræ muscle. It is directed downwards and forwards, and its anterior wall is  $\frac{3}{4}$  in. long, the posterior wall being slightly shorter owing to the obliquity with which it enters the bulb. Cowper's glands lie behind and external, but their ducts run parallel to it and enter the bulb. The bulbo-penile part is surrounded by the corpus spongiosum and varies between 5 and 7 in. at rest and 8 and 10 in. when erect. The bulbous part runs directly forwards making an angle of 93 degrees with the membranous urethra and is fixed. It becomes the free or penile part at the penoscrotal junction. When flaccid the urethra forms an S curve. Its calibre varies as follows :—

Prostatic urethra—beginning and end . . .	$\frac{1}{4}$ in.
"          "          middle . . .	$\frac{1}{8}$ "
Membranous urethra . . .	$\frac{1}{5}$ "
Bulbo-penile—Cul-de-sac of bulb . . .	$\frac{1}{2}$ "

Bulbo-penile—Main channel	. . . . .	$\frac{1}{4}$ in.
„ Fossa navicularis	. . . . .	$\frac{1}{8}$ „
External meatus	. . . . .	about $\frac{1}{8}$ „

The least distensible portions are the external meatus and the membranous.

**Internal Structure.**—The lining membrane is columnar-celled, except in the fossa navicularis, and the prostatic portion, in which it is squamous and transitional respectively. The roof and sides, and to a lesser extent the floor are studded with the openings of the glands of Littré. The ducts of Cowper's glands open on the floor of the bulb 1 in. in front of the triangular ligament. The membranous urethra opens into the roof of the bulb, and the cul-de-sac of the bulb extends back  $\frac{1}{8}$  in. beyond the opening, thus presenting a dangerous area for false passages. This part of the bulb is the most low-lying of the whole tract, and it is in this position that pus collects in urethritis. The penis is supplied with blood by the deep internal pudic artery and by the superficial external pudic artery. The lymphatics go to the inguinal and deep femoral glands and to those on the external iliac vessels.

**Clinical Division of the Urethra.**—A more useful clinical description is to use the compressor urethræ muscle as the dividing line between an anterior and a posterior urethra. The differences between them can be thus summarised (Barringer):—

#### *Anterior Urethra.*

Surrounded by erectile tissue (corpus spongiosum) for entire length, excepting for  $\frac{1}{2}$  in. in the roof of the bulb.

Many glands of Littré in roof and sides.

Ducts of Cowper's glands enter bulb.

External urinary tract in free communication with the surface of the body and harbours all the micro-organisms that may be thereon.

Fixed at one end only (triangular ligament), therefore can assume any curve (*e.g.*, on passing a sound) without causing pain to the patient.

Fluid may be introduced into anterior urethra and held there by compressing urethral meatus.

#### *Posterior Urethra.*

No erectile tissue covering.

Very few glands of Littré.

Ducts of prostatic glands enter prostatic urethra. Verumontanum with ducts of seminal glands in prostatic urethra.

The lowest section of the aseptic internal urinary tract—entirely free from bacteria, harboured by anterior urethra.

Fixed at one end by the triangular ligament and at the other by the prostate; so having a fixed "U" curve which when straightened (for example, on introduction of a cystoscope) causes pain to the patient.

Fluid cannot be retained in posterior urethra. The compression of the surrounding muscles drives it either back into the bladder or forward into the anterior urethra.

*Anterior Urethra.*

The introduction of a foreign body (*e.g.*, fluid or catheter) into the anterior urethra causes only pain or burning.

Inflammation causes simply pain.

There are no voluntary muscles surrounding the anterior urethra which can resist the introduction of a fluid or an instrument.

*Posterior Urethra.*

The introduction of a foreign body (fluid or catheter) into the posterior urethra causes pain plus a desire to micturate.

Inflammation causes pain plus frequency of micturition.

By means of the perineal muscles the introduction of an instrument or fluids can be voluntarily resisted; therefore as the sound or catheter approaches the posterior urethra, manipulations must be very gentle.

**Clinical Examination.**—The penis is available for digital examination in its whole length, as is the urethra, the prostatic and membranous portions being approached by a finger in the rectum. (For ease and accuracy the presence of a bougie *in situ* is of great assistance.) Bougies of metal or gum elastic of the acorn-tipped type demonstrate strictures, stones and other lesions. The urethroscope allows direct vision of the entire length of the urethra. It carries a lamp at the beak, and the hollow tube is closed by a window at the top. The urethra is first distended by air, when it can be thoroughly investigated.

**CONGENITAL ANOMALIES**

**Double Penis** is very rare, only about thirty cases being recorded, all of which were accompanied by some other defect of the genito-urinary system. **Complete absence** is rarer still; concealed penis implies a small penis lying beneath the scrotal skin. A **webbed penis** is attached to the scrotum along its ventral surface, and can be freed by a simple plastic operation. **Occlusion of the urethra** is due to membranes or folds of mucosa near the fossa navicularis or in the prostatic urethra; this condition may be combined with urine being passed into the rectum. Division and bougie treatment, or even a plastic operation may be needed.

**Hypospadias** is a defect of the anterior urethra, whereby the canal fails to reach the external meatus, and according to the position of the opening may be termed glandular, penile or perineoscrotal. The posterior urethra is always intact and sphincter control is perfect. The perineoscrotal variety, fortunately rare, is accompanied by a cleft scrotum, imperfectly descended testes and a condition of pseudo-hermaphroditism. In the other types a well-marked groove between the opening and the meatus represents the imperfectly formed roof of the canal. In the glandular cases nothing need be done, and for the other penile types a variety of plastic operations has been described.

**Epispadias** is a far more serious defect and is very rare. The urethra lies on the dorsal surface of the corpora cavernosa and the roof is wholly or partly missing. In the former an ectopia vesicæ and separated pubic bones are present, while in the latter the canal opens



somewhere on the dorsal surface. In all these cases sphincter control is imperfect. Young's operation brings about, by a reconstruction of the penis and urethra, an almost normal appearance.

## THE PENIS

### PHIMOSIS

Phimosis may be congenital or acquired and consists in an inability to retract the prepuce over the glans penis due to smallness of the preputial opening, unusual length of the foreskin or adhesions.

**Congenital Phimosis.**—In infants, the inner layer of the prepuce is slightly adherent to the glans through the medium of a fine epithelial tissue, which disappears during the first two years. Later, the normal foreskin can be retracted over the glans and slips back without difficulty and without interfering with its blood supply even during erection. The prepuce may be short or abnormally long. In early years, phimosis is of no importance unless it gives rise to complications, but later, difficulties during erection and coitus render treatment necessary. The complications are :—

- (a) Difficulty of and pain on micturition, due either to a small preputial opening or to this and the meatus being out of alignment.
- (b) Retention of urine, either in the bladder, leading to back-pressure and bilateral hydronephrosis, or in the preputial sac leading to balanoposthitis and preputial calculi.
- (c) Herniæ or prolapse of the anal canal, due to excessive straining.
- (d) Paraphimosis.
- (e) Difficult and painful coitus ; sexual neurasthenia.
- (f) Carcinoma of prepuce or glans.

**Acquired Phimosis.**—This may be temporary, when inflammatory lesions, *e.g.*, gonorrhœa, chancre or other sores, narrow the opening and retraction is impossible ; or permanent, as a result of scar formation produced by the healing of the foregoing lesions. It is this type which predisposes to carcinoma.

**Treatment.**—In an attack of balanoposthitis, treatment is limited to an incision along the dorsum of the prepuce until the infection subsides. Circumcision must be performed as a primary measure in all other cases.

### PARAPHIMOSIS

This is a condition in which the prepuce has been retracted, slips into the coronal sulcus and cannot be returned. Interference with the blood supply by the tight ring sets up a vicious circle. The glans becomes œdematous and discoloured, and the ring becomes tighter until necrosis may occur and spontaneous relief be obtained.

The constricting ring is always hidden by a swollen cuff of the inner skin layer of the prepuce which overlaps it from in front. Gangrene of the glans is rare.

**Treatment.**—In early cases the prepuce can be reduced by pressure on the glans with the thumbs and traction on the skin with the fingers. If this fails, an incision into the constricting ring relieves the condition. As soon as any accompanying infection has subsided circumcision should be performed.

**PREPUTIAL CALCULI** are rare and only occur in cases of phimosis with a voluminous prepuce. They may be formed *in situ* from retained urine or from calcification in smegma, or passed into the preputial sac per urethram. Their presence leads to inflammation, and they must be removed and the patient circumcised.

## INJURIES

**Ruptured Frenum.**—The frenum may be so short that curvature of the penis occurs during erection, making coitus painful or impossible and leading to tearing of the frenum with profuse bleeding from the artery. Simple ligature of the vessel and division of the frenum are required.

**Injuries of the Penis.**—Subcutaneous bruising occurs during erection and usually in connection with coitus. It varies from a slight contusion of the skin to a rupture of the sheath of a corpus cavernosum, the so-called fracture of the penis. In the more extensive lesions, there is swelling and pain, and damage to or pressure on the urethra causes retention of urine.

**Treatment.**—The patient is put to bed, and the penis wrapped in dressings of lead and opium lotion. Morphia may be needed for the pain. Usually the blood is absorbed rapidly. In grave cases operation must be considered, and becomes imperative if the urethra is torn. Tears in the corpora cavernosa will heal more rapidly if sutured, and will be less likely to cause angulation or defects in erection afterwards.

**Dislocation of the Penis.**—In rare instances the skin of the prepuce is torn away from its junction with the glans and the penis is displaced from its skin covering, and comes to lie in the groin, or scrotum or in front of the symphysis pubis. The skin hangs down empty in its normal position and blood drips from it or collects in clots inside. Extravasation of urine or retention will follow. In treatment, the penis is replaced in its skin covering and sutured, and injuries to the urethra sought for and repaired.

**Strangulation by Foreign Bodies.**—Strands of hair, thread, string and metal rings are used either as a means of sexual excitation, to prevent eneuresis or as a form of misplaced humour. The penis swells up distal to the constriction and it is impossible to remove the band except by dividing it, which in the case of metallic rings may be difficult.

## INFECTIONS OF THE PENIS

**Balanoposthitis.**—Acute inflammation of the glans penis (balanitis) and of the prepuce (posthitis) can hardly occur separately, and the condition should always be described by the combined name.

The essential factor is a lack of personal cleanliness, and additionally there may be venereal infection, retained smegma, diabetes, or a chemical irritant as in misguided efforts to prevent infection after illicit intercourse.

*Symptoms.*—Burning and itching usher in a swelling of the prepuce which leads to an acquired phimosis. Pus drips from the opening and inflamed lymphatics may be palpated running up the penis. Later, surface ulceration of the glans and prepuce may occur.

*Treatment.*—Adequate drainage and access must be ensured by slitting up the prepuce if necessary. The irritating cause must be identified and dealt with, and absolute cleanliness insisted upon. Bathing with potassium permanganate (1 : 8000) is useful. Circumcision is performed after the infection has subsided.

**Cavernositis.**—Acute cavernositis will be described under Periurethral Abscess. Diffuse cavernositis is rare, is due to pyæmic metastasis or thrombosis, and is usually fatal.

Chronic cavernositis may be due to gonorrhœa, syphilis, gout or tuberculosis. Thickened nodules or plaques appear in one or both corpora cavernosa, and cause great distress as they result in curvature of the penis when erect, rendering coitus painful or impossible. They are most common after gonorrhœa. In treatment, any residual focus of infection in the prostate or vesicles must be searched for and treated. Injections of contramine weekly will prevent any extension of the plaque and may soften the existing fibrosis. Surgical treatment is valueless.

#### NEW GROWTHS OF THE PENIS

There are only two growths worthy of record, a benign papilloma and malignant carcinoma.

**Papilloma.**—The true papilloma differs in no way from a squamous-celled papilloma of the skin elsewhere. It should be regarded at or after middle age as precancerous and removed for microscopy.

An inflammatory wart is seen as a complication of venereal infection, and is described under the complications of gonorrhœa.

**Carcinoma** is rare before 40 and most common between 55 and 70 years of age. Papillomata and leucoplakia (similar to the lingual) are precancerous conditions. Acquired phimosis with a rigid preputial opening, rendering the skin liable to chronic irritation, is favourable to malignant change.

*Pathology.*—Most penile carcinomata start as malignant papillomata on or behind the glans (Fig. 384), either on the inner surface of the prepuce or at its opening. Infiltration at the base causes a spreading warty growth which does not ulcerate early. The thick sheaths of the corpora prevent their involvement until late. An ulcer, when it occurs, has all the characteristics of a squamous-celled carcinoma. The growth may erode the prepuce and appear on its outer surface, or may open up the urethra with the formation of one or more urinary fistulæ.

The vast majority are squamous-celled, but a few are adenocarcinomata with cubical or columnar epithelium.

The inguinal glands on both sides are involved, and direct spread to neighbouring structures occurs later. As soon as the growth erupts into the corpora cavernosa, spread along the erectile tissue is rapid.

*Symptoms.*—Irritation and discharge are first seen; then painful erections, swelling, ulceration and fistulæ occur. In elderly men irritation and discharge with an area of induration to be felt in a penis of normal appearance call for urgent investigation, and circumcision should be urged as a diagnostic measure.

*Treatment.*—Two operations are practised, partial and radical amputation.

In early cases, with growths limited clinically to the glans and with a long penis, a partial amputation just in front of the scrotum is performed and the glands cleared from each inguinal region. In more advanced cases the whole penis back to the triangular ligament is removed, and some surgeons advise the ablation of the scrotum and testes. The urethra must be slit up before implantation into the skin to prevent a stricture forming at the line of suture.

*Prognosis* is fair, as these growths tend to be slow-growing.



FIG. 384

Squamous-celled carcinoma  
of penis.

## THE URETHRA

### URETHRITIS

The vast majority of cases of urethritis, both acute and chronic, are gonococcal in origin and are described under that subject. Others are due to the usual pyogenic cocci and *B. coli*. In the face of denial of the possibility of venereal infection, it is wise to remember that there are other infections of the urethra besides the Neisserian.

The *treatment* is identical with that of gonorrhœa (see p. 61).

### INJURIES

**Direct Injuries** may occur during the passage of instruments, the extraction or passage of stones or other foreign bodies, and as the result of penetrating wounds. The bulbo-penile urethra is most frequently affected, hæmorrhage and occasionally extravasation of urine following. Punctures and linear tears need no treatment, but if the urethra is extensively torn or divided and if extravasation threatens, the wound must be explored and the edges sutured.

**Indirect Injuries** or ruptures of the urethra vary in extent and position. The mucous membrane only may be contused or split, or the tear may spread through the outer sheath and into the corpus spongiosum. The whole or part of the circumference of the canal may be affected. The penile urethra is rarely injured, and then only in erection; lesions of the bulb occur during falls on the perineum astride a bar or from kicks in this region; and the membranous part is lacerated by such injuries as produce fractures and dislocations of the bones of the pelvis.

*Symptoms* vary with the situation. Penile ruptures produce severe hæmorrhage, some pain on micturition but no retention. Bulbous lesions give less bleeding, but a swelling appears in the perineum, and there is retention. In the membranous urethra the grave general condition of the patient so overshadows the urethral lesion that it may pass unrecognised until retention or extravasation make its presence obvious. Extravasation will not occur until an attempt to micturate is made. Later, extensive bruising in the perineum appears.

*Diagnosis.*—In the majority of cases the type of injury, perineal swelling and hæmorrhage make the condition obvious, but in those complicating fractures of the pelvis the diagnosis may give rise to grave anxiety. So serious is it to leave a ruptured urethra unrecognised that every patient with a fractured pelvis or other similar injury should be suspected of a urethral lesion, and immediate steps taken to exclude or treat it. The patient is instructed not to pass his water, and an attempt is made with even more than usual gentleness to pass a catheter. If this passes smoothly and painlessly and urine free from blood is obtained, and if no bleeding follows its withdrawal, a ruptured urethra is improbable.

*Prognosis.*—If recognised early, there is no danger to life, but if extravasation has occurred the mortality is 40 per cent. In every case a traumatic stricture will follow unless prevented by treatment.

*Treatment.*—Open operation is imperative as the future behaviour of the resulting stricture depends directly on the perfection of apposition of the edges. Access is gained to the bulbous urethra by a median perineal incision, and to the membranous by a curved transverse one with the concavity forward. A rubber catheter, passed down to the tear, will define the distal end. All clot is washed away and the proximal end identified. The catheter is then passed into the bladder and the urethra sewn over it, the perineum being closed with drainage. The catheter remains *in situ* for a week. In complete ruptures the retraction of the proximal end may make its recognition difficult, but it *must* be found, even if this needs a suprapubic cystotomy with retrograde catheterisation. If a length of urethra is missing as in gunshot wounds, a catheter is passed as before across the gap and the wound lightly packed and drained. In all cases persistent and prolonged instrumentation is needed to prevent the scar contracting down.

## STRICTURE

This may be congenital, traumatic, spasmodic or inflammatory.

**Congenital Strictures** occur at the external meatus, at the junction of the fossa navicularis with the penile urethra, and in the membranous and prostatic portions. The narrow external meatus is the most common, and may lead to difficulty in micturition in infancy and to imperfect seminal emissions later. The operation of external meotomy consists in an incision in the floor towards the frenum, the mucous membrane being stitched to the skin of the glans.

**Traumatic Strictures** follow injuries (see above). Fibrosis occurs between the torn ends and, unless coaptation be accurate, the scar will be widespread and dense.

**Spasmodic Strictures** follow the so-called "Saturday-night" excesses. In every case a moderate degree of organic stricture is present, to which is added a congestion of the mucous membrane leading to acute retention.

**Inflammatory Strictures** occur between the ages of 20 and 55 years, and 98 per cent. follow gonorrhœa. Tuberculosis, diabetes and urethral chancre account for the remainder.

*Pathology.*—In an attack of urethritis the infection, spreading to the glands in the urethral wall, may be imperfectly drained. As a result the inflammatory process spreads to the submucous coat and finally into the corpus spongiosum. On the subsidence of the resulting periurethritis a scar forms, the contraction of which leads to a narrowing of the canal. The majority of strictures occur in the bulbous urethra within an inch of the opening from the membranous. This is the most dependent part of the fixed urethra, where drainage is least efficient. Strictures never occur primarily in the prostatic urethra, but the fibrotic process may spread backwards from the bulb. Although, clinically, strictures may appear multiple, the several constrictions are integral parts of a single pathological process.

*Types of Stricture.*—*Annular* affects the whole circumference; *Brille* affects part of the wall only and stretches across the lumen; *Cartilaginous*, *Massive* and *Palpable* refer to size and consistence; *Resilient* denotes those which contract down again quickly after easy dilatation; *Permeable* when urine can be passed; *Passable* and *Impassable* refer to the passage of catheters. The lumen may be central or eccentric, straight or tortuous.

*Pathological Results.*—These have been dealt with under Retention of Urine (p. 760). They are hypertrophy, trabeculation and sacculation of the bladder with bilateral hydronephrosis and, later, infection of the kidneys with failing renal function. The urethra proximal to the stricture is dilated and thickened, and false passages, periurethral abscess and fistula may be seen.

*Symptoms* are a history of gonorrhœa, probably a slight chronic discharge, difficulty in micturition, some frequency aggravated by sexual and alcoholic excess, and later, acute retention. Straining is necessary to start the act, and the stream is of small volume and slow. The act does not end abruptly and cleanly, but the last ounce dribbles

away, often after apparent cessation. Alteration in the shape of the stream is of no diagnostic importance.

*Complications* are acute retention, sepsis throughout the genito-urinary tract, extravasation of urine, fistula, stone and carcinoma of the urethra.

*Diagnosis*.—A stricture is identified by passing bougies and by the urethroscope.

*Treatment* rests between dilatation and operation—**A. Dilatation**.—Most strictures can and should be dealt with by dilatation, of which there are three methods, viz., intermittent, continuous and rapid. Intermittent dilatation—the method of choice—consists in a well-regulated technique lasting over a period of years. At the start of treatment the largest bougie, which will comfortably traverse the stricture without undue resistance, is passed. Every third day bougies of gradually increasing size are used until 25 to 30 F. is reached. After this the full-size bougie is passed once a fortnight for three months, once a month for the next six months, then every third month for a year. The patient should then be seen twice yearly. Continuous dilatation is used in cases of acute retention complicating a tight stricture. A filiform bougie is passed and left *in situ* for twelve hours, the urine trickling slowly alongside it. The stricture will now admit a larger bougie and this also is left in for twelve hours, at the end of which time the stricture is suitable for the commencement of intermittent dilatation. Rapid dilatation is performed by a special expanding metal instrument—the Kohlmann dilator. Its use should be reserved for selected cases in expert hands, as much harm can be done by ill-judged rapidity.

**B. Operative Treatment** should be employed only in cases unsuitable for dilatation. Thomson Walker classified these as follows :—

1. Dilatation has been tried and failed.

- (a) Cartilaginous, resilient or irritable strictures.
- (b) Hæmorrhage after dilatation.
- (c) Recurrent epididymitis or retention after passage of bougies.

2. Dilatation unsuitable.

- (a) Impassable stricture.
- (b) Urethral complications, such as stone, periurethral abscesses, extravasation or fistula.
- (c) Other complications demanding treatment, *e.g.*, enlarged prostate, vesical stones, infections or growths.

The following operations are practised :—

1. **INTERNAL URETHROTOMY** consists in division of the stricture by a specially designed knife working within the urethra. The stricture must be passable, as all types of urethrotome depend upon the passage of the leading part of the instrument through it. In the Otis urethrotome the knife is concealed among the blades of a dilator, in the Teevan and Maisonneuve instruments a whalebone or gum-elastic guide serves to direct the knife to the stricture. The incision is made in



the roof of the urethra, because here a too deep cut will merely enter the septum between the two corpora cavernosa and not open up the corpus spongiosum, which would lead to abscess formation. Intermittent dilatation follows the operation. In spite of many superficial attractions, this operation should be restricted to passable resilient strictures in the penile portion only, in which the degree of sepsis is slight, of low virulence and confined to the urethra.

2. **EXTERNAL URETHROTOMY WITH A GUIDE** (Syme's operation). This operation also requires that the stricture be passable, so that the narrower extremity of the Syme's shouldered staff can be passed through it. The urethra is opened through the perineum just distal to the stricture, which is divided by running the knife along the groove in the staff. A No. 16 rubber catheter is passed down the urethra, guided through the stricture area, and on into the bladder and left *in situ* for seven days, while the wound granulates around it. The after-treatment consists in full intermittent dilatation. This operation is reserved for passable strictures in the bulbous urethra, which have failed to yield to instrumentation.

3. **EXTERNAL URETHROTOMY WITHOUT A GUIDE** (Wheelhouse's operation) is the operation of choice for all impassable strictures. The Wheelhouse grooved staff is passed to the face of the stricture and the urethra opened on the groove. The cut edges are widely retracted, and the lumen of the stricture sought for. A filiform bougie is passed through it, the fibrosed area divided along the bougie, and a silver perineal tube introduced into the bladder. The usual after-treatment by intermittent dilatation follows the healing of the wound. In some cases the most careful search fails to reveal the proximal opening, and retrograde catheterisation through a suprapubic opening is needed to identify the urethra above the stricture.

4. **RESECTION OF THE STRICTURE** is admirable in theory but disappointing in practice, and can only be done in very short strictures. Cabot's plastic operation divides the stricture longitudinally and sews it up transversely.

5. **EMERGENCY METHODS** to relieve acute retention. In impassable strictures with retention, when circumstances render operation difficult to arrange for some hours, the distension of the bladder may be sufficiently great to demand relief. Suprapubic puncture above the pubes with a trocar and cannula is suitable for cases with no urinary infection, and can therefore only be used in early cases without cystitis. In all others a suprapubic cystostomy gives adequate drainage, prevents the possibility of abscess formation in the abdominal wall, and further permits retrograde catheterisation in difficult cases. Cock's perineal puncture should be performed only by those skilled in this method and has no advantage over suprapubic drainage.

### PERIURETHRAL ABSCESS

The infection is urethral in origin, the organisms being the gonococcus, staphylococcus, streptococcus or bacillus coli, and is in many cases mixed. It may occur during an acute attack of gonorrhoea,



follow infection behind a stricture or a foreign body, or be associated with false passages from faulty instrumentation, an indwelling catheter or carcinoma of the urethra. It may affect either the penile or bulbous portion.

*Symptoms.*—A small hard nodule appears in the floor of the urethra and enlarges towards the surface. The skin becomes red, tender, boggy and finally fluctuant. If untreated it may rupture either on to the surface, into the urethra, or both ways, in which last case a urinary fistula is formed. The signs of general toxæmia vary, some cases being ill with rigors and a high temperature, while in others the onset is more insidious. Complete or partial retention is common.

*Treatment.*—The abscess should be opened as soon as diagnosed. A median incision in the line of the urethra will permit of access to break down all pockets and secure adequate drainage, thus preventing the danger of urinary fistulæ formation. The coexisting stricture or urethritis must also be dealt with.

### EXTRAVASATION OF URINE

This is due to rupture of the bladder or urethra. The former has been described (p. 760). Rupture of the urethra may be traumatic, but in many cases extravasation occurs behind a stricture. Only rarely does it follow a periurethral abscess. The attachments of Colles's fascia to the triangular ligament and of Scarpa's fascia to the deep fascia in the thigh determine the direction of spread of urine; thus, it will track along the perineum into the scrotum, along the spermatic cords to the thigh and anterior abdominal wall, and between Scarpa's fascia and the muscle sheaths. It also follows the cord along the inguinal canal and enters the subperitoneal planes. Fortunately, the incidence of this serious condition has greatly decreased in the last twenty-five years, and it is now rarely seen.

*Symptoms.*—The determining factor is evident in the history. The onset is abrupt, being ushered in by a rigor and perineal pain. The temperature rises to 102° F. or more, and all the signs of toxæmia are early apparent. Urine is passed in small quantities and with difficulty, and there may be some urethral bleeding. A swelling appears in the perineum and spreads rapidly to the penis, scrotum, abdominal wall and thighs. In the early stages it is dull red, brawny and indurated, while in neglected cases gangrene and sloughing of the skin occur and urine trickles slowly away. A fatal ending is common.

*Treatment* consists in immediate multiple incisions throughout the indurated area. These must be carried through the fasciæ of Scarpa and Colles, and adequate drainage obtained. If possible, a catheter is passed per urethram into the bladder and if this fails, the urine must be side-tracked by a suprapubic cystotomy.

### URETHRAL FISTULA

This may be congenital, traumatic, inflammatory or neoplastic. The congenital are associated with hypospadias and epispadias, or

combined with other malformations such as an imperforate anus. The traumatic and neoplastic types are very few. The majority are the result of an imperfectly drained periurethral abscess, but other inflammatory causes are prostatic and perianal abscesses and gummatous ulceration in the perineum.

The urine is voided only during the act of micturition and, as the openings are frequently multiple, the discomfort of a spray-like effect can be well imagined. Treatment is difficult and often unsatisfactory. Any co-existing condition must be treated, and after excision of the fibrosed area an attempt is made to close the defect by plastic flaps.

### URETHRAL CALCULI

These may be formed in the urethra or arrested there during transit. Provided the urethra is normal their passage should be certain though painful. If impaction occurs, the stone should be pushed back into the bladder and removed with a Bigelow's evacuator. If a stricture is present, an external urethrotomy will be necessary.

### FOREIGN BODIES

An oddly assorted collection of foreign bodies may be passed into the urethra. Pain, burning and hæmorrhage are present and aggravated by erection; later, a purulent urethritis probably follows. If allowed to remain, phosphatic incrustation and a periurethritis occur. They may be removed through a urethroscope or pushed on into the bladder and removed thence.

### URETHRAL GROWTHS

All growths are rare, but a benign papilloma and hæmangioma are described. Carcinoma, apart from the penile type, occurs between 45 and 70 years of age and may be squamous or columnar-celled. It is first noticed by urethral bleeding and some difficulty in micturition. A hard mass can be felt, and later, invasion of the corpus spongiosum and of the skin leads to urinary fistulæ. A radical amputation of the penis is required.

R. M. HANDFIELD-JONES.

## CHAPTER XXXVIII

### THE TESTIS AND SPERMATIC CORD

**S**URGICAL ANATOMY.—The testes are a pair of oval glands, each measuring  $1\frac{1}{2}$ ,  $1\frac{1}{4}$  and  $\frac{7}{8}$  in. in length, breadth and thickness, hanging each in its own compartment of the scrotum, with its long axis directed upward and slightly forward and outward. The left hangs lower than the right and both are attached to the bottom of the scrotum by the fibrous remains of the gubernaculum.

The Epididymis, composed of the convolutions of the excretory duct, lies along the posterior border of the testis. Its upper expanded end, which caps the upper pole, is named the globus major. The lower smaller end reaches the lower pole and is called the globus minor, while the intervening part is the body. The globus major and minor are firmly attached to the testis by fibrous tissue, and the former is continuous with the testicular tubules through the efferent ducts. The vas deferens emerges from the globus minor to turn upwards and enter the spermatic cord. On the anterior aspect of the globus major or of the superior pole of the testis are two small bodies, the stalked and the sessile hydatids of Morgagni, representing remains of the Müllerian duct. Embedded in the cord just above the testis is a collection of rudimentary tubules, viz., the organ of Giraldes, a remnant of the Wolffian body.

The blood supply is from the spermatic artery and the artery to the vas. The veins enter the cord to form the pampiniform plexus which becomes the spermatic vein at the internal abdominal ring. The spermatic arteries arise from the aorta near the renal arteries. The left spermatic vein enters the left renal vein and the right the inferior vena cava. The lymphatics drain chiefly to the glands on and between the aorta and vena cava. The nerves are derived from the aortic and renal sympathetic plexuses.

The tunica vaginalis is a closed sac which embraces the greater part of the testis and epididymis. It is derived from the peritoneal diverticulum, which precedes the descent of the testicle from the abdomen—the processus vaginalis. It is composed of two layers, visceral and parietal, enclosing a potential cavity. The visceral layer clothes the testis and epididymis and is reflected from them at the posterior border to form the parietal layer which lines the scrotal sac. Between the body of the epididymis and the testis it forms a recess—the digital fossa—and it is prolonged upwards for 1 in. along the cord.

The spermatic cord extends from the internal abdominal ring to the testis, traversing the inguinal canal for the first 2 in. It then passes through the external ring and enters the scrotum. It consists of the vas deferens, the artery to it, the spermatic and cremasteric arteries, the pampiniform plexus of veins, the testicular lymphatics, the spermatic plexus of nerves and the genital branch of the genito-crural nerve. These structures are held together by loose areolar tissue and the whole is contained within the following coverings, from within outwards :—

1. The subperitoneal fatty layer,
2. The internal spermatic fascia from the fascia transversalis,
3. The cremasteric fascia from the internal oblique muscle,
4. The external spermatic fascia from the external oblique,
5. The dartos tissue,
6. The integument.

The testis normally lies behind, below and internal to the tunica vaginalis, but sometimes the relations are reversed and it lies above, in front and external.

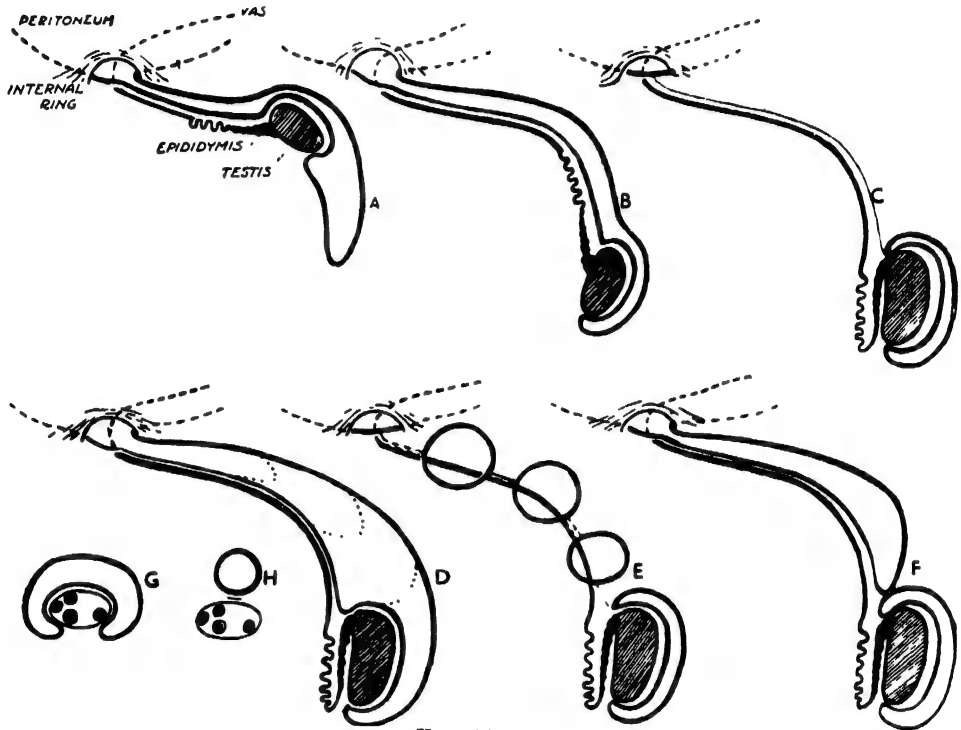


FIG. 385

The relation of the peritoneum to the descent of the testis. The potential sites of hernia and hydrocele formation.

A, the preceding peritoneum during testicular descent. B, patent processus vaginalis. C, complete or normal closure with the epididymis applied to the testis. D, potential sites of hernia sac formation. E, potential sites of hydrocele formation. F, common type of infantile hernia. G, a cross-section of the cord in a congenital sac. Note the sac nearly surrounds the cord. In the female the round ligament constantly shows this relationship. H, the relation of the acquired sac to the cord.

*The Descent of the Testis.*—The testes develop in the abdominal cavity behind the peritoneum at the level of the 1st lumbar vertebra. During foetal life a slow migration occurs, till at birth the testes should be in the scrotum. No convincing explanation of the mechanism which produces this migration has been forthcoming. A brief description of the facts must be given, as without them imperfect descent and certain anomalies leading to congenital hydroceles and herniæ cannot be understood. Attached to the lower pole of the testis is a fibromuscular cord named the gubernaculum, which ends on the posterior surface of the anterior abdominal wall at the site of the future internal abdominal ring. At about the sixth month the testis has reached this point. The inguinal canal is then formed by the

gubernaculum, which penetrates the muscles, taking with it a funnel-shaped diverticulum of peritoneum—the processus vaginalis, this lying anteriorly. The trail for the testicular descent is thus blazed by the gubernaculum and processus. The former now gains attachment to the bottom of the scrotum, the external ring and the pubic crest. The descent through the canal occurs during the seventh and eighth months, and by birth the testis should be in the scrotum. As it penetrates the abdominal wall it takes with it a covering derived from each layer. Later the processus vaginalis is shut off from the peritoneum by closure at the internal ring and finally atrophies and disappears, except around the testis and epididymis, where it forms the tunica vaginalis. Anomalies of this closure constitute the varieties of inguinal hernia and hydrocele (Fig. 385).

### ERRORS IN DEVELOPMENT

*Polyorchidism* implies the presence of more than two testes. The number of proved cases is less than ten. *Anorchidism*, partial or complete, is also a very rare condition. *Synorchidism*, or fusion between two testes, has been recorded on two occasions.

### THE IMPERFECTLY DESCENDED TESTIS

The testis may be arrested at any point in its descent, or having passed the external abdominal ring it may come to rest elsewhere than in the scrotum. This latter position is more correctly designated “Ectopia Testis.” During the first six months of life incomplete descent can hardly be termed pathological, especially when it is remembered that the cremaster muscle is then so active that the testis can be withdrawn into the inguinal canal. No importance need be attached to this condition during the first year. In definite cases the incidence is 1 : 1000, each side being equally affected.

The positions occupied by the testis in imperfect descent are :—

1. Abdominal or retained testis, *i.e.*, somewhere on the posterior abdominal wall,
2. Inguinal, lying at any point in the canal,
3. Just outside the external ring (the commonest of all),
4. Interstitial, *i.e.*, related to the abdominal wall either between the peritoneum and the muscles or between or in front of the muscles,
5. Pubic, on the symphysis pubis,
6. Femoral, in Scarpa's triangle.
7. Perineal,
8. Transverse, *i.e.*, both testes in the same canal.

### EFFECT ON THE TESTIS

**A. On its Function.**—Spermatogenesis is usually, but not invariably, absent ; but the interstitial cells are said to be present. The author has observed many cases in which a delayed puberty has accompanied imperfect descent.

**B. On its Vulnerability.**—Imperfectly descended testes are more liable to injury and torsion. There is no evidence that they are more susceptible to infection and new growth. A retained testis is a source of danger in that it is less accessible to diagnosis, and hence liable to unrecognised complications.

A congenital inguinal hernia invariably accompanies varieties (2) and (3) above, and often coexists with types (4) to (7).

**Treatment.**—Some diversity of opinion exists as to the most suitable age for operation. Much research work has been and is being done on the administration of extracts of the ductless glands, and considerable progress has been made in the technique of producing the most suitable compounds for human use. There is little justification for surgical treatment until the age of puberty, for up to this time some prospect of the testes descending normally into the scrotum



FIG. 386

Author's method of fixation after operation for imperfectly descended testis.

remains, and the chances of this are greatly increased by the use of "Progynon," "Pregnyl" or some other similar endocrine extract. When the boy has reached the age of 15 years and has not improved after endocrine therapy, operation must be advised.

Three operations are possible, orchidopexy, orchidectomy and abdominal replacement. The last should never be practised, and removal of the testis is rarely necessary. Orchidopexy, which consists in fixation of the testis to the scrotum, should be attempted. The shortness of the cord is the one factor leading to difficulty. It is, therefore, dissected up to the internal ring and each layer of its coverings divided. Traction on the testis will usually suffice to bring it well down into the scrotum. There are many methods of fixing it in its new position, but it is the author's practice to sew the testis to the floor of the scrotum and to leave the sutures sufficiently long to reach the level of the knee. A small metal plate carrying a brass hook is incorporated in a plaster-of-Paris bandage around the thigh immediately above the knee, and to this hook the ends of the suture are tied (Fig. 386). In this way the testis is prevented from retracting up into the inguinal canal for ten days, after which time it will remain in place without difficulty. In those rare cases in which the testis cannot be brought into the scrotum without undue tension, then orchidectomy is preferable to abdominal replacement.

## TORSION OF THE TESTIS

Torsion may occur at any age, but is most frequent in adolescence. Two distinct varieties occur.

**Torsion of the Cord** occurs in association with an imperfect descent of the testis which lies just outside the external abdominal ring and held rather firmly against it. Some minor strain or injury may precipitate the attack, but cases have occurred during sleep.

The cord below the twist is congested and the vessels thrombosed; the testis is intensely congested, develops interstitial hæmorrhages, and resembles blood clot in appearance. Gangrene and infection are likely to follow in untreated cases (Fig. 387).

*Symptoms and Diagnosis.*—There is a sudden onset of severe pain in the testis, which becomes swollen and tender. An attack of vomiting is common. When torsion occurs in a testis lying at the external ring, the condition is likely to be mistaken for a strangulated inguinal hernia.

**Intravaginal Torsion** of the testis is less common and occurs in association with a normally placed organ, but a long mesorchium is present and polar rotation has not taken place (Fig. 387). Many patients give a history of strain due to lifting heavy weights and there is never direct trauma to the testis. Its condition is similar to that described above, but the cord and tunica vaginalis are unaffected.

*Symptoms and Diagnosis.*—Pain is of slow and gradual onset, usually wakening the patient during the night following the strain. Within forty-eight hours pain, swelling and tenderness are well marked, but the picture is not so severe as in torsion of the cord, and as a result acute epididymo-orchitis is likely to be diagnosed.

*Treatment* of both varieties is immediate operation at which the twist is undone and the testis replaced if viable, or removed if too severely damaged to recover.



FIG. 387

A specimen illustrating the effects of strangulation of the testis due to torsion of the cord.

## INJURIES TO THE TESTIS

The testes are by their position peculiarly vulnerable, yet their extensive mobility usually protects them from serious damage. Subcutaneous trauma is usually seen as the result of games injuries, or rarely falls from astride beams, etc. Two distinct lesions result, traumatic

orchitis and hæmatocele (p. 807). The one symptom is severe pain, the patient having a distressing feeling that he may faint at any moment. The testis swells up rapidly and the tunica vaginalis fills with blood. Treatment for the hæmatocele is discussed later, but the orchitis needs rest, evaporating lotions, suspensory bandaging and morphia in the early stages. The most scrupulous attention to cleanliness of the parts must be observed to prevent infection penetrating the skin.

### INFECTIONS OF THE TESTIS

These may be classified as follows :—

- |              |   |   |
|--------------|---|---|
|              | ( | 1. Pyogenic from direct implantation,                         |
|              |   | 2. Pyogenic from urethra or prostate,                         |
| A. Acute     |   | 3. Pyæmic,  |
|              |   | 4. Metastatic, in certain specific infectious fevers,         |
|              |   | 5. Gonococcal,  |
|              |   | 6. <i>Bacillus coli</i> .                                     |
|              | ) | 1. Following acute cases, <i>i.e.</i> , imperfect resolution, |
| B. Chronic . |   | 2. Primary chronic pyogenic,                                  |
|              |   | 3. Tuberculous,   |
|              |   | 4. Syphilitic.  |

*Method of Infection.*—This question is one of great controversy. Firstly, there is the localisation of certain infections to the epididymis, and of others to the body of the testis. This is an example of that selective propensity of bacteria which is seen in other parts of the body, and for which there is no satisfactory explanation. Secondly, there is the problem of the route taken by the infecting organisms—either the blood stream, lymphatics or vas deferens.

1. Hæmatogenous. Certain infections provide indisputable evidence that blood-stream infection does occur, *e.g.*, the orchitis of mumps and of syphilis.

2. Genito-urinary lymphatic invasion. The interrelationship between the genital and urinary systems in the male is very intimate. The prostatic urethra is the common meeting ground of all the ducts of both systems. Into it enter the urethra, the ejaculatory ducts, the multiple prostatic ducts, and the base of the bladder with the ureters. In that small area, a limited infective process is strategically placed to attack any or all of these ducts, and similarly, if an infection should reach this area down one duct the remainder are in peril of invasion. The prostate, seminal vesicles and both testes are particularly liable to infect each other, and these infective lesions assume an importance out of all proportion to their local significance, because of the grave possibility of the process spreading to the bladder base and so reaching both kidneys. If the surgery of genito-urinary infections is to be properly appreciated, this interrelationship must be continuously borne in mind. In this type of spread the transmission is not up the lumen of the vas but along its lymphatics. The infection is carried by the vasal and perivasal lymphatics from the prostate or vesicles to the testis. Spread along the lumen of the vas by retrograde peristalsis is not possible.



## ACUTE ORCHITIS

This may be seen as the result of penetrating wounds or of injury without breach of scrotal skin. It may occur very rarely in pyæmia from osteomyelitis and other acute infective conditions. It may also complicate gout, the attacks corresponding to the exacerbations in the joints.

It is a complication of certain specific fevers, *e.g.*, typhoid, small-pox, glanders, scarlet fever, influenza, malaria and most commonly mumps. About the tenth day of the parotitis, a swelling of one or both testes occurs with considerable pain. The gland is enlarged, hard and tender and a small flabby hydrocele is present. Suppuration never occurs and the swelling subsides in seven to ten days. Atrophy of the tubules may occur and in bilateral cases sterility is likely to result. The treatment consists in rest in bed, local applications of lead and opium lotion and a suspensory bandage.

## ACUTE EPIDIDYMO-ORCHITIS

This may be of pyogenic or gonococcal origin.

*Pathology.*—The infection, primarily in the urethra or prostate, reaches the epididymis by the lymphatic route and settles first in the globus minor, from which the whole epididymis is affected, and spread into the testis occurs along the efferent ducts.

**A. Pyogenic Cases** follow infection behind a stricture, false passage, periurethral and prostatic abscesses. It is seen also as a complication of prostatectomy. Suppuration usually results and the testis will be destroyed. Early orchidectomy is the correct treatment.

**B. Gonococcal Epididymo-orchitis** occurs during the second or third week of an acute attack of gonorrhœa, or more rarely during the chronic stage. The onset is abrupt, slight pain being felt in the globus minor, but this rapidly spreads to the entire gland and becomes severe. The swelling attacks first the epididymis, then involves the body of the testis, and finally, the scrotal skin becomes red, glazed and œdematous. A small hydrocele may form. The swelling is exquisitely tender and the patient suffers from fever and malaise. In the early stages examination will reveal no changes in the prostate or vesicles. With the onset of the testicular lesion the urethral discharge ceases, and is not re-established until the swelling of the testis has subsided. The diagnosis is rarely in doubt.

*Treatment.*—All local treatment to the urethra must cease immediately. The patient is put to bed with local applications of lead and opium lotion or glycerin and belladonna, and the scrotum is supported. Sulphanilamide therapy has superseded all other forms of general treatment. When the swelling subsides a small fibrous nodule is apt to remain in the globus minor, and should this occur the injection of contramine may be repeated. When the urethral discharge reappears its treatment must be resumed. Fibrosis in the epididymis may result, and when bilateral, sterility may follow.

### TUBERCULOUS EPIDIDYMITIS

This disease occurs at any age, but the third decade is the commonest period. Each side seems equally liable to infection. The method of infection is disputed, some observers declaring that every case is secondary to a focus in the prostate or vesicles, others maintaining that the majority are blood borne. The subacute form is always hæmatogenous, but in chronic cases 60 per cent. are lymphatic in origin. The original tuberculous lesion is in the lungs or lymph glands.

Tuberculosis of the epididymis assumes an importance out of all proportion to its local effects, owing to the intimate interrelationship between testes, prostate, bladder and kidneys. A lesion in one epididymis may reach both kidneys and effect a fatal result. The pathological changes are:—



FIG. 388

Subacute tuberculous epididymitis, the caseous abscess having erupted through the lower pole into the dartos tissues beneath the skin. The vas is thickened.

**A. In the Epididymis.**—The histology differs in no way from that of tuberculosis elsewhere. The first nodule starts in the globus major or minor according to the route of infection, and spreads throughout the whole epididymis. This becomes enlarged, thickened and studded with tubercles, which eventually coalesce to form large caseous areas.

**B. In the Testis.**—The infection later reaches the testis by the efferent ducts. This takes place early and extensively in the subacute type, but very late in the chronic type. A small hydrocele may be present.

**C. In the Cord.**—The vas becomes thickened, enlarged and hard, the rest of the cord being unaffected.

**D. The Seminal Vesicles** become enlarged and nodular and perivesiculitis obliterates the transverse groove between them and the prostate

**E. The Prostate** becomes slightly enlarged on the affected side, irregular, hard and nodular.

In the primary epididymal cases the changes in the vas and prostate follow the testicular lesion, whereas in those patients in whom the lesion is secondary to disease in the upper genito-urinary tract, they are present before the epididymis enlarges. In the primary cases it may be possible to feel the uppermost limit of the spread in the vas.

**Clinical Varieties.**—Two distinct varieties occur, the subacute and the chronic.

**Subacute Tuberculous Epididymitis** (Fig. 388) is of sudden onset in a man usually known to have pulmonary or other tuberculosis. In a small percentage of patients there will have been a transient mucopurulent discharge from the urethra at some time during the six

weeks preceding the testicular swelling. The pain is moderately severe and the epididymis becomes swollen. The scrotal skin soon becomes adherent and then red and œdematous. Within six weeks an abscess is obvious and may be threatening to break through the skin, and this will certainly happen if early treatment is not carried out. The vas may be thickened for a variable distance, but no signs of prostatic or vesicular invasion can be felt. On removal the testis is seen to be involved.

**Chronic Tuberculous Epididymitis** (Fig. 389) is of such insidious onset that the patient finds it difficult to say when the disease started. Slight aching pain may have been the first symptom, or a nodule been felt when bathing. This nodule may be in the globus major or minor, and invasion of the whole gland will take months. Fixation of the skin is a late sign. The vas is thickened and nodular but the rest of the cord is normal. Rectal examination reveals the typical signs of prostatic and vesicular invasion.

*Treatment.*—In the subacute type, orchidectomy must be performed at once, because there is a good prospect that the testicular lesion is the only genito-urinary one, and further, the abscess will invariably rupture through skin if left, and a persistent sinus remains.

In the chronic type the treatment depends on the extent of the spread. There are certain contraindications to operation, viz. : (a) bilateral epididymitis ; (b) advanced tuberculosis of the prostate ; (c) tuberculosis of the urinary tract ; (d) active and advanced tuberculosis elsewhere. If operation is decided upon, there are varied procedures available. Epididymectomy is directed to the removal of the epididymis, leaving the body of the testis and its blood supply intact, but in practice the blood supply is usually damaged and the testis atrophies, while there is no guarantee that a few early tubercles have not already appeared in the testis. Orchidectomy, with removal of the cord as far as the internal ring, combined with a digital avulsion of the vas as low down in the pelvis as possible, is a more satisfactory operation. Some surgeons do a perineal removal of the seminal vesicles and prostate also if these are obviously diseased. Of the three methods orchidectomy with vasectomy is to be preferred.

In operable cases the full rigour of prolonged convalescent treatment must be enforced on the same lines as laid down for operable renal tuberculosis. Even in inoperable cases local measures will be needed in addition to full sanatorium régime if a sinus is established in the scrotum.

*Prognosis* in the subacute variety is said to be poor. If the existence of this type were more universally recognised, the diagnosis would be made earlier and immediate orchidectomy would produce greatly improved results. In the chronic cases the prognosis depends on the extent of the genito-urinary spread.

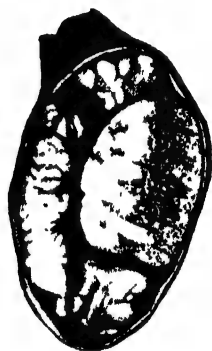


Fig. 389

Chronic tuberculous epididymitis. The epididymis is enlarged throughout and caseous tubercles are seen. The vas is thickened.

**CHRONIC NON-TUBERCULOUS EPIDIDYMITIS**

A considerable number of cases occur in which the clinical picture is distinguished from tuberculosis only with great difficulty. The majority are due to *B. coli* which will be found in the urine in the earlier stages. Mistakes will often be made, an orchidectomy performed and the error exposed by the pathologist. They will be reduced in number if a general genito-urinary investigation is undertaken in every testicular lesion.

**SYPHILITIC ORCHITIS**

The testis may be affected as follows :—

Congenital	.	.	.	.	Diffuse interstitial orchitis.
Acquired	{	Secondary stage	.	.	Subacute epididymo-orchitis.
		Tertiary stage	.	.	{ Localised gumma.
					{ Diffuse interstitial orchitis.

Subacute epididymo-orchitis in the secondary stage is always bilateral and accompanied by hydrocele. It is usually transient, but may recur if treatment is not adequate.



FIG 390

Two gummata in the body of the testis with a hydrocele of moderate size.

**Gumma of the Testis** is not very common (Fig. 390). It presents a painless swelling of the body of gradual onset, often masked by a hydrocele. The lesion is confined to the testis, the epididymis remaining unaffected in most cases. If untreated the gumma will spread to the scrotal skin, which becomes involved with the formation of the typical ulcer and wash-leather slough, followed by a hernia of the testis. Modern methods of diagnosis should make these latter complications impossible. Gummata are occasionally mistaken for growths, but an exact history, careful examination and a Wassermann test should make diagnosis easy.

**Diffuse Interstitial Orchitis** is more common, always bilateral and accompanied by a hydrocele. In the early stages the testes show a rounded, firm, painless and heavy enlargement in which sensation is abolished. Later, fibrosis occurs and they become small, hard and heavy, out of all proportion to their size.

In all types the treatment is that of the causative syphilis.

**GROWTHS OF THE TESTIS**

These may be classified :—

Benign	.	.	.	.	.	Adenoma.
Malignant	{	.	.	.	.	Carcinoma.
		.	.	.	.	Teratoma.
		.	.	.	.	Sarcoma.

Benign growths are practically unknown ; one case of an adenoma has been reported and the many so-called benign connective tissue tumours are examples of teratomata, in which one tissue predominates.

### MALIGNANT GROWTHS

*Etiology.*—Testicular tumours are not common. Teratomata may be seen in childhood, adolescence and up to 35 years of age, while carcinomata occur between 35 and 55, somewhat earlier than the usual age. Trauma seems to play a definite rôle as a causative factor.

*Naked-eye Appearance.* — **Carcinomata** show a wide diversity of appearance ; of the 19 specimens in the St Mary's Hospital museum no two are alike. They tend to maintain the general shape of the testis and to remain within an intact tunica albuginea. Many grow so rapidly that extensive necrosis of the tumour occurs.

**Teratomata** fall into three groups :—

A. The single cystic type or "dermoid cyst," in which the testis is replaced by a cyst with an intracystic growth carrying skin, hair and teeth, etc. (Fig. 391).

B. The polycystic type or "fibrocystic disease," with many small cysts scattered throughout a background of varying structure (Fig. 392).

C. The solid type, which varies considerably in colour and appearance.

All tend to remain within an intact tunica albuginea, and many show a flattened strip of testicular tissue spread over the surface of the growth. The spermatic cord becomes thickened owing to vascular hypertrophy, but the vas is unchanged.

**Sarcomata** present an appearance similar to the fibroid in the uterus.

*Microscopical Detail.*—Carcinomata are of two varieties. The Seminoma of Chevassu or the Spermatocytoma of other authors is a polygonal-celled carcinoma simplex with large cells resembling the more deeply placed ones of the seminiferous tubules. The second

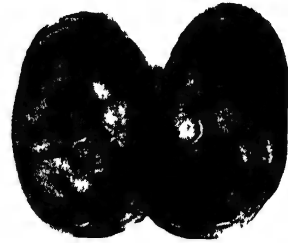


FIG. 391

A testis replaced by a cystic teratoma with an intracystic growth containing skin, hair and bone. An example of the rare "dermoid cyst" of the testis.



FIG. 392

The typical polycystic or "fibrocystic" teratoma of the testis.

type is the more embryonic papillary adenocarcinoma with smaller cells arranged in an alveolar formation showing papillary tufts.

Teratomata contain tissues representing the derivatives of all the three primary germinal layers. Only rarely in testicular teratomata do fully formed adult structures occur. The single cystic dermoid is extremely rare, and in it hair, teeth and skin are found, but in the others the characteristic feature is the indiscriminate mixture of tissues without any attempt at regular arrangement. Many show also carcinomatous and sarcomatous changes, and about 125 cases of chorionic carcinoma are recorded. These tumours are malignant, apart from any such degeneration, and metastases in the lungs and glands show the same admixture of cartilage, nerve, muscle and cysts as the primary growth. Carcinomatous change is apt to destroy the original teratoma, and if left long enough may predominate to such an extent that no trace of recognisable tissue is to be seen.

Sarcoma is of the spindle or round-celled variety.

*Nature of these Tumours.*—The old controversy raised by Ewing's contention that all these tumours were primarily teratomatous is dead, since the discovery that, whereas teratomata give the Aschheim-Zondek reaction, carcinomata do not.

*Symptoms.*—Patients complain of a swelling of insidious onset, possibly following a blow in the recent past. There may be slight dragging pain along the cord. Hæmatocele is not often seen unless the swelling has been explored by needling. Involvement of the scrotal skin—"fungus testis"—can only be seen in cases of neglect. The vas deferens is not affected, but the cord is thickened from hypertrophy of the vessels. The lymph glands along the aorta and vena cava are involved early in the disease.

*Diagnosis.*—In early cases tuberculous epididymitis may be suggested, and in others a gumma may prove misleading. In teratoma prolan is present in the urine and an Aschheim-Zondek test will give a positive reaction; to this extent, therefore, this test is of assistance. It must be clearly understood that a negative result does not exclude malignant disease.

*Treatment.*—The testis with the cord as far as the internal abdominal ring should be removed. The radical removal of the glands is a procedure which has little support in this country. If enlarged glands are palpable the mass should be exposed through the loin and radon seeds implanted, and in every case deep X-ray therapy of the lymphatic field should be carried out.

*The Prognosis* is always grave, these tumours being amongst the most malignant known. Recent statistics suggest a slightly more hopeful attitude to these neoplasms.

Growths of the epididymis are exceedingly rare. A few benign tumours are on record and carcinoma does occur.

**Diagnosis of Testicular Swellings.**—In the following table the clinical features of the important testicular diseases are set out.

Careful attention to every clinical sign should give a correct diagnosis in a high percentage of cases. Small nodules, however, may give rise to difficulty, and in view of the terrible mortality of testicular new

growths, no expectant policy can be tolerated in doubtful swellings. In the presence of small nodules in testis or epididymis and in the absence of any positive findings in the history, symptoms or signs, the

	EPIDIDYMO-ORCHITIS.		TUBERCLE.	SYPHILIS.	NEW GROWTH.
	ACUTE.	CHRONIC.			
Body . . .	Affected.	Not affected.	Not affected.	Affected.	Affected.
Epididymis .	Affected.	Affected.	Affected.	Not affected.	Not affected.
Tunica vaginalis (hydrocele)	Sometimes.	Occasionally.	Rarely.	Always.	Never.
Skin . . .	Red, œdematous, glazed.	Not affected.	Sinus.	Hernia testis (in late cases).	Rarely affected (fungus testis).
Vas deferens .	Not affected early. Thickened late.	Thickened.	Thickened.	Not affected.	Not affected.
Spermatic cord	Some œdema.	Not affected.	Not affected.	Thickened.	Thickened.
Seminal vesicles	Affected later, but not in early stage.	Affected.	Always affected sooner or later.	Not affected.	Not affected.
Prostate	Affected later, but not in early stage.	Affected.	Always affected sooner or later.	Not affected.	Not affected.
Testicular sensation	Retained.	Retained.	Retained.	Lost early.	Retained till quite late.
Testicular weight	Unchanged.	Unchanged.	Unchanged.	Much increased.	Increased.
Glands . . .	Not affected.	Not affected.	Not affected.	Not affected.	Early enlarged.
Other clinical signs	History of gonorrhœa. Cessation of discharge.	Previous history. In rare cases, nothing.	Evidence of history of tuberculosis elsewhere.	History and other stigmata of syphilis.	Nothing. Possibly history of injury.

surgeon's duty is to look and see rather than to wait and see. In this way only can the present high mortality of testicular growths be reduced.

## THE TUNICA VAGINALIS AND SPERMATIC CORD

### HYDROCELE

A hydrocele is a collection of fluid in the tunica vaginalis, in the persistent processus vaginalis, or a cystic swelling in the testis, epididymis and cord. They may be classified thus :—

#### A. Congenital (Fig. 393).

1. Of the tunica vaginalis . . . . . Vaginal.
2. Of the tunica and processus vaginalis . . . . . {
  - Congenital,
  - Infantile,
  - Hydrocele with an imperfectly descended testis.

*A. Congenital—continued.*

- |   |  |
|---|--|
| 3. Of the processus alone                               | Encysted hydrocele of cord.  |
| 4. Of the testis  | } Spermatoceles.   |
| 5. Of the epididymis                                    |  |
| 6. Dilatation of developmental remains                  | { Hydatids of Morgagni,<br>Organ of Giraldes<br>Vas aberrans of Halle. |
| 7. Diffuse hydrocele of the cord is a lymphangiectasis. |  |

*B. Acquired.*

- |                |                                    |
|----------------|------------------------------------|
| 8. Idiopathic. | 10. Infective { Acute,<br>Chronic. |
| 9. Traumatic.  |                                    |

## CONGENITAL HYDROCELE

This is comparatively common (Fig. 394). The condition may be bilateral, and infants are brought for advice in the first three months. The **congenital** variety with the persistent processus vaginalis in free continuity with the peritoneal cavity may not become noticeable until the child begins to walk. It will then be seen at the end of the day and has disappeared by the morning. This variety is likely to have a congenital hernia coexistent with it, and the treatment is directed principally to that. The **vaginal and infantile** varieties remain constant and have no hernia. These should be tapped and injections of sodium morrhuate seem to give good results.

**Encysted Hydrocele of the Cord.**—This is due to the closure of the processus vaginalis both above and below, the central part remaining patent. The resulting cystic swelling does not necessarily become evident in infancy and may be seen in the second or third decade. Its treatment is simple dissection from the cord.

**Spermatoceles** are cystic swellings in the epididymis and testis containing a whitish opalescent fluid in which will be found spermatozoa; these may be alive or dead and in varying stages of development. These cysts may be congenital in origin, occurring in children and young adults, or acquired from fibrosis around the efferent ducts leading to a retention cyst, or from rupture of the ducts leading to an extravasation cyst. The majority occur in the globus major and form a well-defined spherical swelling attached to the upper pole of the testis, which latter is normal in shape and consistence. Spermatoceles produce symptomless slowly enlarging swellings; they are occasionally bilateral, in which case the patient may complain of impotence. They are apt to cause atrophy of the testis after a long period and should therefore be excised. In elderly men whose sexual life has waned, tapping at regular intervals will suffice to keep them comfortable.

## ACQUIRED HYDROCELE

This may be primary, due to disease of the tunica vaginalis alone or secondary to disease of the testis, epididymis or distant structures.



**Acute Hydrocele** as a primary condition is very rarely seen. It is usually secondary to such testicular lesions as acute gonococcal epididymo-orchitis, in which the hydrocele is apt to be overlooked owing to the severity of the testicular lesion. The hydrocele subsides with the primary condition. Rarely suppuration takes place and the tunica vaginalis will have to be opened and drained.

**Chronic Hydrocele** will result from the imperfect resolution of an acute attack or as a complication of chronic testicular disease. It is present in many patients with syphilitic orchitis and occasionally in chronic tuberculous epididymitis. The treatment is directed to the cause.

**Idiopathic Hydrocele** is the commonest variety of all. It occurs without apparent cause in men over 35 years of age. The fluid is straw-coloured and may be opalescent from the presence of cholesterin crystals. Its specific gravity is between 1022 and 1030, it contains 6 per cent. of albumen and clots solidly on boiling, but otherwise remains fluid indefinitely. The tunica vaginalis is distended and thin (Fig. 395), but fibrosis may occur, especially after repeated tapplings. The testis is rarely affected, except in old long-standing cases when atrophy may result from pressure. In very large hydroceles, especially when bilateral, the penis may be withdrawn into the distended scrotal skin and its position marked only by a puckered dimple.

*Clinical Signs.*—In small hydroceles the testis is surrounded by a lax fluid swelling and is capable of exact definition. In the large ones the tunica vaginalis may be so tensely distended that the testis cannot be identified (Fig. 396),

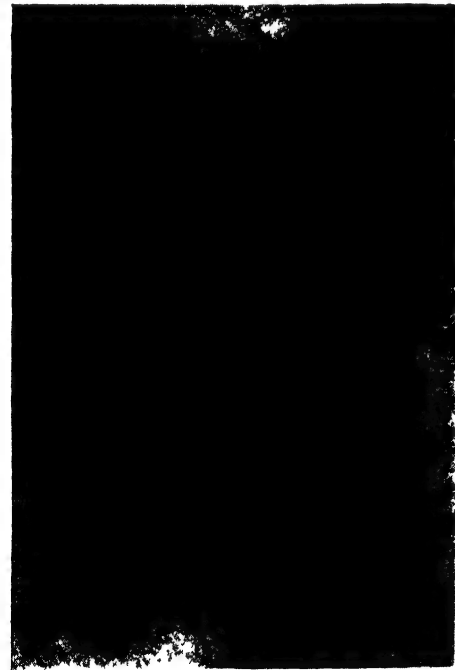


FIG. 394

Bilateral congenital vaginal hydroceles in a boy of 16 years.

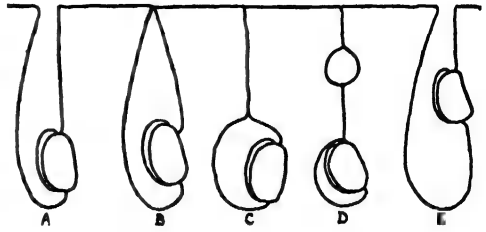


FIG. 393

A diagram illustrating the varieties of congenital hydrocele.

A, Congenital; B, Infantile; C, Vaginal; D, Encysted hydrocele of the cord; and E, that associated with an imperfectly descended testis. The straight line at the top represents the peritoneum.

though pressure from behind may elicit testicular sensation. However large it is, its upper limit can be recognised and above it a normal spermatic cord identified passing on into the inguinal canal. If a light

is placed behind the scrotum, a pink glow is transmitted through the hydrocele, a dark shadow marking the position of the testis.

The *differential diagnosis* rests between hydrocele, inguinal hernia, hæmatocele, spermatocele and solid enlargements of the testis and epididymis. If the hernia is reducible no difficulty arises; if irreducible its impulse on coughing, absence of translucency and its prolongation into the inguinal canal should serve to distinguish the two conditions. A hæmatocele corresponds in shape and position, but is heavier, less elastic and not translucent. In other conditions the definition of the testis and epididymis in relation to the swelling should serve to establish the diagnosis.

*Treatment.*—In nearly every case the hydrocele should be tapped when first seen in order to allow a complete examination of the testis and epididymis. In secondary hydroceles tapping may be needed as a palliative measure, while the exciting cause is being treated.

In idiopathic hydroceles radical cure should always be advised except in old debilitated subjects. Repeated tapping is not otherwise desirable. The technique of tapping demands a preliminary identification of the position of the testis by transillumination. The swelling is then grasped in the hand in such a way that the testis lies in the palm, while the thumb and index finger grip the apex of the sac to steady it and prevent it sliding upwards towards the inguinal canal. The thumb also holds the penis aside. Pressure makes the skin taut and an area free of veins on the upper and outer surface is cleansed with surgical spirit. An exploring needle (No. 10 or 12) mounted on a large syringe is passed into



FIG. 395

An idiopathic acquired hydrocele showing the splaying out of the epididymis and the stretching of the digital fossa.

the sac in an upward direction, and the fluid is aspirated until the sac is empty. This method is preferable to the use of a trocar and cannula, as being neater, cleaner and less painful. The old methods of injecting tincture of iodine or carbolic acid cannot be too strongly condemned, but injection with sodium morrhuate has a limited field of usefulness in small congenital hydroceles.

The radical operation consists in exposing the testis by a short incision stretching from the external ring towards the scrotum. The distended tunica vaginalis is made to present in the wound and is emptied by a trocar and cannula, after which the testis can be brought out of the scrotum. The parietal layer of the tunica vaginalis is removed. The cut edge will be underrun by a continuous

interlocking stitch to prevent oozing. Every bleeding point must be secured and a small drain brought out through a stab wound in the scrotum before the testis is replaced. These precautions must never be omitted as the occurrence of a scrotal hæmatoma is of grave significance owing to the danger of its becoming infected. The operation of incising the tunica vaginalis, turning it inside out and fixing it around the cord is not to be recommended.

### HÆMATOCELE

This is a collection of blood in the tunica vaginalis. It is caused by injury either from direct blows, *e.g.*, kicks, cricket balls, etc., or from surgical trauma in piercing a vein while tapping a hydrocele, or in exploratory puncture when a needle is thrust into a growth of the testis. It does not occur as a complication of growth except after tapping or when the growth has penetrated the tunica albuginea and invaded the tunica vaginalis. It is rarely seen in hæmophilia, leukæmia and scurvy. The history of injury, the shape and position of the non-translucent swelling lead to a sure diagnosis. In those cases without injury a blood count should be done and the testis examined for the presence of a growth.



FIG. 396

A large right-sided idiopathic hydrocele in a middle-aged man. The retraction of the penis is well shown.

The *treatment* consists in rest in bed and firm strapping of the scrotum. It must not be tapped during the first forty-eight hours for fear of the bleeding recurring, but this should always be done on the fourth day, and the scrotum again firmly strapped. This routine tapping will save many patients a tedious convalescence. If the swelling has not absorbed within a fortnight, no further time must be lost and the tunica vaginalis should be opened and the clot removed. In later cases the radical cure for a hydrocele should be practised; and in still later cases, with great thickening of the tunica and atrophy of the testis, an orchidectomy will be needed to relieve pain. In non-traumatic cases in the absence of any blood disease, the testis should be exposed and searched for any sign of growth.

### VARICOCELE

This is a varicose condition of the veins of the pampiniform plexus. It is usually seen in the left spermatic cord of young men. Various conditions are suggested as being contributory factors, none of which can be adequately supported. The left spermatic vein

enters the left renal vein at a right angle, and so the flow of blood in the spermatic vein is said to be impeded; again, the drag of a loaded sigmoid colon in habitually constipated youths is said to compress the vein at the pelvic brim. It does occur, though rarely, as a result of malignant disease of the kidneys or other abdominal organs and a varicocele of insidious onset in elderly men should always lead to a searching abdominal investigation. In the majority of cases no demonstrable cause exists.

*Symptoms* are absent until the patient's attention is drawn to the condition by the exaggerated stories of friends as to its effects, or by his rejection as a candidate for the Services or big industrial concerns. He will then complain of a sense of weight, aching pain and anxiety as to the future. The appearance presented by the dilated veins is unmistakable, and their feel has given the condition the name of "a bag of worms."

*Treatment*.—It cannot be too strongly asserted that treatment is rarely necessary. The profession is, however, forced into performing an unnecessary operation owing to the regulations of all the Services, public bodies and big employers that varicocele is to be a cause for rejection of candidates on physical grounds. In such cases the dilated veins are injected with one of the sclerosing solutions used for varicose veins, *e.g.*, quinine and urethane or sodium morrhuate. A suspensory bandage can never be anything but a placebo, and may do harm by centring a young man's attention on his external genital organs. Operative treatment consists in excision of a length of the varicose veins from the cord, leaving two veins to carry on the circulation.

## THE SCROTUM

The skin of the scrotum is liable to any of the skin diseases which occur elsewhere. Special mention needs to be made only of a few conditions.

*INFECTIONS*.—Acute infection is usually due to extravasation of urine (p. 796) or results from acute orchitis, or from an infected scrotal hæmatoma.

Chronic infection will follow the spread of a chronic process in the testis or epididymis, such as tuberculosis or syphilis, or be the result of sebaceous eczema.

Certain specific conditions, primary chancres, molluscum contagiosum and elephantiasis are not uncommon.

*NEW GROWTHS*.—Benign growths occur, such as sebaceous cysts and dermoids in the median raphe, and these need simple excision. Squamous-celled carcinoma of the skin appears to be almost entirely an occupational disease amongst chimney-sweeps, tar and paraffin workers. It may be preceded by an occupational dermatitis, in which develops a scaly indurated patch that later breaks down into a typical squamous-celled ulcer. The glands in the groins will become affected but the growth is usually of low malignancy and of slow spread. Treatment is the excision of the affected area with the glands.

R. M. HANDFIELD-JONES.

## CHAPTER XXXIX

### DISEASES OF THE FEMALE GENITAL ORGANS

**T**HE purpose of this chapter is to draw attention to those gynæcological conditions which are commonly met with in general surgical practice, and in particular to such as may give rise to difficulties in diagnosis. Problems of purely gynæcological interest will therefore not be dealt with here.

*Surgical Anatomy.*—The female genitalia comprise external and internal organs. The external structures are the vulva with the orifices of the vagina and urethra and the glands and ducts of Bartholin, the whole being enclosed by the labia majora and minora. The internal organs are the vagina, cervix and body of the uterus, Fallopian tubes, ovaries and certain vestigial remnants. These structures are supported and held in position by the levatores ani muscles, the broad and round ligaments, the ovarian ligaments and those condensations of cellular tissue in the base of the broad ligament known as the cardinal or transverse ligament of the cervix and the uterosacral ligaments.

### DISEASES OF THE EXTERNAL GENITALIA

#### VULVITIS

**Vulvitis** is an inflammatory condition of the vulva caused by injury, uncleanliness or, more commonly, by discharge from the cervix and infection with the gonococcus or other organism. It is characterised by redness, soreness, difficulty and pain in walking and sitting and pain on micturition from an associated urethritis.

*Treatment* consists in rest in bed, punctuated by sitting in hip baths of warm, mildly alkaline or antiseptic solutions such as dettol for half an hour at a time. If a cervical discharge is present and the parts are not too tender, the vagina should be douched daily. After each treatment the vulva is dried and a mild ointment, *e.g.*, zinc oxide and castor oil is applied. With increased tolerance to manipulations local applications to the affected cervix can be commenced. If a Bartholin's abscess should supervene, it must be incised and drained.

Certain special types of vulvitis may occasionally be seen though, with the exception of diabetic vulvitis, they are all rare. *Gangrenous vulvitis* may occur in the puerperium and in association with severe venereal infection. A similar condition is seen as a complication of the acute specific fevers, *e.g.*, measles, and to these types the term *Noma vulvæ* is applied (p. 177). *Membranous vulvitis* occurs during diphtheria and is recognised by the greyish appearance of the membrane and by

identification of the Klebs-Löffler bacillus. In like manner *Typhoid vulvitis* may occur with or without ulceration in cases of enteric fever, and other still rarer examples are the herpetic and the erysipelatous. *Diabetic vulvitis* is caused by the irritating urine and is quite commonly observed. It can often be recognised by the "raw ham" colour of the vulval skin which may also exhibit excoriations and perhaps a scaly appearance. Any case with some or all of these features must have the urine tested for sugar. Should this be found, the usual investigations and treatment must follow.

### LEUKOPLAKIA

**Leukoplakia** is essentially a disease of the *outside* of the vulva which causes itching (*pruritus vulvæ*). It is in the nature of a chronic inflammation and exhibits superficial proliferation of the skin of the labia majora and perineum so that areas of white thickening are present. In the deeper layers there are hyperæmia and a round-celled infiltration with subsequent fibrosis. Eventually shrinkage of this fibrous tissue leads to the appearance of fissures. Leukoplakia is definitely a precancerous condition.

*Treatment.*—Care must be taken to exclude any possible predisposing cause such as vulvitis or vaginitis. When itching is troublesome in the early stages, calamine lotion may afford some relief and menthol ointment (1 per cent.) is useful. The more severe cases sometimes yield to X-ray treatment, though many authorities consider this dangerous and likely to increase the chances of malignant change. Other patients are benefited by œstrin therapy (see below). If the disease is of long-standing and resistant to treatment and especially if fissures are present, the affected area must be excised for fear of the development of carcinoma.

### KRAUROSIS VULVÆ

**Kraurosis Vulvæ** is an atrophic condition of the superficial epithelium of the vestibule, *i.e.*, of the vulval skin within the labia minora. It is thus to be contrasted with leukoplakia which affects the outside of the vulva and causes itching, whereas kraurosis is a disease of the more delicate and deeply placed skin which causes pain. It must, however, be remembered that itching may also be one of the earliest symptoms of kraurosis. Later when the thin vulval skin shows the classical white "ironed-out" appearance with tiny red spots where the more vascular dermis shows through, pain on touching, on micturition and attempts at coitus becomes the predominant symptom.

This condition is always due to ovarian deficiency either post-menopausal or as the result of surgical or radiological castration. It responds well to therapy by ovarian hormones or the synthetic oestrogens such as stilbœstrol (1 mg. three times a day). It is this response, which occurs also in some cases of leukoplakia, that has been one reason for the revival of the theory that kraurosis and leukoplakia are not two separate conditions but slightly varying

responses to the same pathological process. But it must be remembered that it is only the latter which is liable to lead to carcinoma.

### INFECTIVE GRANULOMA

**Papillomata** occurring in the vulva are frequently due to gonorrhœa (p. 63) or, less commonly, to uncleanness. Patches of sodden thickened skin resembling papillomata are found on closer examination to be sessile and these are likely to be due to syphilis.

### CYSTS

**Cysts** which occur on the labia majora may be due to retention of sebaceous secretion. **Bartholin's cysts** are larger and are due to occlusion of the duct. They appear first as elastic swellings in the posterior part of one or both labia minora, but with increasing size they spread outwards into the labia majora. They should be excised together with the remains of the gland which lies deep to them. A rapidly developing swelling in this position which seems to be a "cyst" one day but is much bigger and more tender within twenty-four hours is a **Bartholin's abscess**, which should at once be incised and drained. Such abscesses are of gonococcal origin only in some 50 per cent. of cases.

Other types of cysts sometimes met with in the vulva will be found to contain tarry or chocolate-sauce fluid. They are now classified as "**endometriomata**" and are believed to be due to the implantation of functioning, *i.e.*, menstruating fragments of endometrium. They should be excised.

### GROWTHS

**Innocent tumours**, which are often pedunculated, are found growing from the labia majora. If causing inconvenience they should be excised, when they will be found to be fibromata or lipomata.

**Carcinoma** is usually squamous-celled, but occasionally an adenocarcinoma arises in Bartholin's gland. The common type occurs on the labia or clitoris, the patient being usually an old woman often in the region of 70 years of age. If the growth is primarily on one labium a contact tumour may develop upon the other lip. Rare cases of squamous-celled carcinoma arise from the region of the urethral orifice. Leukoplakia is known to be a precursor of all these growths which exhibit the typical appearance of such tumours, namely, an ulcer with raised, everted and indurated edges. They are as a rule of rapid growth and spread to the inguinal glands as an early complication.

It should be remembered that when a columnar-celled tumour is excised from the vulva it may be a secondary implant from a similar neoplasm higher up in the genital tract, *e.g.*, the body of the uterus.

*Symptoms* are pain, itching and a blood-stained discharge. As a rule they do not appear until well after the menopause. Examination may prove to be difficult as they are tender.

*Treatment* in old women should be restricted to local excision and



X-ray therapy to both vulva and inguinal regions. In younger and more robust subjects thorough surgical removal should be employed, the affected vulva and the inguinal glands being removed in one piece. Inoperable growths are treated with radium needles, while the groins are irradiated with X-rays.

**Sarcoma** is a rare disease and when it does occur is more likely to be of the melanotic variety. It is treated by wide excision.

### DISEASES OF THE URETHRAL ORIFICE

**Prolapse** of the mucous membrane may occur, either a mere pouting of the mucosa just inside the posterior margin or as a protrusion of the entire circumference. The former must be distinguished from a caruncle, and indeed is often referred to as a "false" caruncle. Complete prolapse is painful both on sitting and walking and the mucosal cuff is liable to strangulation. The prolapsed tissue should be completely excised, the cut end of the urethra being sutured to skin margin.

A **caruncle** is usually an infective granuloma, but rarely is an adenoma arising in the urethral glands. It presents a typical appearance of a miniature cockscomb growing from the posterior margin of the urethra immediately within the meatus. It is very tender, causing pain on micturition and making walking and sitting uncomfortable. It is highly vascular and blood may be seen on the clothing or appear in the urine.

*Treatment.*—The most satisfactory method is to excise or destroy by diathermy the caruncle and that area of skin from which it arises. This operation is not easy as the tissues are friable and difficult to handle. Unless they are completely eradicated caruncles tend to recur, and these recurrences should be examined histologically to exclude a possible malignant change.

### DYSFUNCTIONS OF THE FEMALE GENITAL ORGANS

These conditions are essentially of gynæcological interest, but some description is necessary if only to convey certain warnings of real importance to the general surgeon.

#### DYSMENORRHOEA

Premenstrual pain is always due to pelvic disease and its treatment is that of its cause. Pain during the first twenty-four hours of the period is a troublesome symptom which, known as spasmodic or nulliparous dysmenorrhœa, occurs in young women. If it fails to respond to antispasmodic and analgesic drugs or to hormone therapy, it may demand surgical treatment in the form of dilatation of the cervix and curettage of the uterus. The former needs to be done thoroughly but slowly in order to avoid tearing the internal os. Care must also be taken not to perforate the fundus uteri by a sudden slip of the dilator; should this occur no harm will be done in a clean case, provided



all further manipulations are abandoned at once and no foolish attempt is made to explore the rent or disinfect the uterine cavity.

Of recent years certain cases which have not benefited by any other form of treatment are being dealt with by presacral neurectomy. This operation can only be recommended with caution for its results are by no means always satisfactory. It is true that only the worst cases are considered suitable and therefore the results achieved are among the least favourable patients. Results show approximately a 50 per cent. cure rate; despite the claims of some enthusiasts this represents the true figure. The question arises as to whether an abdominal operation of this sort is justifiable in a young woman for a condition which nearly always disappears when she reaches the age of 30 or as a result of marriage and childbirth.

In other intractable cases the production of a temporary menopause by X-rays or radium is sometimes suggested. This is a matter for the specialist and will not be considered here. Hysterectomy is mentioned only to condemn it as being entirely unjustifiable for dysmenorrhœa.

### STERILITY

Certain minor gynæcological abnormalities (*e.g.*, retroversion, pinhole os) are worthy of treatment in any woman complaining of sterility, but if the patient is apparently normal no operative treatment should be considered until her husband has been tested for the presence of active motile spermatozoa in his semen. This test is most easily carried out by the husband having connection in the morning, using a rubber teat-ended condom which is free from spermicidal chemicals. It is removed immediately after coitus, tied firmly with string at the top, placed in a box surrounded by cotton-wool and brought for examination as soon as possible afterwards. Sixty million sperms per cubic centimetre of semen is the minimum for fertility, and the number of deformed heads and non-motile sperms must not exceed 20 per cent. of the total.

### MENORRHAGIA

**Menorrhagia**—excessive or too frequent menstruation—may be due to :—

1. **PELVIC DISEASE** : Fibroids, salpingitis or displacements.
2. **ENDOCRINE DYSFUNCTION**.—This is not uncommon at puberty and responds to daily intramuscular injections of 2 units of progesterone. Other women may menstruate normally until between 30 and 40 years of age and then have increasing monthly loss. This is no longer regarded as consequent upon "endometritis" but due to some hormonal imbalance. No attempt at a more definite pathological explanation is made. Others are labelled "metropathia hæmorrhagica" if both clinical and histological findings conform to a certain pattern. These patients may be relieved by injections of progesterone or curettage. Some intractable cases, however, demand hysterectomy. The production of an artificial menopause by X-rays or radium at this age is quite indefensible.

Yet other cases of menorrhagia do not appear until the time of the menopause; this used to be attributed to "metritis" or "fibrosis uteri." The fibrosis is now recognised as the normal condition for parous women at this time, and the disturbance is regarded as being due not to a pathological change in the uterus but to endocrine upset.

## INFECTIONS OF THE FALLOPIAN TUBES AND OVARIES

These structures are conveniently described together, since involvement of one leads to a spread of infection to the other and also to the opposite tube and ovary to a greater or lesser degree. Approximately 80 per cent. of such cases are due to gonococci or one of the pyogenic cocci, the remaining 20 per cent. being caused by the tubercle bacillus, pneumococcus or *B. coli*.

### ACUTE SALPINGITIS

**Acute Salpingitis** (acute salpingo-oöphoritis) occurs in young women who have had a recent miscarriage or an attack of gonorrhœa. Such cases appear clinically as a localised pelvic peritonitis, with low abdominal pain and an attack of vomiting which may precede the onset of the pain. The lower abdomen is tender, especially just above the inner half of each inguinal ligament and above the pubes. Lower abdominal rigidity appears later. Temperature tends to be somewhat higher than that seen in acute appendicitis. On vaginal examination there is marked tenderness postero-lateral to the cervix on both sides, a discharge is evident and some uterine bleeding may be demonstrated by its presence upon the examining finger. The tenderness is apt to prevent the recognition of a palpable swelling in the pelvis.

*Differential Diagnosis* from acute appendicitis is the chief difficulty. The points to be noted are: (1) the history of a recent abortion or discharge; (2) position of the tenderness, which is either bilateral or medial both on abdominal and vaginal examination; (3) vomiting is usually marked; (4) the uterine bleeding and rather high temperature; and (5) a purulent or muco-purulent discharge. Urinary infections must be borne in mind and will be distinguished by frequency of micturition and the presence of pus and organisms in the urine. Twisted ovarian cysts and ectopic gestation are not so suggestive of an acute inflammatory condition.

*Treatment.*—If the diagnosis is certain treatment is palliative—heat to the abdomen by hot-water bottles or an electric pad, morphia to relieve pain and to restrain intestinal peristalsis and by the administration of sulphapyridine. As the acute phase passes off absorption of the residual pelvic mass may be assisted by the use of hot vaginal douches and short-wave therapy. If acute appendicitis cannot be excluded a laparotomy must be done. Should the condition prove to

be tubal and the inflammation not too severe, it is wise to close the abdomen without drainage. If the damage is extensive both tubes should be removed, ablation of the more seriously infected one alone being futile since the other must become involved. These cases may lead to dense adhesions in the pelvis and to chronic invalidism, possibly calling for more drastic treatment at a later date.

### SUBACUTE AND CHRONIC SALPINGO-OÖPHORITIS

These may follow an unresolved acute attack or occur primarily as a mild infection. Recurrent attacks of pain and pyrexia punctuate a slight chronic illness characterised by backache or other pelvic pain and by menorrhagia and discharge. A vaginal examination reveals inflammatory swellings behind and to one or both sides of the cervix. The uterus itself will be fixed.

Prolonged palliative treatment must always be given a trial and short-wave therapy is most valuable in this connection. If the trouble persists or if recurrent attacks are frequent and disabling, surgical treatment is required. Its guiding principle must be to remove both tubes and to conserve some healthy ovarian tissue if possible.

Cases of chronic salpingitis in virgins or women with a tuberculous history may be found to exhibit a **tuberculous pyosalpinx** of one or both tubes (Fig. 397).

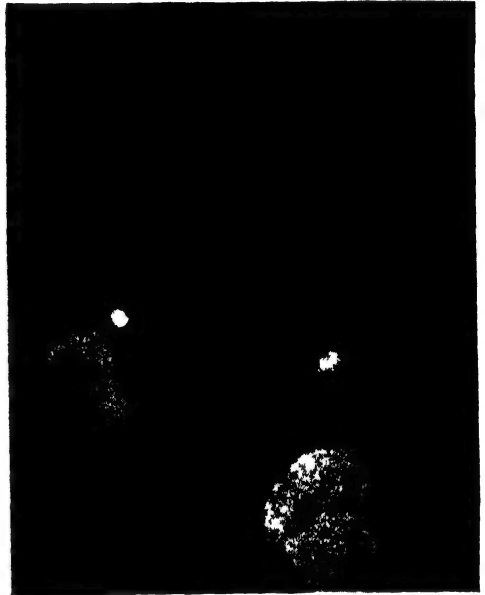


FIG. 397

Bilateral tuberculous pyosalpinx.

One other complication of inflammation is a **pelvic abscess**. This causes downward bulging of the posterior fornix and an intermittent pyrexia. Such an abscess may burst into the rectum or can be opened and drained through the posterior fornix.

### PARAMETRITIS

**Pelvic Cellulitis** occasionally complicates salpingitis, but more commonly is seen during the second week of the puerperium. It causes pelvic pain, pyrexia and a mass which pushes the uterus towards the opposite side. Under palliative treatment it usually resolves, but may result in an abscess which points just above the inguinal ligament. Rarely it comes to the surface in Scarpa's triangle, the buttock or ischiorectal fossa.

## EXTRA-UTERINE GESTATION

Though ovarian pregnancy is known the majority of ectopic gestations are tubal. The common sequence of events is: (1) a mild tubal infection; (2) occlusion of the tubes for a time; (3) a period of sterility; (4) spontaneous reopening of the tube; (5) ascent through this opening of sperms and fertilisation of the ovum; (6) the failure of this latter to find its way into the uterus; and (7) its impaction in a cul-de-sac of the tube.

The growing ovum embedded in the tube is bound, sooner or later, by its trophoblastic erosive action to open into a blood vessel, the resulting hæmorrhage leading to death of the ovum. The severity of the subsequent symptoms is proportionate to the amount of intra-abdominal hæmorrhage.

*Symptoms.*—An ectopic pregnancy may be diagnosed occasionally before rupture by the discovery of a pulsatile swelling in the position of one tube in a woman with symptoms of pregnancy. This, however, is very unlikely, and for all practical purposes it may be accepted that no ectopic gestation is diagnosed until some symptoms have been produced. These can be described in two groups.

*A. Symptoms of Tubal Rupture.*—The history of one missed period is present in the majority of patients, although the erosion of a blood vessel may take place so early that the time for the next period has not arrived. The clinical picture is that of severe internal hæmorrhage. It is heralded by a sudden acute abdominal pain which may be precipitated by straining at stool or other effort, and the patient may faint. It is usual for there to be slight external bleeding following the onset of pain and the patient may assume that the expected period has started. Examination shows all the signs of internal hæmorrhage with some tenderness in the lower abdomen. Blood will be found on the examining finger per vaginam, slight uterine enlargement may be detected and tenderness in the fornices is well marked. A palpable fullness in the pouch of Douglas indicates the presence of clotting blood and pulsation may sometimes be felt.

*B. Symptoms of Slight Tubal Bleeding.*—Not all ectopic gestations end in this dramatic manner. Others cause a slight hæmorrhage which kills the ovum and allows a slight trickle of blood into the pelvic peritoneum. This collects around the tube and clots to form a palpable mass known as a “hæmatocele.” (Clinically some cases present themselves as milder editions of the acute ruptures, but others are difficult to diagnose, although the history is highly suggestive in a young woman with continuous or intermittent uterine bleeding and periodic attacks of pain. If a period has been missed and a mass can be felt on one or other side of the pelvis, the diagnosis is clear.

*Treatment.*—The ruptured gestation with severe internal bleeding demands immediate laparotomy and removal of the affected tube. A transfusion of blood will be needed in many cases, but despite the apparent gravity of this condition the prognosis is good. In the other group of cases it may be possible to incise the tube and remove the mole, but in most patients the tube must be taken away.

## TUMOURS OF THE OVARY AND BROAD LIGAMENT

### OVARIAN CYSTIC TUMOURS

The **Multilocular Pseudomucinous Cystadenoma** is the commonest ovarian cyst, forming about 80 per cent. of the total number (Fig. 398). When small it sometimes gives rise to pain and may be recognised as a spherical elastic tumour to one side of and posterior to the uterus (bilateral masses would suggest inflammatory disease). More usually, these cysts give rise to no symptoms till they are large enough to cause abdominal distension (Fig. 399). They are then to be distinguished from pregnancy by the absence of any menstrual changes, of audible foetal heart sounds or of palpable foetal parts; from fibroids by their more elastic consistence, from ascites because the cysts are

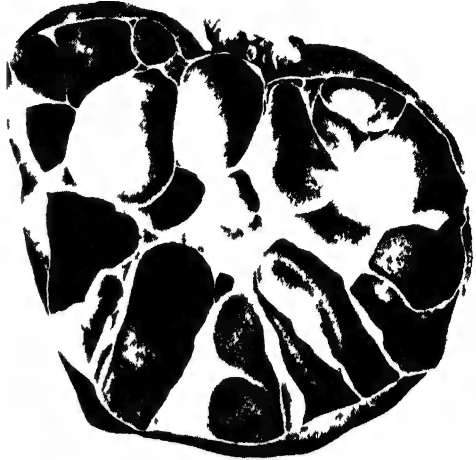


FIG. 398

A multilocular pseudomucinous cyst



FIG. 399

An elderly patient showing the great abdominal distension due to a very large ovarian cyst.

dull to percussion while the flanks are resonant and from a distended bladder by the passage of a catheter.

*Treatment* consists in removal, care being taken to avoid tapping or rupturing the cyst and to ligate securely the very large vessels in the pedicle.

The **Serous Papillary Cystadenoma** will come under the notice of the general surgeon either as a swelling noticed during a rectal examination in a case of ascites or at an exploratory laparotomy in such patients. The condition present comprises bilateral masses in the pelvis postero-lateral to and adherent to the uterus. These tumours will be partly cystic and partly solid, with masses of papillary intracystic growths. Histo-

logically they cover a wide range, some being innocent and others

being active papillary carcinomata, while between these limits there exists a group of cases which appear malignant and have secondary peritoneal deposits, but in which removal of the primary tumours causes death and absorption of the secondaries.

Owing to this difficulty in recognition of the type of growth, these tumours should be removed if they can be freed without undue bleeding. Even the most hopeless looking cases may yield unexpectedly good results. But the reverse is unhappily more common. An apparently innocent ovarian cyst is removed and the appearance of malignant deposits within a year comes as an unpleasant surprise. This points to the necessity for a careful histological examination of every ovarian cyst after its removal.

**Ovarian Dermoid Cysts** are teratomata. They occur at all ages.



FIG. 400

A typical example of an ovarian dermoid cyst showing teeth and hair.

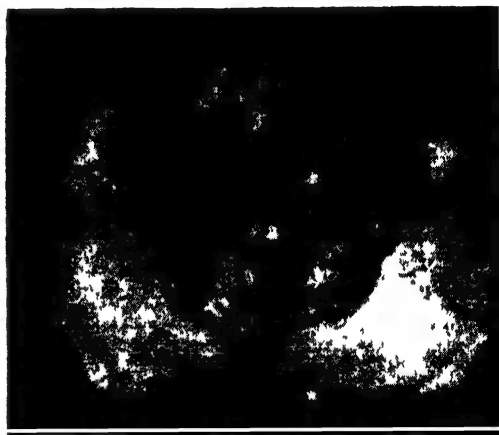


FIG. 401

An ovarian dermoid cyst containing teeth embedded in its wall which has developed a massive squamous-celled carcinoma.

are sometimes bilateral, grow slowly and rarely exceed 4 in. in diameter. The elements which tend to predominate in these tumours are those derived from epiblast, and many are filled with hair and sebaceous material (Figs. 400 and 401). They are discovered in the pelvis during a vaginal examination for such symptoms as pain, frequency or retention of urine.

*Treatment* consists in removal.

**Endometrioma** or tarry cyst of the ovary is believed to be due to fragments of endometrium which succeed in implanting themselves upon the ovary. They then burrow deeper and, by taking part in the process of menstruation, distend the ovary into a cyst containing blood which comes to look like liquid tar or chocolate sauce. They occur in women about 40 years of age and their presence is suggested by menorrhagia and dysmenorrhoea, the periods having been painless previously.

A vaginal examination reveals bilateral masses postero-lateral to the uterus. These are probably diagnosed as salpingo-oöphoritis and their true nature is recognised only at operation, when great

difficulty is encountered in separating the adhesions and the diagnosis is clinched by the typical fluid when the cyst bursts. They should be removed, but any healthy ovarian tissue should be conserved if possible.

**Broad Ligament Cysts.**—Small cysts, about the size of a cherry, arise in the mesosalpinx from remnants of the Wolffian duct and are of no clinical significance.

Large cysts, called **fimbrial cysts**, which may grow to many inches in diameter (Fig. 402), originate in a similar position between the layers of the broad ligament. These are unilocular and have the tube, the ovarian fimbria, the ovary and ovarian ligament spread out over one part of the cyst in the form of an oval ring. They should be removed by shelling them out of their cavity in the broad ligament.

### SOLID OVARIAN TUMOURS

**Fibroma** is usually mistaken for a uterine fibroid, but it is often accompanied by ascites, and is freely movable apart from the uterus. It should be removed.

**Carcinoma.**—Primary carcinoma may be a solid tumour or may be papillary and cystic. It is usually bilateral and is discovered on rectal or vaginal examination in a case of ascites. Secondary carcinoma usually follows a primary growth in the breast, in the colon or in the stomach. It may be discovered as a bilateral mass in the pelvis, accompanied by a typical wedge-shaped growth developing from fragments which have fallen to the bottom of the pouch of Douglas, the process known as Transcœlomic Implantation (see p. 88). A special variety of ovarian carcinoma secondary to a gastric neoplasm is described, and is seen microscopically to contain curious "signet-ring" cells. This type of ovarian new growth is known as the "Kruckenberg Tumour."



FIG. 402

A large fimbrial cyst.

### THE SURGICAL COMPLICATIONS OF OVARIAN TUMOURS.

1. **Rupture** of an ovarian cyst causes few, if any, symptoms, but the signs of internal hæmorrhage may be present if a vessel has been involved in the tear. The rupture may be spontaneous or due to trauma. Surgical treatment is clearly demanded.

2. **Suppuration** is not common. It is due to adhesion to the bowel or appendix and gives the signs and symptoms of localised peritonitis over a tender elastic mass. Immediate operation is indicated.

3. **Torsion of the Pedicle.**—If the twist is sudden and complete, the vessels of the pedicle are occluded and strangulation is followed by necrosis of the tumour and possibly suppuration. More usually



the process is slower, so that the veins are occluded while the arteries still continue to pump blood into the tumour, which becomes tremendously engorged. Some hæmorrhage into the peritoneal cavity is likely. The symptoms are a sudden onset of abdominal pain with an initial attack of vomiting, temporary paralysis of the bowel and possibly the symptoms and signs of internal bleeding. The lower abdominal wall will be rigid and tender but the cyst may be palpable on bimanual examination. The swollen pedicle is sometimes felt *per vaginam*. If the diagnosis is beyond question, the shock should be overcome by heat, fluids and restoratives, and then the cyst removed and the hæmatoma of the pedicle evacuated.

The torsion may, however, be only partial, and untwisting sometimes occurs. The condition is liable to repeated recurrences, and the patient suffers from intermittent attacks of pain, vomiting and abdominal tenderness. The cyst should be removed.

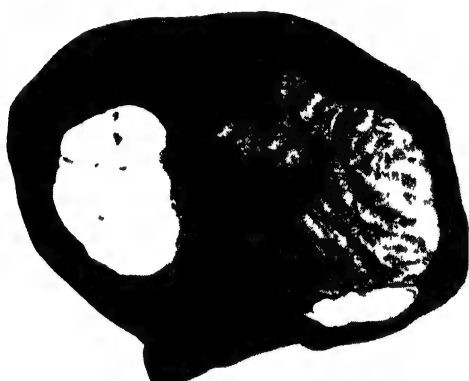


FIG. 403

A cross-section of the uterus showing multiple fibroids, one of which is in a state of red degeneration.

## NEW GROWTHS OF THE UTERUS FIBROIDS

Fibroids occur in the latter half of menstrual life, that is, from 30 to 50 years, in women who have borne few or no children. They are more common in the body of the uterus than in the cervix in the

proportion of 24 to 1. They are usually multiple and vary in size from that of a pin-head to a tumour of great size. They form spherical masses consisting of fibrous and muscular tissues in varying proportions and are classified as "**fibro-myomata**." They start in the uterine muscle and are then referred to as *interstitial*, in which position they may remain; but many move either towards the peritoneum and are then designated as *subserous*, or towards the lumen of the uterus, when they become *submucous* and finally *polypoid*. A further variety spreads between the layers of the broad ligament—*intraligamentary*. The subserous and the submucous may both become pedunculated. Fibroids may undergo certain changes, *e.g.*, red degeneration, atrophy, torsion of the pedicle and infection (Fig. 403). They are said to become sarcomatous, but this is denied, such a tumour probably being a sarcoma from its inception.

**Symptoms.**—Many fibroids cause no symptoms and need no treatment. On grounds of size alone they need not be removed until they reach that of a fourteen weeks pregnancy. The classical symptom is menorrhagia either alone or in company with pain. Other symptoms are pressure effects, such as hæmorrhoids, sciatica, or frequency of micturition, and complications arising during pregnancy.



Retention of urine is caused by fibroids which grow in the pelvic cavity, *e.g.*, cervical or low corporeal tumours, and which enlarge until they fill the pelvis. This clearly displaces the bladder up into the abdomen, the urethra is considerably stretched and urinary retention follows.

*Treatment.*—Those cases in which menorrhagia is the only symptom may be successfully treated with X-rays or radium, or even by ergot alone. When other complications or other symptoms coexist, various surgical procedures should be adopted. Vaginal myomectomy is suitable for submucous and polypoid fibroids; abdominal myomectomy is indicated when it is desired to preserve the uterus, *e.g.*, in a married woman under 40 years of age; and finally in many cases hysterectomy should be performed. Complete removal of the uterus is necessary in all women who have suffered any damage to, or infection of, the cervix. Subtotal hysterectomy is reserved for nulliparous women who have never had any infective process in the cervix. Much unnecessary suffering and many needless operations would be avoided if these conditions were fulfilled.

### ADENOMYOMA

This is a form of endometrioma of the uterus and may occur as a localised tumour or as a diffuse invasion of the whole organ. The diagnosis is rarely made until the uterus is examined after operation. It suffices to state that clinically this condition closely resembles fibroids and it is upon this diagnosis that hysterectomy is proposed.

### POLYPI

1. **Mucous Polypi** of the body cause menorrhagia, those of the cervix discharge and occasional bleeding. Treatment is removal and curettage.

2. **Placental Polypi** consist of remnants of placenta covered by blood clot and fibrin. They give rise to continued bleeding after childbirth or a miscarriage and should be removed by curettage.

3. **Fibroid Polypi** are submucous fibroids which have gained a pedicle by projecting into the lumen of the uterus. They cause metrorrhagia and the hæmorrhage may be so severe that a profound degree of anæmia results. Their presence may be diagnosed by continued bleeding from a uterus which is obviously enlarged by fibroids. Alternatively one may be discovered as a friable mass presenting through the cervix. They should be removed and the uterine cavity explored with a finger, sponge holder or curette to exclude the presence of a second smaller fibroid polyp.

### CARCINOMA OF THE UTERINE BODY

This growth usually occurs in women well past the menopause who have borne few or no children. It causes irregular bleeding, sometimes accompanied by pain due to uterine contractions. All cases of post-menopausal bleeding must be suspected of malignancy and curetted so that the diagnosis may rest upon an histological basis.

*Treatment* consists in a panhysterectomy together with removal

of both tubes and ovaries. Results of this operation show a 60 per cent. cure and it should be advised in preference to radiation.

### CARCINOMA OF THE CERVIX

This occurs at an earlier age, often between 45 and 50 years, in women who have borne children. It is predisposed to by injury and infection of the cervix and gives rise to a watery blood-stained discharge, which later becomes offensive. It is unfortunate that women at this age regard irregular bleeding as a more or less natural phenomenon and, by neglecting to seek advice, allow the growth to become advanced before they are examined.

On examination the cervix bleeds easily and a rough friable area is felt. A useful test is to press the point of a sound against such a spot. If the point penetrates easily the diagnosis of carcinoma is established. In every case of doubt a piece of suspected tissue must be removed for microscopy.

*Treatment.*—In early cases in women under 65 years of age who are not too fat, Wertheim's extended abdominal hysterectomy is still considered the method of choice by some gynaecologists. But the general trend of opinion is to regard treatment by radium locally (following the Stockholm or Paris techniques) and deep X-ray therapy to the gland areas as preferable.

*Differential Diagnosis*—**Erosion of the cervix** is an inflammatory process in that part of the cervix around the external os which is covered with columnar and not squamous epithelium. This columnar type of cell bleeds easily when touched and, as it is secreting actively, produces an excess discharge which appears per vaginam. On inspection by the inexperienced its appearance may be confused with carcinoma, but it may easily be distinguished by the absence of friability when tested with the sound. This condition is treated by destruction of the mucous membrane by diathermy.

**Ectropion of the Cervix** results from the splitting of the cervix during labour into an anterior and a posterior lip. These have become covered with columnar epithelium. The cervix is seen as a red gaping structure which bleeds easily and produces a profuse discharge. It may also be associated with low backache. The sound test for friability serves to distinguish it from carcinoma.

*Treatment* is operative reconstruction of the normal shape of the cervix.

### SARCOMA

Sarcoma of the uterus is rare and occurs in the body rather than the cervix. In the former it is seen as a diffuse growth causing bleeding and uterine enlargement. It shows a marked clinical resemblance to fibroids, but the rate of growth is more rapid.

*Treatment* is panhysterectomy with bilateral salpingo-oöphorectomy.

### CHORIONIC CARCINOMA

This rare and fatal disease occasionally occurs in the tubes after an ectopic gestation, but is more often found in the uterus. It is apt to

follow a hydatidiform mole (Figs. 404 and 405) or, much more rarely, a normal pregnancy. Such cases are suggested by continued bleeding after the passage of a hydatidiform mole. It is probably unwise to explore the uterus with a curette in these patients since this may serve to disseminate the tumour. It is safer to rely upon the Aschheim-



FIG. 404

A hydatidiform mole after being passed from the uterus.



FIG. 405

A hydatidiform mole *in situ*. At the right-hand lower corner a chorionic carcinoma has developed.

Zondek reaction which is positive in a 100 times dilution of urine for both the mole and this type of carcinoma. The mole having been diagnosed by the test is passed or removed; the test becomes weak or negative; a return of uterine bleeding together with a strongly positive test provides a sufficient basis for a diagnosis of chorionic carcinoma. Under such conditions a panhysterectomy must be performed, but the prognosis is poor. These growths are radio-sensitive and radium and deep X-ray therapy may help to improve the results.

LESLIE WILLIAMS.

## CHAPTER XL

### DISEASES OF THE SCALP AND SKULL

#### THE SURGERY OF THE SCALP

**S**URGICAL ANATOMY.—The soft tissues covering the vault of the skull have a highly specialised structure. The *skin* is thick and profusely supplied with hair follicles, while the *subcutaneous tissue* comprises a thin fibrous layer containing lobules of coarse fat. Underlying this is the *occipito-frontalis* (or epicranial) *aponeurosis*, a broad sheet of fibrous tissue acting as an intermediate tendon between the occipitalis muscle behind and the frontalis in front. These three strata of the scalp are so closely attached to each other that they enjoy a limited range of movement together through the action of the occipito-frontalis muscle. Beneath the epicranial aponeurosis is a space filled with loose areolar tissue, lying directly upon the *pericranium* (i.e., periosteum of the skull). It is this space which permits the movements of the scalp. The epicranial aponeurosis fuses laterally with the fascia covering the temporal muscle, while the occipito-frontalis is attached in front to the superciliary ridges and behind to the superior curved line of the occipital bone. These relations are of great surgical importance, in that effusions of blood or pus beneath the aponeurosis can spread widely in all directions.

The *blood supply* of the scalp is derived from the supra-orbital branch of the internal carotid artery in front and from the superficial temporal, posterior auricular and occipital branches of the external carotid behind and laterally. There is a free anastomosis between the two sides. The scalp is particularly richly supplied with vessels which run in the fibrous subcutaneous tissue; to this their outer coats are attached, so that they cannot retract when incised or lacerated. It is for this reason that wounds of the scalp bleed so profusely. The *venous return* also is free, and presents one anatomical relation which has a surgical significance of the highest import, viz., the intercommunication between the veins of the scalp and the great venous sinuses of the interior of the skull by means of "emissary veins." These are found in the temporal and occipital regions (with the lateral sinus), in the parietal and nasal areas (with the superior longitudinal sinus) and at the inner angle of the orbit, where the angular vein effects a communication with the cavernous sinus.

The *lymphatics* run to the pre-auricular, occipital and posterior cervical glands; consequently, infective lesions of the scalp will usually cause enlargement of the glands in the posterior triangle of the neck.

The *nerve supply* is from the auriculo-temporal, supratrochlear and supra-orbital branches of the Vth cranial nerve, and from the great and small occipital nerves derived from the second cervical.

The *pericranium* has a loose attachment to the bones of the skull, except at the sutures, where it is closely adherent. Inflammatory and hæmorrhagic effusions beneath this membrane, therefore, can spread only to the extent of the bone concerned.

### INJURIES OF THE SCALP

**Hæmatoma** of the scalp is caused by blows of moderate violence, either by blunt instruments or as the result of a fall. It is seen in the heads of newborn babies after a prolonged or difficult labour with or without forceps. Three types are described :

1. **SUPERFICIAL BRUISES** are confined to the fibrous subcutaneous tissue and are therefore small and circumscribed.

2. **SUBAPONEUROTIC**, *i.e.*, beneath the epicranial aponeurosis, are often a result of fracture of the vault of the skull. The extravasation is limited only by the attachments of the occipito-frontalis and, if the bleeding is severe, the scalp appears to be floating on a fluctuating cushion, which may pulsate when a large artery has been torn. The swelling tends to collect at the dependent margins, *i.e.*, the eyebrow, temporal and occipital regions. In many cases, however, the effusion remains localised to the zone of trauma.

3. **SUPERICRANIAL** collections are uncommon. The effusion is confined to the extent of the bone involved by the firm attachment of the pericranium to the sutures. They are seen as a result of injury either at birth or at a later age. These **cephalhæmatomata** appear as soft fluctuating swellings which rapidly achieve a raised and indurated surrounding wall of blood clot and fibrin. The clinical signs are sufficiently misleading to suggest to the inexperienced a depressed fracture. The two conditions should in reality easily be differentiated, for the edge of a hæmatoma is raised above the level of the bone and can be made to pit on firm pressure.

*Treatment* of all hæmatomata of the scalp consists in rest in bed and the application of cooling lotions to the head. It must be remembered that their importance lies in the likelihood of their being associated with far more serious injury to the underlying bone and brain.

**Wounds** are of great frequency, and it is fortunate that the scalp with its generous blood supply possesses remarkable powers of healing. These wounds tend to be triangular in shape, a flap often being torn down from the skull and, even although the pedicle is quite narrow, the blood supply will be sufficient to prevent sloughing. If the wound is superficial to the aponeurosis the edges do not gape and little damage is likely, but if this is divided the margins retract and there is great danger of sepsis being implanted in the loose areolar tissue, in which it may spread far and wide.

*Treatment.*—Before suturing the wound, the hair around it must be shaved and its depth examined for the presence of bone injury or foreign body. It is then thoroughly cleansed with a 1 in 1000 acriflavine lotion. The edges are approximated with a few points of suture after dusting the wound lightly with sulphanilamide powder. It is as a rule impossible to seize any but the larger vessels, and the tissues are so tough that ligatures cannot usually be tied ; therefore it is fortunate that the support afforded by skin sutures is enough to stop bleeding. The repair of accidental, and therefore possibly septic, wounds should be done with few stitches, leaving considerable

gaps to allow for drainage, and these should be removed promptly should sepsis appear. On the other hand, suture of surgical incisions should be done meticulously in layers with fine ophthalmic silkworm gut or waxed silk, so as to secure perfect coaptation. The stitches can be removed after forty-eight hours, buried sutures uniting the aponeurosis being relied upon to prevent gaping of the wound.

**Avulsion of the Scalp** is produced in women workers by their hair being caught in machinery. The tear occurs above the ears and eyebrows and the scalp is pulled backwards.

*Treatment* consists in thorough cleansing of the area followed by repair. If the scalp has been completely severed, it should be secured, and if it is unmangled, it is worth suturing it in position as a full-thickness graft. Even if only a small part survives, it assists in the

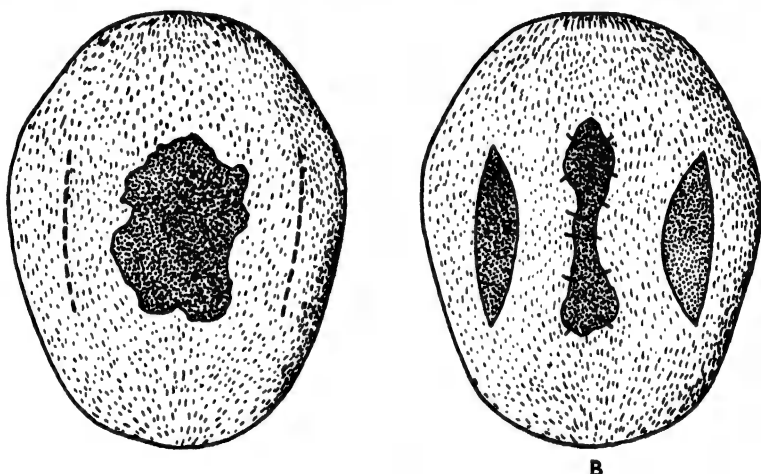


FIG. 406

A shows the incisions made on either side of the central raw area ;  
B illustrates the decrease in size of the latter.

subsequent healing. Large raw areas can be covered by the method shown in Fig. 406. An incision is made on each side and the two strips are slid inwards and sutured together. All uncovered areas will be filled later with skin grafts.

**Burns of the Scalp** are of three types : (1) Mild superficial burns from hair-waving machines do not cause much pain, but lying concealed under the hair are liable to become septic. (2) More severe burns result from accidents with celluloid combs. They leave intractable ulcers, which lead to difficulties in treatment. (3) Deep burns sometimes follow epileptic seizures or alcoholic stupor as a result of the patient falling and resting his head either in the fire or on the hot bars. The bone may be destroyed and septic intracranial complications follow.

### DISEASES OF THE SCALP

**Sepsis in the Scalp** arises in the spread of infection either from the surface or from the bones of the skull. As in the description of hæmatomata, so in this case three types of abscess are defined.

1. **SUBCUTANEOUS ABSCESES** are small and circumscribed, usually following surface infections such as eczema, impetigo, pediculosis or boils in young patients.

*Treatment* is incision.

2. **SUBAPONEUROTIC INFECTION** is serious owing to the absence of any barrier to its spread over the whole vault. Pus may point at one or more places around the periphery of the epicranial aponeurosis. This type of sepsis is especially common to-day in association with motor accidents.

*Treatment* consists in one or more incisions at the margin of the swelling.

3. **SURPERICRANIAL ABSCESS** must always be a grave lesion. It is due either to disease of the bone or to tracking of pus through the skull from intracranial suppuration. Early incision and drainage is called for.

**Cellulitis and Erysipelas** are common complications of scalp wounds. They have the same appearance here as in other parts of the body. Their treatment has been greatly simplified since the introduction of sulphanilamide (p. 27).

**Cysts of the Scalp.**—**SEBACEOUS CYSTS** (Epidermoids or Wens) are very common and are frequently multiple. They have been described on p. 242. They rarely become malignant, giving rise to a "Cock's peculiar tumour" (Fig. 407). *Treatment* is excision under local anæsthesia.



FIG. 407

Cock's peculiar tumour.

**DERMOID CYSTS** occur only in the neighbourhood of the anterior and posterior fontanelles and in the temporal area. They are described in general on p. 114, and those on the face on p. 305. They are not attached to the skin, but have a fibrous pedicle to the underlying bone.

*Treatment* is preferably postponed until after puberty, when the cyst should be removed.

**Vascular Swellings.**—**Nævi** and aneurysms of all types occur in the scalp as elsewhere.

**CIRROID ANEURYSM** (p. 287) is rarely seen except in the scalp and face, commencing usually in the temporal region, whence it may spread over the whole head and downwards to the face and ear. Large tortuous pulsating vessels are seen, and the patient complains of rushing and roaring noises, giddiness and headaches. These aneurysms develop from angiomas, generally of the "port wine stain" type.

*Treatment* is not very satisfactory, but a recent two-stage operation marks an advance. A flap of scalp including the aneurysm is turned slowly down and all the open vessels at the edge coagulated by diathermy. The flap is replaced with a pack of rubber tissue between it and the bone. Ten days later it is again reflected, when the vessels will be found to have thrombosed and can be dissected out.



**New Growths.**—**ADENOMATA** of both sebaceous and sweat glands are rarely seen in the scalp, and a still more uncommon tumour is the adenoma adenoides cysticum (p. 242).

**PAPILLOMATA** in the form of small warts are common but give little inconvenience. **LIPOMATA**, often arising from the pericranium, appear as flattened circular swellings. **NEUROFIBROMATA** are occasionally met with in the scalp, usually in the distribution of the supra-orbital nerve. When large they become pedunculated, being then known as **PACHYDERMATOCELES**. All these tumours should be removed if causing symptoms.

A **SQUAMOUS-CELLED CARCINOMA** is far less common in the scalp than on the face, but sometimes arises in a wart, scar or sebaceous cyst. It must be removed and the raw area covered, if necessary, by skin grafting.

**SARCOMA** rarely occurs except as the result of invasion from the underlying bone or in the form of secondary deposits.

**Cephalhæmatocele** (sinus pericranii) is an exceedingly rare swelling, consisting in a collection of venous spaces, which communicate with the superior longitudinal sinus through a gap in the suture. It appears as a soft tumour in the midline over the vertex and has an impulse on coughing. It must be excised and the communicating channel ligated.

## THE SURGERY OF THE SKULL

Head injuries form a large proportion of the emergency admissions to hospital at the present time. It is of the greatest importance that, at the outset, students should appreciate the relative significance of fractures of the skull. Intrinsically an uncomplicated fracture of any part of the cranium is of little import and requires no treatment beyond rest in bed. The force, however, capable of fracturing the skull, must be such that the intracranial contents are hardly likely to escape injury. Attention, therefore, must be directed more critically to the signs and symptoms of damage to both brain and cranial nerves rather than to the diagnosis of fracture. An inexperienced resident so frequently regards an X-ray as an urgent necessity in a patient who obviously has a severe contusion or laceration of the brain. Certain complications are more often associated with fractures of different parts of the skull; consequently it is customary to describe the latter under separate headings.

A **contusion** of the skull is usually accompanied by injury to the overlying soft parts. A **hæmatoma** may collect beneath the pericranium and will have no serious effects, provided that it does not become infected. Should this occur, osteomyelitis may follow with grave possibilities, such as subdural or intradural abscess.

## FRACTURE OF THE CRANIAL VAULT

### FISSURED FRACTURES

A fissured fracture, as its name implies, is a crack in the skull, usually of the vertex, without displacement. It may consist in one



fissure or may assume a star shape with radiating cracks passing in several directions. The length of any one fracture varies greatly; in some cases it may pass downwards until it involves the base of the skull. Care must be taken that the normal sutures and vascular channels are not mistaken for fracture lines. This type of injury is the result of a fall or blow from a blunt instrument. It may be closed or compound, but in every case a considerable degree of bruising of the soft tissues will be present.

In the closed fracture there will be no physical signs to reveal its presence and only an X-ray will determine the diagnosis. If it is compound it can be felt with a probe and blood may be seen oozing from the line of fracture. While any intracranial complication may follow this type of fracture, those regarded as direct results are concussion, middle meningeal hæmorrhage, and laceration of the longitudinal sinus.

*Treatment.*—If the fracture is compound, attention should be directed to the toilet of the wound and the prevention of sepsis. In every case absolute rest in bed for four weeks is necessary, and a close watch kept for intracranial complications. In a small percentage of cases delayed symptoms arise many weeks after injury; these are grouped under the heading of “Traumatic Neurasthenia.”

#### DEPRESSED FRACTURES

A depressed fracture occurs in the vault of the skull as a result of a fall or blow, when the injury may be either simple or compound. A **punctured fracture** is a type of the foregoing, being due to a penetrating wound from bullets, shell fragments, and the like. In all cases there is considerable comminution of bone and in this type spicules of bone may be driven into the brain. It is usual for both tables to be broken simultaneously, but occasionally the outer alone is depressed (e.g., over the frontal sinus) or the inner may yield, the outer having sprung back without fracture, which is rare except in children.

Usually the inner table is damaged slightly more extensively than the outer, and especially is this marked in penetrating wounds by high-velocity missiles. The appearance of the depression is of some clinical importance; a **pond fracture** is saucer-shaped, having gradually shelving walls, whereas a **gutter fracture** has clean-cut margins, below which lies the stove-in fragment.

Intracranial complications are less likely to occur in this type of injury, presumably because the force has been expended in smashing the bone. The clinical picture will depend upon the shape, size and position of the fragment, the presence of an open wound and the integrity of the dura.

*Clinical Picture.*—A. A closed depressed fracture is unlikely to occur except in children (Fig. 408). It will be covered by a contusion of the scalp and a subpericranial hæmatoma, the presence of which will tend to obscure the diagnosis of fracture (cf. p. 825). Careful palpation should enable the clinician to recognise (1) the raised edge

of the hæmatoma, which can be made to pit on pressure, (2) the edge of the gap in the skull, and (3) in the centre the depressed fragment lying definitely below the level of the skull surface. The associated intracranial lesions vary considerably from mild concussion to extensive laceration of the brain. These are not usual, however, and the patient rapidly improves. If no treatment is adopted and the fragment allowed to remain depressed, delayed symptoms are likely, though not certain to occur. These are headache, giddiness and traumatic epilepsy.

*B. Compound depressed fracture* can hardly escape immediate diagnosis. An open wound is present, the hæmorrhage is profuse and the depressed fragment can be felt or seen. If the dura is torn, cerebrospinal fluid and possibly even brain substance appear in the wound. The intracranial lesions associated with this type of injury may be surprisingly slight, but its true significance lies in the grave dangers likely to follow the advent of sepsis in the wound.



FIG. 408

A small boy with a depressed fracture in the right temporo-frontal region. The superficial bruising and the extravasation of blood into the upper eyelid is well shown.

*C. Punctured wounds* are always compound but differ somewhat from those described above, in that the wound of entry is small but the underlying tissues are seriously damaged. A bullet, for instance, will cause a punctured wound of entry, while the bone is fractured and

depressed and extensive laceration of the brain by both missile and indriven bone occurs. These wounds are even more sinister in their possibilities and must never be judged by the size of their wound of entry.

*Treatment.*—Certain general principles govern all treatment. They are (1) prevention of sepsis, (2) elevation of the depressed fragment and (3) appropriate attention to associated intracranial lesions.

Simple depressed fractures in children should not be operated upon at first, because a great many recover spontaneously. If later elevation of the fragment is deemed necessary, all bruising of the soft tissues will have subsided and the danger of infection thereby lessened. In adults there is some difference of opinion as to the correct treatment. It is well to operate when the fracture is over the motor or speech cortex. When over silent brain areas elevation of the bone is not so important.

Compound fractures must invariably be submitted to operation, in order to render the wound as aseptic as possible. The head is shaved and cleansed, the wound excised, and the bone exposed by a large curved incision, which allows a flap to be turned down. All

loose spicules of bone are removed and the depressed fragment levered up into position. If the dura is intact this will suffice, the flap being replaced and firmly sutured with a small wick of rubber tissue drainage.

If the dura has been torn its edges must be trimmed and the exact extent of the damage gently investigated. All foreign material must be removed from the track and damaged cerebral tissue washed out by a stream of warm saline or aspirated through a suction tube. Gentleness of handling and thoroughness of the cleaning-up process are equally important if gross sepsis is to be avoided. The "sew-up" should be water-tight to prevent fistula of cerebrospinal fluid or hernia cerebri. The use of sulphanilamide powder makes this a safe procedure except in exceptionally soiled or late cases when a drain may be required.

In gunshot wounds the internal damage is usually far more serious than the external appearance suggests. They will demand extensive exposure and most thorough exploration together with meticulous care in cleansing and drainage.

The complications of a depressed fracture are primarily concerned with infection, and a careful watch must be kept during convalescence for osteitis of the broken bone, extradural abscess, meningitis or cerebral abscess. An uncommon sequela of uninfected closed fracture is known as a **Traumatic Cephalhydrocele**. It appears in about a week after injury, when the hæmatoma is subsiding, as a soft fluctuating swelling. It is of slow growth, painless, reducible on pressure, has an impulse on coughing and may pulsate. It contains cerebrospinal fluid which has escaped through a rent in the dura and a crack in the bone and has come to lie either beneath the pericranium or more probably in the subaponeurotic space.

*Treatment* is not necessary for the small ones, but if they continue to enlarge it is wise to expose the pedicle and close the dural defect. Good results are also obtained by repeated aspirations.

### FRACTURES OF THE BASE OF THE SKULL

The base of the skull is considerably weaker than the vertex. The bone itself is thinner in places, especially in the floor of the three fossæ, while it is further weakened by a large number of foramina of varying size, which afford transit to important vessels and nerves.

Fractures of the base, therefore, are far more common than those of the vertex. The great majority are due to indirect violence, the head coming into violent contact with some object, and only a small proportion are the result of direct injury. Examples of this latter are penetrating wounds of the nasal cavities, roof of the mouth or orbit, and fractures produced by impact of the vertebral column when a patient falls from a height on to the feet or buttocks. Fractures due to indirect violence follow a force applied to the vertex, which, being elastic, yields but does not break. The base, being rigid, gives way either because a fissured fracture from the lower aspects of the vertex "radiates" into the base (Aran's Theory) or from a "bursting or

compression force." This latter theory is founded on the elasticity of the cranium; when a heavy force acts on the skull in one axis, a narrowing must occur in it, and this is accompanied by a widening or elongation in the opposite plane. If the limit of elasticity is exceeded, a bursting fracture will result.

The fracture is usually fissured in type, and its line may pass in any direction, so that it affects one or all of the fossæ. The great majority are compound, although the surface wound is rarely apparent and can be deduced only from the symptoms. Thus a fracture of the anterior fossa opens the nasal cavity, via the cribriform plate, the ethmoidal or sphenoidal air sinuses; that of the middle fossa enters the nasopharynx, middle ear, or external auditory meatus; that in the posterior fossa may communicate with the nasopharynx, but is less likely to be compound than any of the others. If the dura also is breached, the subdural space is vulnerable to direct spread of infection from the exterior and meningitis may follow.

The intracranial lesions associated with a fracture of the base are usually severe, and extensive contusion or laceration of the brain must be expected. The complications directly attributable to the fracture concern injury to those vessels and nerves which pass through the foramina in the base of the skull, so that extradural hæmorrhage and lesions of the cranial nerves are commonly seen.

*Clinical Picture.*—It must be understood that a fractured base will probably give no signs referable primarily to itself, and its presence must be deduced from other evidence. This includes:—

1. External hæmorrhage from the ear, nose, and mouth.
2. Hæmorrhage into the eyelids and beneath the conjunctiva—the "anterior fossa black eye."
3. Escape of cerebrospinal fluid from the nose, ear or mouth.
4. Involvement of cranial nerves, of which the VIIth and VIIIth are the most significant.

It is convenient to group these fractures under the heading of the fossa implicated. It will be remembered, however, that more than one is involved in many cases.

**A. Anterior Fossa.**—The salient features are a characteristic extravasation of blood into the orbits and external bleeding from the nose and mouth. Some of this blood will be swallowed and appear later in the vomit. Cerebrospinal fluid is likely to escape into the nose and mouth, but its presence will probably not be recognised owing to its admixture with blood. The orbital bleeding first appears at the lower and outer part of the conjunctiva, spreads forward from the back of the orbit and finally reaches the corneal margin around its entire circumference. Simultaneously bruising becomes apparent in the lower and then the upper eyelid, but the typical purple discoloration stops short at the orbital margins and never encroaches either on the face or forehead. In these ways can an "anterior fossa black eye" be distinguished from one due to local trauma.

Fractures of the accessory sinuses and cribriform plate, in addition to allowing cerebrospinal fluid to escape, also permit ingress of air

when the patient blows his nose. The air may (1) form a localised pneumatocele, (2) spread over the cortex of the brain, or (3) even fill the ventricles. Fig. 409 is an excellent example of all these three types of air collection after a fractured skull.

The nerves liable to injury are the Ist, II<sup>nd</sup>, III<sup>rd</sup>, IV<sup>th</sup>, first division of the V<sup>th</sup> and the VI<sup>th</sup>, though it is not common for them to be sufficiently damaged to give evidence of their involvement.



FIG. 409

An X-ray showing air over the cerebral convolutions A, encysted in the cortex B and in the ventricle C. The hole D marks the site of bone removed to evacuate an extradural hæmatoma. The fracture can be seen just above the hole, and the groove for the middle meningeal artery below it.

**B. Middle Fossa.**—The majority of fractures of the base involve the middle fossa, and show a marked tendency to pass through the petrous portion of the temporal bone, thereby implicating the tympanic cavity and possibly tearing the tympanic membrane. In these fractures both blood and cerebrospinal fluid will probably escape from the external auditory meatus, often in considerable quantities. The blood is dark in colour, flows continuously, and is apt to persist for

several days, whereas the bleeding from a simple tear of the tympanic membrane is bright red and of short duration.

In this situation the involvement of cranial nerves constitutes the most important aspect of the picture, for the second and third divisions of the Vth nerve, the VIth, VIIth and VIIIth nerves are all dangerously placed. The facial and auditory nerves are more frequently injured than all the others, and they may be damaged in several ways. Either they are torn across at the time of accident, compressed by blood clot in their canals in the temporal bone or later interfered with by subsequent callus formation. The symptoms and signs of facial palsy and of deafness may therefore appear either immediately, within the first five days or after a period of some weeks. The deafness may be due to damage to the labyrinth rather than to the nerve itself or to interference with the ossicles in the middle ear. In all cases the prognosis with regard to hearing is poor.

**C. Posterior Fossa.**—External bleeding is unlikely to occur, but after a few days bruising will make its appearance behind and below the mastoid process in the neck. The intracranial damage is probably severe, but there will be no symptoms directly due to the fracture.

*Prognosis.*—The future of a patient after a head injury depends almost entirely upon the extent and nature of injury to the contents of the skull, and the fracture of the cranium has little, if anything, to add to those data which are being weighed in the balance. Suffice it to say that a simple uncomplicated fracture of the skull is of less significance than that of a long bone in the lower limb.

*Treatment* is devoted to the relief of complications. A simple fracture without associated lesions needs rest in bed for fourteen days, a similar injury with concussion demands twenty-eight days in bed, and in neither case may the patient return to work before eight weeks have elapsed. Treatment of those fractures accompanied by intracranial lesions is dictated by the requirements of these latter and not by the bony injury.

Bleeding and loss of cerebrospinal fluid from the ear deserve a special word of warning. Once every day the external meatus must be gently swabbed to remove blood clot and debris. Its opening is carefully bathed with a warm solution of mild antiseptic and then lightly plugged with sterile wool. On no account must the ear be syringed, and it is wiser to postpone inspection of the drum with a speculum till the seventh day.

Loss of cerebrospinal fluid almost always ceases spontaneously, but certain rare cases of "liquorrhoea" through the cribriform plate occur, in which the loss may be so great and so persistent that an operation must be done to arrest it. The frontal lobe is exposed and raised, the rent in the dura identified and a small muscle graft inserted.

Facial palsy may call for surgical measures to relieve pressure on the nerve. If there is reason to believe that the nerve is injured in the Fallopian aqueduct, muscle weakness is persisting and the reaction of degeneration present, the nerve must be exposed and decompressed, the wall of the canal being removed and the fibrous sheath slit up.

## DISEASES OF THE SKULL

### CONGENITAL ANOMALIES

**Congenital Aplasia Cranii** is a condition of incomplete ossification of the skull, which may persist into adult life. It is a rare defect seen only in children borne to an enfeebled mother. No treatment is of any avail.

**Cephaloceles** are protrusions of the dura mater, with or without brain tissue, through an opening in the bone, being similar in many respects to spina bifida. They are due to the failure of the mesoblast completely to enclose the primary cerebral vesicle, so that a small portion of the latter is left outside the skull with a channel of communication to the dura passing through the bone. Cephaloceles are covered by normal skin, but this may become thinned and adherent if the swelling reaches a great size. They are seen only in certain situations, commonly at the root of the nose and in the occipital region and very rarely in the neighbourhood of the anterior fontanelle, the ear and mastoid process, or even in the basisphenoid area, thereby bulging the posterior pharyngeal wall. They indicate an increase in intracranial pressure and are associated with some degree of hydrocephalus. Other congenital anomalies such as spina bifida, cleft palate or hare-lip are often present in these cases. Three varieties are described.

1. **MENINGOCELE** is a protrusion of dura containing only cerebro-spinal fluid and is the commonest form, being found in the occipital region. It presents a soft fluctuating swelling which is translucent, reducible on pressure, and pulsates with respiration but not with the heart beat. It becomes larger and more tense when the child strains, cries or coughs. Gentle pressure reduces the swelling, but causes vomiting and possibly convulsions. The skin and hair are normal over the smaller varieties, which tend to remain stationary in size, while the larger ones progressively increase so that the skin becomes thin and finally bursts, the child dying from meningitis.

2. **ENCEPHALOCELE** occurs at the root of the nose. The swelling is less soft, does not transmit light, and pulsates synchronously with the heart. Pressure causes convulsions, while spastic paralysis and altered reflexes are usually present.

3. **MENINGOCEPHALOCELE** is seen in the occipital region, either above or below the level of the tentorium. It is always associated with hydrocephalus, and a portion of the posterior cornu of the lateral ventricle may be included in the sac. It is probably incompatible with life.

*Prognosis* is exceedingly poor. Death is likely at an early age, and if the child survives it will be subject to fits and of feeble mental development.

*Treatment.*—Small simple meningoceles can be excised and the opening in the dura closed, but in nearly every case a hydrocephalus will follow. In other patients aspiration succeeded by firm bandaging may arrest the increase in size.

**Microcephaly** is associated with premature closure of the sutures and fontanelles and is often accompanied by imbecility. **Macrocephaly**,



on the other hand, is not always a cause of mental deficiency, as all the structures of the head are larger than normal. Historical examples of this condition are those of Lord Byron and Bismarck. **Oxycephaly** or steeple skull is a peculiar anomaly, the forehead being high and the skull egg-shaped. Its importance lies in the almost certain development of increased intracranial pressure and blindness.

### INFLAMMATORY DISEASES OF THE SKULL

**Acute Osteomyelitis** is an inflammatory process due to pyogenic cocci. Its pathology is similar to that in long bones, except in certain respects. The infection is rarely carried to the skull by the blood stream, but reaches it from a focus of local disease or injury. Thus infected scalp wounds and compound fractures are common causes, whilst the bone can be involved by direct spread from the frontal sinus or intracranial suppuration. The inflammation does not necessarily affect both tables at first, but later both pericranium and dura are lifted off the bone by the inflammatory exudate. Sequestra form in the same way as in a long bone, but take an unduly long time to separate. The pericranium seems unable to produce an involucrum, and the defect may be closed only by a thick fibrous membrane, although in some cases a new calvarium is regenerated from osteoblasts attached to the dura. The necrosis is limited at first by the sutures to the bone primarily infected, but the infection can jump the sutures.

*Symptoms.*—The onset is sudden; headache, local pain and a rigor usher in the disease. The temperature is high ( $104^{\circ}$  to  $105^{\circ}$  F.), and there is a tender swelling over the affected bone. At first tense and indurated, this area softens and fluctuates as pus is formed. A particular type of swelling is seen in certain cases of extradural abscess, sometimes associated with a fracture of the skull. It appears some days after the onset of the illness as a localised, boggy, oedematous tumour, to which the name "**Percival Pott's Puffy Tumour**" is given.

*Prognosis* in this disease is grave because of the serious complications which may arise. These include meningitis, cerebral abscess, sinus thrombosis, septicæmia and pyæmia.

*Treatment* should be instituted at the earliest possible moment. The outer table is excised to ensure free drainage to the infected diploe; if signs of extradural abscess are present the inner table also must be removed. The later procedure is a matter of controversy, one school advocating radical removal of bone, another advising drainage of abscesses as they occur and picking out sequestra when loose. Nafziger's method is ingenious, consisting in removing the whole plaque of diseased bone with a generous margin. This removed bone is boiled and kept in safety for six months and restored to its bed after all sepsis has died down.

**Chronic Osteoperiostitis** of the skull is usually syphilitic, but a number of simple cases occur as a result of a blow or long-continued pressure, as, for example, in Covent Garden porters who carry piles of baskets on their heads. The new bone, which takes the form of a node, should be chiselled away only if it is causing definite pain.



**Tuberculous Disease** of the skull is one of the least common manifestations of this infection. It usually occurs within the vault in young people, but may be seen in the mastoid process or malar bone. The organisms reach the bone either via the diploic vessels or the meninges. Caries follows, the inner table being more extensively diseased than the outer. Headache, local pain and tenderness call attention to the condition and an X-ray photograph will reveal the caries. It is wise to operate before an abscess makes its appearance, for the results of early radical removal are far superior to palliative measures.

**Syphilitic Disease** of the skull is seen both in the tertiary stage of the acquired form and in congenital syphilis. These manifestations are described in full in Chap. XLVII.

### NEW GROWTHS OF THE SKULL

**Osteomata** of both types occur in the skull, their general pathological characters differing in no way from those seen elsewhere. *Cancellous osteomata* are not common in the skull, whereas *ivory exostoses* are seen only in the bones of the head. They grow from either table, projecting inwards towards the brain or outwards beneath the scalp. The ivory osteoma can occur anywhere in the skull but favours the neighbourhood of the organs of special sense, and may consequently interfere with their function. These tumours are readily diagnosed by X-rays, and their removal is indicated for either pain or deafness. A very peculiar complication of these tumours is cerebrospinal liquorrhœa from the nose when the osteoma involves the accessory sinuses.

**Hæmangiomata** arise in the diploë and cause a characteristic area of destruction of bone.

**Sarcoma** arises either in the pericranium, diploë or dura. It is spindle or round-celled and exhibits the utmost rapidity of growth. So true is this, that the swelling may be mistaken for an inflammatory process, the rapid onset, redness, heat and softness of the tumour suggesting this type of lesion. Prognosis is hopeless and treatment of no avail.

**Secondary Carcinoma** is commonly met with in malignant disease of the breast, thyroid, kidney, testis and prostate. Frequently a large number of malignant emboli are widely scattered throughout the diploë, and X-rays show a very typical "moth-eaten" appearance. The skull may also be involved by direct spread from a squamous-celled carcinoma of the face or scalp.

**Osteosarcomatous Metastases** seem to have a special affinity for the skull, as does multiple myeloma. **Chloroma** is a peculiar greenish, small round-celled tumour, having a predilection for the orbit and cranial bones, and associated with the blood changes of myeloid leukæmia. **Neurocytoma** of the adrenal medulla tends to metastasise to the orbit and skull bones on the same side of the body as the primary tumour (Hutchison's syndrome). **Cholesterin deposits** from faulty lipid metabolism occur in the skull in Hand-Christian-Schüller disease, in which blindness may result from pressure on the optic nerve.

A. DICKSON WRIGHT.

R. M. HANDFIELD-JONES.

## CHAPTER XLI

### THE BRAIN AND ITS COVERINGS

**S**URGICAL *Anatomy and Physiology*.—So complex is the structure of the brain, so diverse and complicated are its functions, that it is impossible for lack of space to enter here upon a description of either.

The science of neurology has become a vast specialised subject, and the student must master its essentials before he can aspire to an understanding of the many diseases to which the brain is subject. The intracranial problems which come within the sphere of surgery are concerned chiefly with injury and new growths. Underlying all these lesions is an increase in intracranial pressure which in its turn is dependent on disturbances of the circulation of blood within the skull.

*Physiology of Intracranial Circulation*.—It must constantly be borne in mind that (1) the brain and its vessels are enclosed within an unyielding box (the skull), and (2) the vessels inside it are devoid of vaso-motor nerve supply.

Blood enters the rigid box by the internal carotid and vertebral arteries at a pressure equal to that in the extracranial portion of the internal carotid artery, viz., about 130 mm. of mercury. It leaves the skull by the internal jugular veins, the pressure in which is susceptible to respiratory variations of between minus 5 mm. and 30 mm. of mercury. During its passage through smaller arteries, arterioles, capillaries, venules, veins and venous sinuses, the circulating blood is evidently at successively falling pressures. Nevertheless, the volume of blood leaving the skull under normal conditions *must* necessarily be equal to that entering. The explanation of these two apparently contradictory facts is that the calibre of the venous outlet greatly exceeds that of the arterial inlet. A large quantity of sluggishly moving blood is contained in soft-walled, easily compressible venous sinuses just before leaving the skull. It is evident that these latter can accommodate themselves to a considerable external pressure before their calibre is diminished to the point at which it would approximate to that of the arteries. Up to this moment the only result would be to accelerate the venous flow.

The intracranial circulation, therefore, should be considered under three headings:—

- (a) The arterial system, the pressure in which is always that prevailing in the internal carotid, i.e., about 130 mm. of mercury.
- (b) The capillary system. In so far as the capillary network may be regarded as a skeleton framework on which brain tissue is built, the capillary pressure is in effect that of the brain itself.
- (c) The venous and cerebrospinal fluid systems, in which the pressure is maintained at an equal level. In quiet respiration this varies between 0 and 10 mm. of mercury. It must be regarded as the general intracranial pressure.

**EFFECTS OF INCREASING INTRACRANIAL PRESSURE**

These can best be illustrated by taking as an example a persistently increasing hæmorrhage from the middle meningeal artery. These effects, to which the clinical picture is closely allied, will be described in three stages.

**Stage I.**—Since the skull is rigid, the escaping blood must displace some of the intracranial contents, and the most easily compressible structures will be the first to feel the effects. Thus the thin-walled venous sinuses will be slowly pressed upon, so that their capacity diminishes. The process continues until the calibre of the venous outlet has been made equal to that of the arterial inlet. Venous blood will be expelled from the skull at a greater rate than normal, but is not impeded in any way. Clinically, then, this may aptly be termed the “silent stage.”

**Stage II.**—After this point has been reached further hæmorrhage will compress the venous channels still more, with the result that the flow of blood is now definitely obstructed. A condition of venous congestion is established and experimentally this has been demonstrated by the appearance of cyanosis of the brain. Clinically, this results in increased excitability in all its areas, the “stage of irritative phenomena.”

**Stage III.**—If bleeding still continues, the blood can find more space only by compressing the capillaries, as the venous sinuses are already collapsed. Within the skull the capillary pressure is synonymous with that of the brain tissue, so that in fact the brain itself is being compressed. Experimentally, this is demonstrated by the sudden transition from the blue colour of cyanosis to the dead-white of anæmia. Clinically, this may be translated into the “stage of paralysis.”

*Clinical Picture.*—A. The brain regarded as a whole. The following table, modified from Wilfred Trotter and Julian Taylor in Choyce’s “System of Surgery,” gives a concise idea of the leading symptoms.

STAGE II		STAGE III
<i>Cerebral Hemisphere</i>		
Consciousness . . .	Irritability, delirium, slowness, drowsiness.	Coma.
Motor cortex . . .	Rigidity, Jacksonian fits.	Hemiplegia, exaggerated reflexes, positive Babinski’s sign, absent abdominal reflexes.
<i>Mid-brain</i>		
Oculomotor nerve . . .	Contracted and sluggish pupil.	Dilated and fixed pupil.
<i>Bulb</i>		
Respiratory centre . . .	Deep, slow breathing.	Slow, gasping, irregular breathing.
Cardiac centre . . .	Slow, full pulse.	Rapid, small, weak pulse.
Vasomotor centre . . .	Rising blood pressure.	Falling blood pressure.

*B.* The brain in its component parts. The illustration given assumes that the brain is a solid entity occupying a simple chamber. Such, of course, is not the case, for the falx cerebri and the tentorium subdivide the cavity, so that the two cerebral hemispheres and all brain tissue lying below the tentorium can be regarded as three separate structures. These are not necessarily affected either simultaneously or equally.

In practice the successive stages described above, although perfectly correct, do not involve the brain as a whole, but the effects of increasing pressure are first felt in the neighbourhood of the injury. Symptoms will be primarily irritative and later paralytic, and those parts immediately in contact with the hæmorrhage must be more seriously affected than those at a distance. Thus, in the early stages the cerebral cortex adjacent to the blood clot will be anæmic, *i.e.*, paralysed, whilst the deeper and more distant surface areas in the same hemisphere are cyanotic, *i.e.*, in the irritative phase. Later, the whole of one half of the cerebrum will exhibit paralytic phenomena, whereas the opposite half and the mid-brain are in a state of irritation. Clearly the whole brain cannot be equally involved unless the patient is past all hope of recovery. The clinical picture of "Head Injury" must vary greatly according to the site of the blow and the nature of its effects. Nevertheless, this general description, if constantly borne in mind, will simplify the students' task in understanding those different lesions, a description of which now follows.

## RESULTS OF HEAD INJURY

### CONCUSSION

Nothing is gained by long discussion of the many theories purporting to explain the cause of concussion, the exact nature of which remains unknown.

*Symptoms.*—Severe concussion presents a characteristic picture, although this is usually seen only by eyewitnesses of the accident and rarely by the surgeon. Its onset *immediately* follows the blow. The victim, having been felled to the ground, lies in a flaccid heap in the exact position into which he has collapsed. There is complete loss of consciousness and absolute muscular relaxation, which latter may lead to incontinence of both urine and fæces. Pulse and respiration are barely perceptible, the former being either rapid or very slow. The face is pale, cold and clammy, the pupils are dilated and do not react to light. To the casual onlooker the victim appears to be dead, as indeed may be the case in a very small percentage of such accidents. In the majority this "near approach to death" lasts a few moments (at the most two to three minutes), after which signs of recovery become apparent. The pulse can be felt at the wrist, weak respiratory movements are visible, and the pupils now react to light. Almost immediately an attack of vomiting occurs, and the physical effort entailed raises the blood pressure. The vital centres are once more

revived with blood, consciousness abruptly returns and muscular control is re-established.

Concussion is not always of this severe type and many milder degrees occur. A blow may be followed by a momentary attack of unconsciousness or merely of dizziness. A not uncommon example is that of the player "knocked out" by a heavy tackle at rugby football. After a few moments flat on the ground, a somewhat dazed and giddy man resumes his place in the field, which he attempts rather ineffectually to keep. If given the ball at an opportune moment he is likely to score even against unexpected odds. In the changing room afterwards and at home that evening he will have no recollection of the game or his part in it.

Between these extremes many variations occur, and it is important to review the results which may follow.

1. Severe concussion may lead to death, which in some cases is instantaneous.

2. Severe concussion is almost certainly associated with grave lesions of the brain or of intracranial blood vessels. The unconsciousness of concussion will either merge gradually into the coma of severe brain injury or pass off completely—even if later it reappears.

3. Uncomplicated concussion quickly passes into the post-concussive phase. This includes headache, giddiness, nausea, sleeplessness, irritability, restlessness and a feeling of weakness and insecurity. These vary in number and degree in different patients, in some of whom complete recovery follows within forty-eight hours, whilst in others distressing sequelæ may persist for a long time. One of the most characteristic features of all grades of concussion is the complete loss of memory of the actual accident, and of the few minutes preceding it, as well as the succeeding two to twelve hours.

*Treatment.*—There is no indication for strenuous resuscitative measures. The patient is merely kept horizontal and warm until either he recovers consciousness or drifts into the coma of cerebral laceration or compression. A rapid neurological examination should be made at this stage, although rarely is any information of value obtained. If there is a laceration of the scalp, this should receive attention.

During the next twenty-four hours the patient must be kept in bed and watched with the utmost care and attention. The pulse rate is recorded on a special chart every half an hour. During this period repeated examinations by an expert neurologist should be carried out. In favourable cases the patient will improve and give little cause for further anxiety; in others the symptoms and signs of intracranial injury will begin to manifest themselves, and it is for the earliest of these that a close watch must be kept in every patient.

### CONTUSION AND LACERATION OF THE BRAIN

*Pathology.*—The difference between contusion and laceration is one merely of degree. The pathological processes which result are :  
(1) hæmorrhage, which may be either from surface vessels or into the

brain substance, (2) œdema of the brain, and (3) delayed softening of the damaged area. Before defining these more exactly, it is necessary to inquire how injuries of the brain occur. **Direct Injury** is produced immediately beneath the point of application of the force and will obviously vary in extent with the degree of violence, the presence and type of fracture and of foreign bodies. **Injury by Contrecoup** is less easy, though equally important, to understand. At the moment when a heavy blow falls upon the skull, the force travels from the point of impact in a definite direction through the brain to reach the skull at a point along this axis opposite to the area primarily struck. At the time of injury the brain is momentarily displaced and comes into violent contact with this zone of bone. This part of the brain, therefore, may be contused or lacerated with less, equal or greater severity than that immediately below the actual point of trauma. This is known as "Injury by Contrecoup." In the localisation of intracranial injuries this may produce a most perplexing combination of symptoms and signs, and must never be forgotten when a head injury is being examined.

**Hæmorrhage** follows a rupture of the vessels either in the meninges, on the surface of the brain or within its substance. It is evident that it may be of varying degrees of severity. A large vessel rupturing into the subarachnoid space may bleed profusely and cause death within a few moments. Usually it is of slower occurrence and more limited in extent.

Bleeding into the brain itself may occur as one large hæmorrhage or as focal points scattered throughout its substance, especially along the axis of the harmful force and at the area of contrecoup.

**Œdema.**—The brain substance in the neighbourhood of the lesion becomes œdematous in the same way as does any other soft tissue after injury. Œdema of the brain, however, assumes an altogether unusual significance because of the inelastic skull, within which it is contained, as a result of which the space available is strictly limited. The effusion of fluid causes venous engorgement in the tissues surrounding the contused or lacerated area, but the pressure of the œdema can rarely if ever rise to such an extent that anæmia takes the place of venous congestion. It is clear that the symptoms will be those of irritation and not of paralysis.

In many cases there will be multiple focal lesions of œdema and hæmorrhage along the axis of the causative force together with a possible contusion at the zone of contrecoup. The extent of œdema, therefore, is not localised to the lacerated area of brain beneath the site of injury, and consequently the clinical picture of irritation is likely to be widespread, even if not severe.

**Delayed Softening** of the damaged area of brain may occur after several weeks. If a blood vessel of any size is involved in this process, a severe hæmorrhage occurs quite suddenly with a fatal result. This condition is known as "Spät-apoplexie" of Bollinger.

**Clinical Picture.**—The widespread diffusion of the œdema makes a clear-cut description impossible. There may be localising signs as a result of interference with the functions of damaged areas of brain

tissue, but chiefly the clinical condition is dominated by the effects of oedema.

**Cerebral Irritation** is the term used to describe the state of a patient after concussion has passed off. It must clearly be understood that it does not imply that the cerebral cortex is being irritated by blood, depressed fragments of bone or other foreign substance, but that the brain is in so excitable a state that it responds more easily and more violently to stimuli than it would normally do.

The stage of post-concussive recovery is somewhat protracted, signs of shock persist, and return to full consciousness is delayed. Within twelve hours oedema is sufficiently advanced to produce the state of irritation. The patient complains of a severe headache, nausea and dizziness, all of which are instantly increased by attempted movement, for which reason he lies motionless and silent. He will be found curled up on one side, with trunk and limbs flexed. The eyelids are kept tightly shut, for there is marked photophobia. Temperature is slightly raised to, but not exceeding,  $100^{\circ}$  F., pulse rate is slow (between 60 and 70) but the volume remains good, while respiration is usually quiet, regular and normal in rate. Such is the picture as long as the man remains undisturbed, and the true state of affairs is not revealed until an attempt is made to question or examine him. Such interference is bitterly resented and he demands to be left alone. If the interrogation is pressed, he becomes noisy and angry, mutters or shouts and throws himself about in the bed. Attempts to examine the pupils are strenuously resisted. Later, especially during the night, this irritability may pass into delirium and restlessness, so that gentle restraint may be necessary. These are sometimes indications that the intracranial lesion is increasing in extent and severity. As a rule, however, a patient with typical cerebral irritation will not die from his injuries.

This stage of excitability lasts for periods varying between forty-eight hours and fourteen days, after which a gradual improvement sets in, but a state of mental confusion is apt to persist for a considerable time.

Cases of severe laceration as a rule pass to an early fatal issue, with rising pulse and temperature and terminal oedema of the lungs. The onset of bubbling in the trachea is the most ominous of all signs in head injuries and indicates a complete cerebral breakdown rather than a terminal broncho-pneumonia. If a miracle occurs and the cranial condition improves, it is amazing to find how quickly the bronchial mucus disappears and the "pneumonia" clears up.

*Treatment* aims at the avoidance of all exciting stimuli and the reduction of increased intracranial pressure. The patient must be kept completely quiet in a darkened room. Highly nutritious food in fluid form must be given and is, fortunately, well tolerated. Careful attention to the action of both bowel and bladder is important. If signs of high intracranial tension appear, lumbar puncture helps, especially in the relief of headache. It must, however, be done very carefully. A rubber tube and glass manometer are attached to the spinal needle and the fluid allowed to run over the top of the manometer, which is slowly



lowered so that half an hour is spent in reducing the pressure to normal high limits, *i.e.*, 100 mm. of water. Together with this it is wise to administer  $\frac{1}{2}$  oz. of magnesium sulphate in 2 oz. of water daily, or, if the patient is unable to swallow, 8 oz. of 50 per cent. solution of the same salt is run warm into the rectum. A still stronger method of bringing down the pressure is to inject intravenously (Weed and McKibben's method) 100 c.c. of certain hypertonic solutions, of which 50 per cent. glucose and 15 per cent. saline are suitable examples. They cause a rapid absorption of oedema fluid into the circulation by osmotic action and, when judiciously used, are of the greatest life-saving value.

Restlessness, noisiness and excitability may demand the use of sedatives. Morphia and its derivatives should not be used, but 3 gr. of sodium luminal intramuscularly is the best sedative for these cases.

### COMPRESSION OF THE BRAIN

This is produced by an increase of intracranial tension. the sequence of events in which has already been described. It may be due to intracranial hæmorrhage, increasing oedema of the brain, inflammatory lesions and tumours. Clearly the rapidity with which symptoms appear varies with the cause of the compression; thus, those of hæmorrhage will be obvious within a few hours, those of abscess are delayed for some weeks, while a tumour may remain silent for many months.

The term compression is established by long usage, but its use is open to grave criticism. Too frequently it is used to describe the later stages of increasing intracranial pressure. These, it will be recalled, are due to anæmia of the brain and are manifested by paralytic symptoms. Herein lies the danger, for this interpretation must result in failure to recognise the earlier signs of increasing tension. It should clearly be understood that the term "Compression of the Brain" is here used to cover both the pathological changes due to alterations in intracranial circulation and the clinical pictures with which they are associated.

*Symptoms and Signs.*—After a severe blow on the head concussion is an immediate result. This is followed by return to consciousness, except in those cases in which death occurs at once. No matter what may happen afterwards, be the intracranial injuries grave or trifling, this return to consciousness is almost invariably present. It may be so brief as to be momentary or it may last for many hours before the patient slowly sinks into unconsciousness. If the student will turn to the beginning of this chapter he will understand that this period of consciousness corresponds to Stage I—*i.e.*, the silent stage, of increasing intracranial tension. The quantity of blood and the speed with which it is extravasated affect the relative duration of the stages of irritation and paralysis, but every case does pass through these definitely recognisable phases. It will make the description more clear if each system is taken individually.

**A. Alterations in Consciousness.**—Middle meningeal hæmorrhage provides a classical picture. The initial concussion is succeeded by



normal consciousness for several hours. With the onset of "irritation" the patient becomes excitable, irritable and violent. His mental condition remains perfectly clear, but he is not really responsible for his actions. As the pressure increases he is offensive, pugnacious and resentful, and is in fact an exceedingly dangerous "automaton." Later, consciousness is lost and wild delirium follows. Finally, as anæmia of the brain occurs, the patient rapidly sinks into coma.

If the hæmorrhage is sub-dural and extensive, this sequence of events is greatly accelerated. The period of consciousness is momentary and the stage of excitation passes almost immediately into coma, to which the patient rapidly succumbs.

On the other hand, a very slow hæmorrhage will present a delayed and somewhat variable picture until the limit of toleration is reached, when coma abruptly appears.

**B. Motor Cortex.**—If the injured area includes the motor cortex and pyramidal tract, the irritative stage is marked by muscular rigidity and Jacksonian fits. The muscles affected depend upon the site of injury in the brain, but the whole length of the motor cortex is frequently involved. The defects are naturally seen on the opposite side of the body to the brain injury. As pressure increases, paralysis will occur, and it is possible at some stage to find paralysis of the opposite half of the body and irritative phenomena on the same side as the cerebral damage.

**C. Changes in the Pupils** are of the greatest value in diagnosis, and can be explained in tabular form, thus :—

RIGHT-SIDED CRANIAL LESION

	Right Pupil.	Left Pupil.
Early . . . .	Contracted and sluggish, <i>i.e.</i> , irritative.	Normal.
Medium . . . .	Slightly dilated and fixed, <i>i.e.</i> , early paralytic.	Contracted and sluggish, <i>i.e.</i> irritative.
Late . . . . .	Widely dilated and fixed oval, <i>i.e.</i> , late paralytic.	Slightly dilated and fixed, <i>i.e.</i> , early paralytic.

**D. Respiratory Centre.**—In the irritative phase respiration is deep and slow, in the paralytic stage stertorous, rapid and bubbling. Complete paralysis is an extremely grave sign, but provided the patient can be kept alive by artificial respiration he can still be saved if the pressure on the bulb is relieved.

**E. Cardiac Centre.**—The pulse rate is one of the chief indications of changes in the cranial circulation, and a half-hourly record must always be kept. In Stage II it is of full volume but slow, falling as low as 30 per minute. Later it changes to a rapid, small and feeble wave. The falling pulse rate, therefore, is an early indication of rising tension, the importance of which cannot be exaggerated.

**F. Vasomotor Centre.**—Cushing's *vasomotor reaction* is probably the most perfect example of the defence mechanism in the human body. It consists in successive elevations of blood pressure designed

to preserve the blood supply to the vasomotor centre in face of a steadily increasing intracranial pressure which is threatening to destroy the vitality of the life-centres in the bulb. It works in this way: the increase in tension will reach a point at which the circulation through the bulb is impeded; immediately the vasomotor centre produces a constriction of the splanchnic vessels, by which means the blood pressure rises and the bulb is adequately supplied with blood. After a time the tension beneath the tentorium has risen still further, so that once again the vital centres are imperilled. Again the vasomotor centre sends out its S O S message and the blood pressure rises still higher. Again and again is this cycle repeated until blood pressures as high as 400 mm. of mercury are recorded. Cushing's explanation proves that this increase in blood pressure is a life-saving measure, and under no circumstances must misguided steps be taken to reduce it, as, for example, by venesection.

Occasionally this steady rise in blood pressure does not occur, but in its place the *Cheyne-Stokes phenomenon* is seen. The vasomotor response is periodic, and the brain is subjected to alternating phases of activity and inaction. In the former the blood pressure is high and there is an adequate circulation throughout the brain, while in the latter the blood pressure is low. During the period of inaction the patient lies completely still as if dead, even respiratory movements being absent. During the phase of activity, respiration returns, each succeeding movement gathering strength and depth till a maximum is reached; the pulse likewise gains in volume and pressure. Muscular movements return and the patient regains semi-consciousness. As this phase dies away there is a fading of these functions, until once again the patient lies as if dead.

**G. Other Non-focal Symptoms.**—Headache, giddiness and vomiting are present in all brain injuries. Interference with speech and all special senses is present, but is rarely of value in diagnosis owing to the rapid approach of impaired consciousness.

**Differential Diagnosis.**—When the facts of an accident are fully known the diagnosis should not be in doubt. Occasionally, however, an unconscious person is found and no history can be obtained. This type of case may give rise to great difficulty.

The conditions which cause confusion are: (a) alcohol and opium poisoning; (b) coma due to uræmia and diabetes; and (c) other intracranial lesions which produce increased pressure, *e.g.*, meningitis, cerebral hæmorrhage, thrombosis, embolism, abscess and tumours.

Most of these diseases should not lead to difficulty in diagnosis, since they have certain characteristic features. But alcohol poisoning presents so many points of similarity that it has been responsible for many disastrous mistakes. The fact that a patient smells strongly of alcohol cannot be considered as conclusive evidence, for a drunken man may fall and sustain a severe head injury, while the first impulse of every eyewitness of an accident is to administer brandy to the victim.

During the stage of excitability both the alcoholic and the patient with compression are in a similar state of excitement, irritability and

aggressiveness, and in the more advanced stages both are delirious. Nevertheless, there is one point of difference between them. It has been shown that compression of the brain produces a state of dangerous automatism; the patient cannot be influenced in any way and is made more violent by even the most gentle and kindly ministrations. The alcoholic, on the other hand, is nearly always amenable to careful handling and can be persuaded, however reluctantly, to do what is wanted. Later, drunken sleep and compressive coma present such obvious differences that no difficulty should be experienced. There must always be one golden rule in all these cases. If the slightest doubt exists in the observer's mind, the patient **MUST** be admitted to hospital and not sent to a police station. It is better that a hundred drunken men should enjoy a night's lodging in hospital than that one case of head injury be found dead in a police cell on the following morning.

*Treatment* is directed towards the cause. In the presence of symptoms of gradually increasing intracranial pressure a decompression and ligation of the bleeding vessel is the ideal procedure. Many patients die before any such treatment is possible, whereas a large number never reach that stage at which decompression is necessary.

The *after-treatment* is of the greatest possible importance if disastrous sequelæ are to be avoided. Two things need special emphasis. Firstly, rest is absolutely essential in all head injuries. Patients quickly become restive in the face of enforced idleness and implore to be allowed to get up and return to work. Even in the mild cases every patient must be kept in bed for at least twenty-eight days. A darkened room, absence of noise, rigid exclusion of all visitors and prevention of worry are essentials of treatment. After one month further prolonged convalescence must be insisted upon and a return to work permitted only after complete cessation of symptoms. Secondly, a small percentage of cases require operation in the early stages; so important is this decision that the closest co-operation between neurologist and neuro-surgeon is necessary to yield the best result.

### INTRACRANIAL HÆMORRHAGE

**Extradural Hæmorrhage** is of rare occurrence and is usually from the middle meningeal artery, which is torn across during fracture of the temporal fossa. The clinical picture has already been described. The sequence of events is: concussion, a lucid interval lasting from thirty minutes to twenty-four hours, and a story of prolonged excitability passing eventually into coma. Typical pupillary changes are present and the diagnosis should never be in doubt.

The condition is easily dealt with. A straight incision (Fig. 429) is made which allows the temporal bone to be perforated; the clot is then sucked and washed out. If the bleeding point is difficult to discover, the foramen spinosum should be exposed and the artery coagulated by diathermy, but almost invariably, after the clot has been removed, the slight oozing can be dealt with by a small cigarette drain for twenty-four hours.

**Sub-dural Hæmorrhage** is found in nearly all fatal cases of head injury, although death may actually be due to laceration of the brain produced by coup or contrecoup. The hæmorrhage may be extensive and rapidly fatal, or small and localised. The clinical picture, therefore, differs from that of the classical middle meningeal hæmorrhage only in the duration of the various stages. There are other **chronic types of sub-dural hæmorrhage** arising from more trivial head injuries, generally in males after the age of 40 years. In this type the patient after the accident complains of headaches, irritability and photophobia; this condition slowly passes and about one month later he is alarmed to find symptoms such as hemiplegia, aphasia and drowsiness returning. Progressive mental changes may be severe and, finally, coma sets in. Exploration should be carried out by multiple trephine holes on the suspected side and the blood clot washed and sucked out from one trephine hole to the next. In certain cases the collection of blood will have formed a loculated cyst to which the name "Sub-dural Hæmorrhagic Cyst" is given. Should this be found at operation the trephine holes, which have been purposefully placed, can be linked by a Gigli saw and a flap turned down, after which the cyst can be removed with its enclosing membrane. The possibility of bilateral hæmatomata should always be kept in mind, and the making of small bore holes on the opposite side carried out if there is the slightest indication. These conditions are usually found close to and on either side of the superior longitudinal sinus.

**Delayed Hæmorrhage**, *i.e.*, "Spät Apoplexie" of Bollinger has already been described (p. 842).

**Hæmorrhage from the Venous Sinuses** is not common in civilian practice. The venous blood pressure is so low that little bleeding will occur unless the dura is torn. Even so, injuries to the venous sinuses are unlikely to cause serious trouble unless followed by thrombosis or infection.

**Intracranial Hæmorrhage in Newborn Babies.**—During an obstructed labour a sub-dural hæmorrhage rarely occurs inside the foetal skull. It is caused by moulding of the bones and injury to the superior longitudinal sinus. There will be some difficulty in getting the child to breathe, and its general condition gives rise to considerable anxiety. After a few days one of the limbs is found to be paralysed and, later, convulsions appear. Neglect to recognise the condition and to perform a decompression is likely to have grave results.

### SEQUELÆ OF HEAD INJURIES

**Traumatic Neurasthenia** is a common and sometimes very troublesome result of head injury, especially when litigation is pending. It includes a change for the worse in the patient's mental outlook and disposition. He complains of headache, giddiness, inability to concentrate, insomnia and phobias of various descriptions. He believes himself unable to work and is consumed with worry and anxiety. Treatment is difficult, but consists chiefly in prolonged rest. Operation can do no good, but the favourable settlement of claims for compensation may work wonders.

**Headache** is a very common and distressing sequel to many head injuries. Pain is produced or aggravated by violent exercise, stooping, worry, mental or ocular effort and changes in the weather (barometric headache). Pain localised to a small area is likely to have an organic cause, such as scarring and thickening of the meninges or damage to the brain. These latter cases may benefit by decompression.

**Epilepsy** takes the form of either grand mal, petit mal or focal, *i.e.*, Jacksonian type. Encephalography should be carried out in these cases in one of three ways: (a) *rèparage*, in which 20 c.c. of air are injected by lumbar puncture, no fluid being withdrawn; (b) replacement of 70 to 150 c.c. of cerebrospinal fluid by a slightly less quantity of air or oxygen; or (c) 50 to 70 c.c. injected by cisternal puncture. This seems to be the most popular method at present. Skiagrams may show obliteration of the sub-dural space by adhesions or cyst formation, and operative procedures are planned accordingly.

A damaged area of brain, the focus of epileptic seizures, can often be located by electro-encephalography, the Berger waves showing a slowing and increased amplitude over these areas. If good localisation is obtained, exploration and excision of the damaged cortex are always worth doing.

**Cranial Nerve Injuries.**—Blindness in one or both eyes may result from fractures traversing the optic foramina. Sometimes a chronic arachnoiditis develops in the chiasmal region and produces scotomata and other irregular visual field defects and sometimes total blindness. There should be no hesitation in exploring either type of case and relieving the pressure on the nerves, which may be due to arachnoid bands, cysts or involvement of the optic foramen in the fracture.

Permanent damage to any of the cranial nerves can result from fracture of the base, but in only one of these (the facial) is any operative interference of occasional value.

*Prevention of Sequelæ.*—Many of the results of head injury can be prevented by proper treatment. Once again emphasis is laid on the necessity for absolute rest during a long period in all these patients.

## INFLAMMATORY LESIONS WITHIN THE SKULL

### MENINGITIS

Meningitis may be a complication of any compound fracture of the skull, especially those of the base causing cerebrospinal fistulæ into the nose or ear. It also follows infected lesions of the cranial bones, of the accessory nasal sinuses and the mastoid antrum, and finally it occurs as a manifestation of certain specific infections, which reach the meninges by the blood stream.

The exudate may be either serous or purulent, generalised or localised, and affects either the outer aspect of the dura or the three membranes together. From a clinical point of view little is to be gained by pursuing these subdivisions further. The picture is a

combination of increased intracranial pressure and an acute inflammatory process. The whole problem of prognosis and treatment has been entirely revolutionised since the adoption of sulphanilamide therapy.

### EXTRADURAL ABSCESS

This follows septic compound fractures, osteomyelitis of the skull and suppurative lesions of the mastoid antrum and frontal sinus. The boggy swelling of the scalp, known as Pott's Puffy Tumour, has already been described (p. 836). If untreated, meningitis, sinus thrombosis or cerebral abscess will follow.

*Symptoms* are pyrexia, localised pain and tenderness and mild signs of increased intracranial tension. These abscesses vary considerably in their virulence and, especially as a complication of middle-ear disease, may run a prolonged and chronic course.

*Treatment* is that of the cause. The abscess must be given adequate drainage, all dead bone being removed and the primary lesion receiving appropriate attention.

### ABSCESS OF THE BRAIN

*Pathology.*—A localised inflammation of the brain may proceed to softening and suppuration, surrounded by an area of œdema. Such an abscess may run a rapid course, and within one to three weeks lead to definite symptoms of increased intracranial tension. Others are of such low virulence that they become encapsuled, and after many months produce a picture clinically indistinguishable from that of a cerebral tumour.

*Causation.*—1. **Direct Implantation** of infection occurs in compound fractures and penetrating wounds.

2. **Local Spread** of an infected exudate from neighbouring foci is unfortunately very common. It is frequently met with as a complication of mastoid suppuration and to a less extent of frontal sinusitis. In the former instance the abscess is either in the temporal lobe or cerebellum. Abscess in the temporal lobe results from perforation of the roof of the tympanum, the pus spreading into the brain, so that the collection is wholly within the cerebral tissue. In the sub-tentorial region infection tracks through the posterior wall of the tympanum, and cerebellar abscess is usually associated with lateral sinus thrombosis and extradural suppuration (see p. 394).

Frontal sinusitis and cranial osteomyelitis account for a small number of abscesses which, unfortunately, do not always lie immediately beneath the septic area as do those in middle-ear disease. An example is an abscess of the frontal lobe on the opposite side to the infected sinus. Careful neurological and possibly ventriculographic examination may therefore have to be made in these cases.

3. **Pyæmic Abscesses**, although infrequent in ordinary pyæmia, are an inexplicably common complication of chronic pulmonary infections.

especially bronchiectasis and empyema. In these diseases the abscesses of the brain are often multiple.

*Symptoms.*—Brain abscess produces symptoms due to inflammation, increased intracranial tension and its anatomical localisation. The clinical picture of abscess is commonly grafted upon that of the causative lesion, so that the one passes gradually into the other.

Temporal lobe abscess is accompanied by localising signs of great value in diagnosis. These will include hemiplegia starting in the opposite hand, Jacksonian fits, and possibly a homonymous hemianopia. If the lesion is on the left side, aphasia and word blindness will be present.

Cerebellar abscess is likely to produce affections of the Vth to XIIth cranial nerves. Signs of true cerebellar involvement are nystagmus, inco-ordination of movement and weakness in the limbs, especially on the same side as the lesion. This weakness does not involve the face and the affected muscles are flaccid (Fig. 410).

*Treatment.*—If the abscess is secondary to middle-ear disease, it is customary to open up the mastoid area thoroughly and to explore and drain the abscess through this wound. The chronic types, which resemble cerebral tumours, have so definite a capsule that they can be dissected out as if, in



FIG. 410  
Cerebellar abscess.

fact, they were true neoplasms. In every other abscess no attempt should be made to drain it, but an osteoplastic flap raised directly over it without opening the dura. Through this the abscess is aspirated, thus tiding the patient over the period of cerebral compression, so as later to remove the abscess when it is more completely encapsuled (Vincent's method). Sometimes it is necessary to tap the abscess during the period of decompression because of high intracranial tension. Cushing and Horrax favour drainage by the method of marsupialisation, *i.e.*, stitching the opened abscess capsule to the dura and packing lightly with gauze. Some cerebral herniation often occurs at the margins of the opening but subsides as the infection dies down. Other methods of treating abscesses are repeated aspiration (Dandy), packing (King), drainage through a small catheter, making only a very small trephine hole (Coleman), and drainage with a special type of wire cage (King). If an abscess is suspected, but cannot be localised, it must be remembered that ventriculography can be used in these cases.



### SINUS THROMBOSIS

Thrombosis of the cranial venous sinuses may follow suppuration in the mastoid air cells (lateral sinus) and in the frontal sinus (superior longitudinal sinus). When the **lateral sinus** thromboses in ear cases, rigors occur and the patient becomes very ill, often with a tender swelling in the neck over the jugular vein. Sometimes the nerves which share the foramen lacerum medium with the jugular bulb become involved, producing the "jugular syndrome" of IXth, Xth and XIth cranial nerve palsies. The treatment consists in proximal ligation of the jugular vein after opening the sinus in the mastoid wound. The **superior longitudinal sinus** thrombosis is more serious, and paralysis of both legs may develop and spread to the arms as a result of infarction of the motor cortex. This syndrome is associated with the names of Sargent and Holmes.

Other cases of thrombosis occur as a result of trauma and operations, and others in debilitating illnesses, such as gastro-enteritis in children, chlorosis in young girls, typhus, etc. This type is known as marantic thrombosis.

The **cavernous sinus** may thrombose spontaneously and, this being a non-septic process, a large number of cases recover. Septic thrombosis on the other hand, has almost a 100-per cent. mortality rate, and follows in the wake of orbital cellulitis, facial erysipelas, carbuncle of nose or upper lip, severe dental sepsis and otitic suppuration. In facial cases the septic process is carried by the ophthalmic vein, and in aural cases by the petrosal sinus. The appearance of a case of this condition is unmistakable, the lids being terribly swollen, the eyeball proptosed and fixed, the pupil inactive and the eye blind, while the fundus, if it can be seen through the hazy media of the eye, shows hæmorrhages and intense congestion. The patient suffers most agonising pain and, of course, has signs of severe septic infection. Heroic surgical measures have been tried in the septic cases, generally without any success, the sinus being attacked after removal of all the orbital contents or through the middle fossa.

A prophylactic measure in severe cases of facial sepsis, the value of which is somewhat disputed by some surgeons, lies in ligation of the angular vein at the inner canthus.

### HYDROCEPHALUS

Hydrocephalus is a condition of dilatation of the ventricular system. In congenital cases it is observed at birth or soon after, and because the cranial bones are unfused the head goes on enlarging and the bones never join completely (Fig. 411). In acquired cases the same enlargement of the head is not permitted, and that of the ventricles is at the expense of the compressed brain tissue. The cases due to tumours of the 3rd ventricle and posterior fossa need not be considered, as the treatment of the hydrocephalus is the removal of the tumour.



There is another group due to post-inflammatory obstructions of the cerebrospinal fluid pathways. Meningococcal meningitis causes a large number of such cases, but there are others of unknown etiology. The obstruction occurs at the foramen of Majendie, or a stenosis of the aqueduct of Sylvius develops. The diagnosis is generally made by ventriculography, and operation should be undertaken to free the obstruction by dividing adhesions, evacuating cystic collections, or by leaving a seven-day indwelling catheter in the aqueduct. Some gratifying results are obtained; but, unfortunately, in many cases the adhesions re-form after operation and the condition recurs.



FIG. 411

A and B, two views of a baby with hydrocephalus.

Other ingenious operations have been designed for hydrocephalus, as for example :—

Dandy's operation, viz., the removal of the anterior wall of the 3rd ventricle by the same route as for pituitary tumour.

Cerebellar decompression with removal of a peculiar embryonic tissue which often surrounds the medulla in these cases. This peculiar pressure cone produces the Arnold-Chiara syndrome.

Heile's operation consists in anastomosing the pelvis of the kidney to the spinal theca after nephrectomy, thus permitting drainage of the cerebrospinal fluid into the bladder.

Tracy Putman's suggestion is to diathermise the choroid plexuses through a trephine hole over the convexity of the brain, using the ventriculoscope—an instrument somewhat resembling the cystoscope.

A. DICKSON WRIGHT.

R. M. HANDFIELD-JONES.

## INTRACRANIAL TUMOURS

The classical triad of headache, vomiting and papilloedema are produced by increase of intracranial pressure either from the mass of the tumour and its surrounding oedema or from hydrocephalus, in which latter case the growth interferes with the fluid pathways in the 3rd or 4th ventricles. To this triad must be added epilepsy, generalised or local, which is the initial symptom in a large number of cerebral tumours, and even occasionally in cerebellar neoplasms. Change of personality and loss of vision of any type should also be regarded as possible indications of tumour. In children the head is enlarged and gives a "cracked pot" sound on tapping (MacEwen's sign).

*Methods of Localisation*—**A. Clinical Signs.**—1. Above the tentorium these are generally produced by interference with (a) the motor or sensory cortex, shown as hemiplegia or hemianæsthesia, and (b) the visual cortex or optic radiation shown as homonymous hemianopia when the whole radiation is affected, or a quadrantic homonymous defect when only a portion is affected, as occurs, for example, in tumours of the temporal lobe. Involvement of speech or writing centres is also of value. Epileptic seizures of Jacksonian type may indicate a localisation in the motor cortex, while uncinæ fits with aura of smell suggest temporal lobe growths. Incontinence points to frontal tumour, as do mental deteriorations, such as Witzelsucht (inordinate grinning) or Seelenlahmung (loss of culture). Anosmia is a sign of frontal lobe tumours and may affect one or both sides according to the involvement of the olfactory nerves.

2. Below the tentorium there are special signs which in vermis tumours may be absent even in advanced cases. Vertigo and ataxia are common signs; the latter is sometimes shown in the hands by an inability to perform rhythmical co-ordinated movements (dys-diadokokinesis). Involvement of any of the cranial nerves from Vth to XIIth also points to posterior fossa tumour.

**B. X-ray Indications.**—*General Signs* of tumour are those of increased intracranial pressure, namely, atrophy of the posterior clinoid processes (Fig. 416, 2), increase of the indentations of the inner table produced by the convolutions ("copper beaten" skull or convolutional atrophy) and, in children, spreading of the sutures and even increase in size of the foramen magnum. The shadow thrown by pineal calcification, visible after the age of 30 years, may be shifted to one side or other by the presence of a tumour.

*Localising Signs* are of great importance and are likely to pass unobserved unless specially looked for:—

1. Erosion, rarefaction or hyperostosis of overlying bone.
2. Increased vascularity of bone due to the nutrient meningeal vessels of the tumour.
3. Calcification in the tumour or its pedicle.
4. Enlargement of optic foramen in chiasmal tumours.
5. Enlargement of internal auditory meatus in acoustic tumours.

*Ventriculography*, devised by Dandy, is of the greatest value in the localisation of tumours, and although it has a definite mortality

rate thousands of lives have been saved by its help in the accurate planning of operations. Air is introduced into the ventricles through burr holes made 4 cm. to each side of the midline on a line 10 cm. above the external occipital tuberosity. After opening the dura a blunt needle on each side is passed between blood vessels in the direction of the inner canthus of the eye, and at a depth of about 7.5 cm. the body of the ventricle is entered. The patient's head is then turned on one side and the fluid flows from the bottom needle, air entering the upper one. When the ventricles are empty the needles are removed, the wound closed and X-rays taken; from the dilatations and deformities of the ventricles the localisation is made. When the lumbar puncture pressure is not high, air can be introduced through the needle (encephalography), and frequently good ventricular pictures are obtained by this much simpler method.

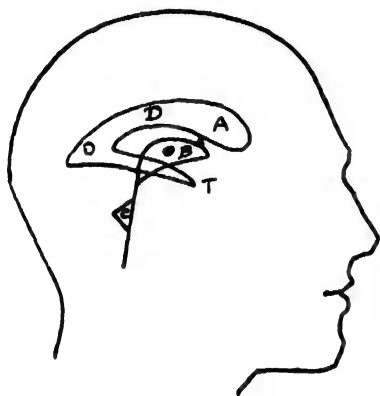


FIG. 412

Diagram showing approximate shapes of ventricles. A, D, O, T, the lateral ventricle (cap of liberty). B, third ventricle (Napoleon's hat), the dot in the centre of which is the middle commissure. C, the fourth ventricle (boy scout's hat).

The ventricular pattern in normal cases is very constant. The side-view can be well memorised by regarding the lateral ventricles as shaped like an attenuated cap of liberty enclosing Napoleon's hat (3rd ventricle) complete with its cockade (middle commissure), and hanging on from the 3rd ventricle a boy scout's hat (the 4th ventricle) (Fig. 412). The anterior horns are filled when the patient lies on his back, and the occipital horns when lying on his face.

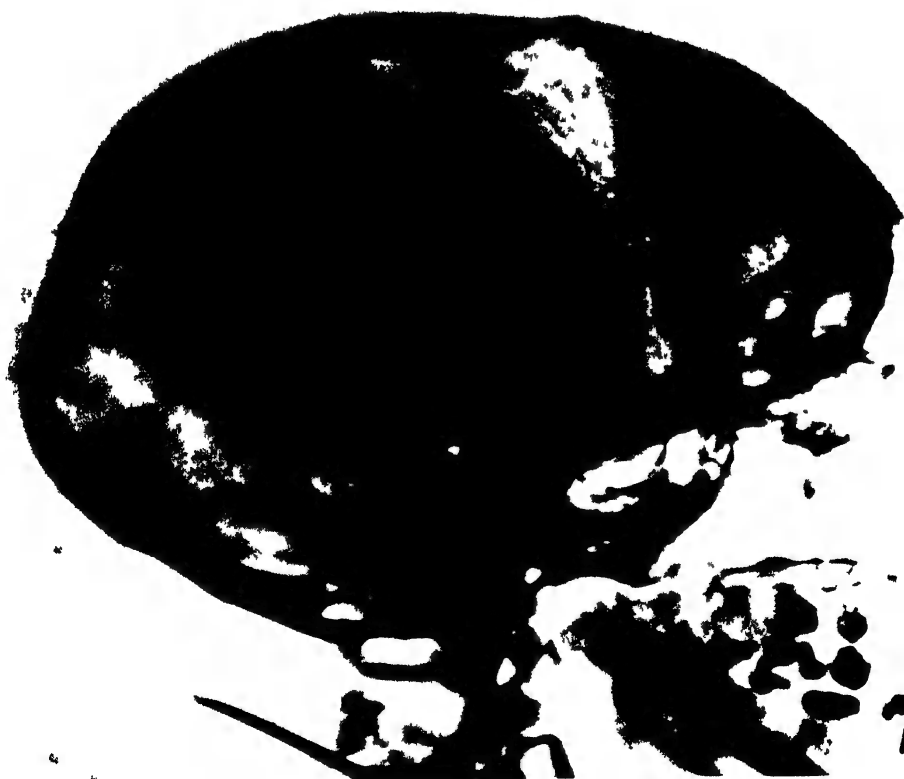
*Arteriography* is another method of localising aneurysms and tumours of the brain; 15 c.c. of warm thorotrast are injected rapidly into the internal carotid artery, exposed under local anaesthesia and, as the injection is finishing, a picture is taken which shows the arteries and, two seconds later, another shows the cerebral veins. Injection of the vertebral artery has also been done for lesions of the temporal lobe and cerebellum. The position of the tumour is determined by the displacements of the main blood vessels or by visualisation of the blood supply of the tumour itself. Filling of an aneurysmal cavity or arteriovenous communication will also give an accurate diagnosis of these conditions (Fig. 413).

*Electro-encephalography* is the latest method of localising brain lesions, especially tumours. The minute action currents of the brain are rendered visible by cathode-ray apparatus, the amplitude of the waves being increased and their periodicity reduced when the electrode lies over a lesion.

Intracranial tumours, of which over 50 per cent. are benign, provide the bulk of all surgical work of the head. As in few other parts of the body do benign growths preponderate to such a degree, the results of this type of surgery are very satisfactory. It is clear, however, that exact localisation is all-important.



A



B

FIG. 413

### TUMOURS OF THE BASE OF THE BRAIN

Comparatively easy is the diagnosis of many of these growths, because they produce such definite syndromes, owing to their proximity to the cranial nerves. The *First Group* consists of tumours in the neighbourhood of the optic nerves. These are :—

1. Pituitary adenomata—with eosinophil, basophil or neutrophil cells.
2. Suprasellar cysts.
3. Suprasellar meningiomata.
4. Gliomata of the optic chiasma.

**Pituitary Tumours** (Fig. 414) betray their presence by their endocrine effects and by the pressure they exert upon the optic nerves. The visual field changes are characteristic, being due to chiasmal pressure. The decussating fibres from the nasal halves suffer, and so the temporal fields are lost at first (bi-temporal hemianopia), but later complete blindness may result, and once optic atrophy has supervened all hope of recovery of sight is gone. These pituitary tumours are of the following types :—



FIG. 414

A pituitary tumour.

- (a) Chromophil adenoma, causing gigantism or acromegaly, often optic nerve changes, and sometimes a peculiar cachexia after many years.
- (b) Basophil adenoma, causing the syndrome of Cushing—high blood pressure, cyanosis, hirsuties and adiposity. This disease is generally fatal before the adenoma reaches any size or causes nerve pressure. It may be ameliorated by implanting radon seeds in the pituitary fossa.
- (c) Chromophobe adenoma, showing marked optic nerve pressure and less noticeable endocrine changes. It is often cystic. The endocrine changes are those of pituitary inadequacy, viz., amenorrhœa, impotence, diabetes insipidus, etc., and are due to the pressure of the adenoma on the functioning gland.

**Congenital Cysts in the Suprasellar Region** arise in the pharyngeal diverticulum from which the pituitary develops. These cysts are best known in children, but they may not produce symptoms till even as late as 70 years. They are multiloculated and generally have a solid root of characteristic adamantinoma cells. In children they often cause signs of pituitary inadequacy (Fröhlich's fat type or

Lorain's infantile type). The visual field changes, though often bi-temporal, are sometimes very irregular. These cysts sometimes press to such an extent on the floor of the 3rd ventricle that hydrocephalus and papilloedema follow.

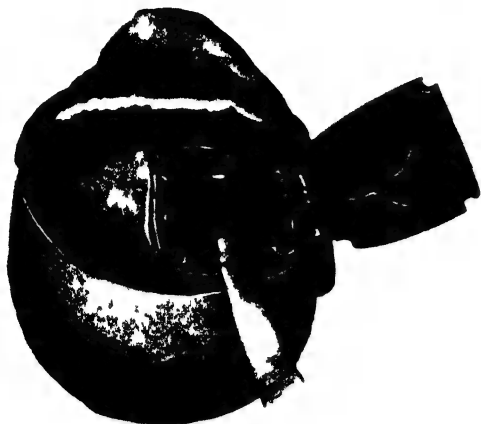


FIG. 415

Exposure of pituitary region. Incision of dura along line of sphenoidal wing not shown.

*Prognosis.*—Operations for pituitary tumour have the lowest mortality of all intracranial procedure, which is as it should be, since they are usually performed only to save sight and not life. The dangerous cases are those in which there is a large extension of the growth into the mid-brain or frontal lobes.

*Treatment.*—These tumours are now always removed through the transfrontal approach, preferably on the right side (Fig. 415). The tumour is then incised

between the optic nerves and its contents emptied if cystic, or sucked and curetted if solid. After collapse of the neoplasm its wall can often be removed as there are only feeble attachments to surrounding vessels. If the capsule of an adenoma must be left behind, it is a good practice to place a small

1, normal sella; 2, atrophy of posterior clinoid process through increased intracranial pressure from any cause; 3, chromophil adenoma, acromegalic thickening of clinoid processes delays extension of adenoma upwards; 4, chromophobe adenoma quickly bursts out of the sella; 5, "Gourd" sella of glioma of optic chiasm; 6, suprasellar cyst, diffuse calcification in solid part of tumour and "comet's tail" in wall of cyst; 7, small hyperostosis marks root of meningioma growing from anterior clinoid process.

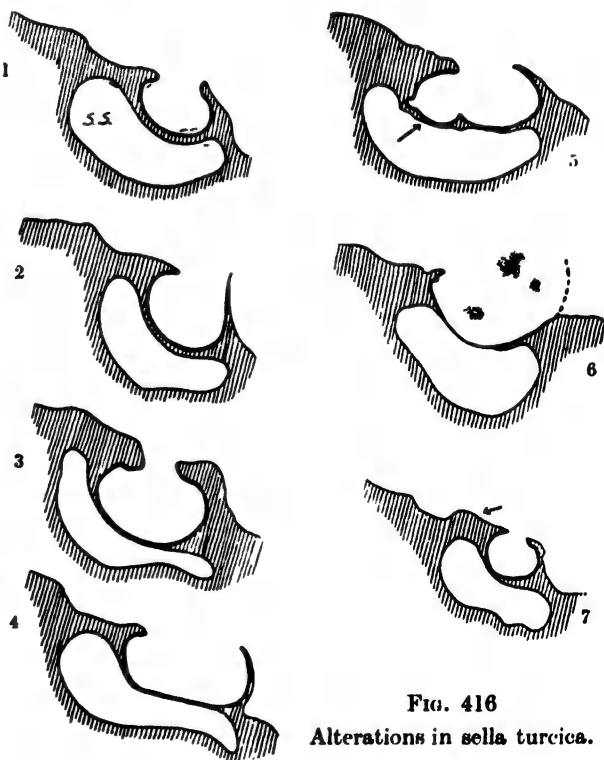


FIG. 416

Alterations in sella turcica.

pledget of wool soaked in Zenker's solution inside it at the end of the operation for two minutes; any remaining adenomatous tissue is destroyed by this powerful fixative.

**Suprasellar Meningioma** produces a bi-temporal hemianopia without

signs of endocrine dysfunction or enlargement of the sella turcica. They declare themselves so early in their career that they are generally small and easily removed. Since they are benign they have a low mortality rate and do not recur if completely removed.

**Glioma of the Optic Chiasma** gives a syndrome of progressive blindness with optic atrophy and sometimes appearance of a tumour on the optic disc. These cases are practically always inoperable. Apart from the different clinical syndromes of tumours adjacent to the chiasma, the separation of the various types can be made from characteristic X-ray changes in the region of the sella (Fig. 416).

*The Second Group* includes growths in the neighbourhood of the olfactory groove, corpora quadrigemina and auditory nerve.

**Olfactory Groove Meningiomata** grow to a very large size before declaring themselves, because they grow upwards into the "silent"



FIG. 417

Auditory nerve tumour.

regions of the frontal lobes. They often produce the characteristic Foster Kennedy syndrome of optic atrophy on the affected side and papilloedema on the other. Anosmia on one or both sides is, of course, a constant feature of these tumours. A wide exposure is necessary for their removal as they often reach a gigantic size.

**Tumours in the Neighbourhood of the Corpora Quadrigemina** produce changes in eye movements, inability to look upwards and ptosis being the main signs. Pineal growths form the main bulk of these cases and occur as either tumours of the pineal cells, chlosteatomata or true dermoid cysts.

**Auditory Nerve Tumour** (acoustic neuroma) (Fig. 417) produces a very clear-cut syndrome. Firstly there are the general signs of a cerebellar tumour (ataxia, drunken gait, vertigo, etc.) and, secondly, the localising nerve signs on the affected side (nerve deafness, facial palsy of lower motor neurone type and facial anæsthesia). The combined involvement of the last two nerves causes the corneal

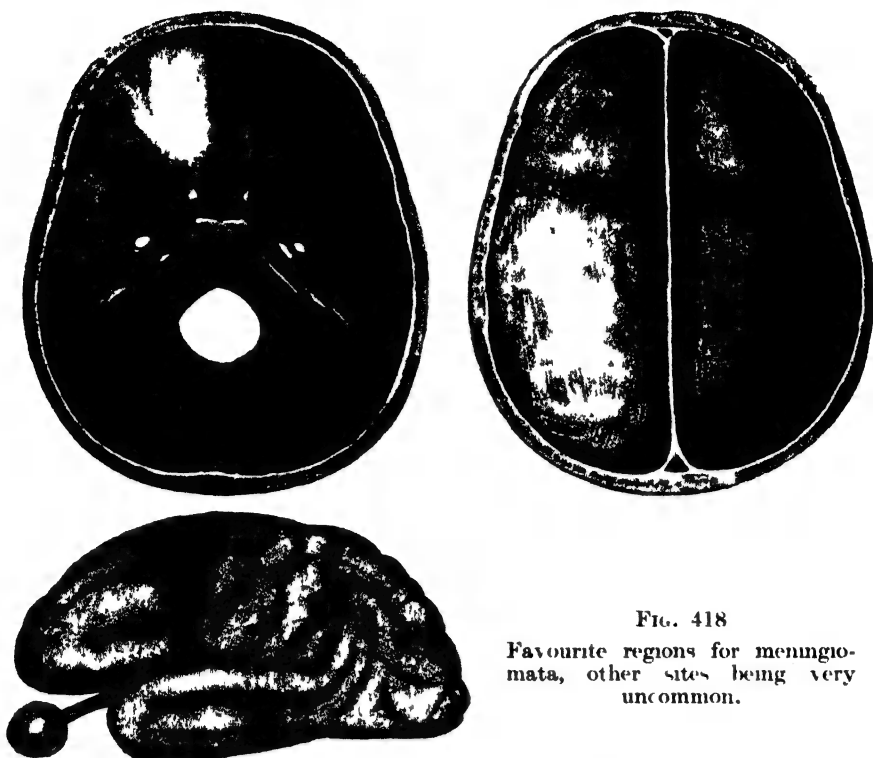
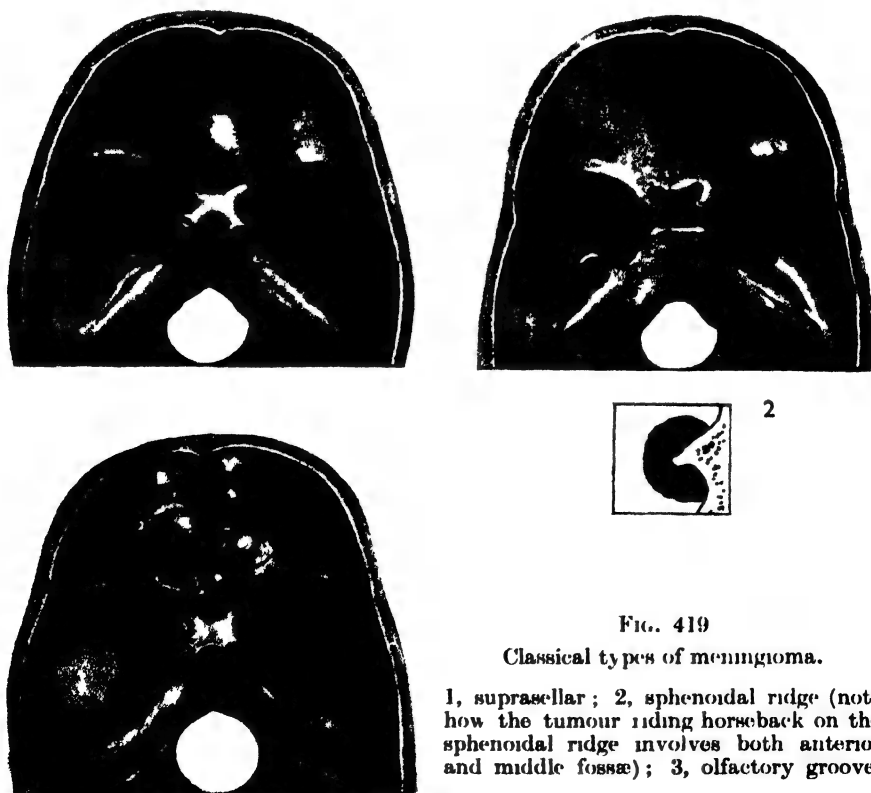


FIG. 418

Favourite regions for meningioma, other sites being very uncommon.



2

FIG. 419

Classical types of meningioma.

1, suprasellar; 2, sphenoidal ridge (note how the tumour riding horseback on the sphenoidal ridge involves both anterior and middle fossae); 3, olfactory groove.



reflex to be lost early on the affected side. The skin papillomata and pigmentation of von Recklinghausen's disease are sometimes associated with acoustic neuroma. The age incidence is from 25 to 50 years, and it is interesting that the majority of cases are female.

### TUMOURS OF THE MENINGES

**Meningiomata** constitute about 15 per cent. of cerebral tumours and, being benign, they provide the greatest successes of brain surgery. They derive as a rule from the dura, and show a characteristic histology of whorls and columns of endothelial cells with frequent changes, such as myxomatous, fatty and calcareous degenerations. The word "psammoma" is used to indicate a meningioma in which much calcification has taken place. Meningiomata probably originate from arachnoid villi and cell clusters, and therefore have a corresponding localisation as shown in Fig. 418. It will be seen that they follow a roughly cruciate distribution, both on base and vertex, well-known

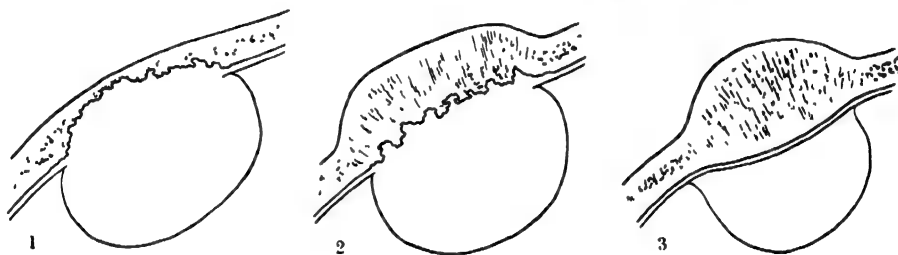


FIG. 420

Alterations in bone over a meningioma.

1, erosion ; 2, erosion and hyperostosis ; 3, hyperostosis due to invasion of bone.

types being olfactory groove, suprasellar, sphenoidal ridge and parasagittal meningiomata (Fig. 419).

These tumours reveal themselves sometimes in plain X-ray pictures of the skull by increased vascularisation of the adjoining bone, calcification of the pedicle of the tumour and various secondary changes in the overlying bone, such as rarefaction, erosion, hyperostosis, or combinations of these (Fig. 420).

In the absence of these characteristic X-ray changes the presence of a meningioma can often be anticipated from the slow march of symptoms and also from the frequent association of epilepsy with these tumours. Operative removal is as a rule straightforward, but is sometimes rendered dangerous by their great vascularity, and one or more blood transfusions may be necessary during the operation. Injury of large important vessels adjoining the tumour may cause a fatality, as in suprasellar and sphenoidal ridge types.

### TUMOURS OF THE BRAIN

**Gliomata** are not uniformly malignant tumours. Careful pathological classification has resulted in four main groups being described. These, with their favourite sites of occurrence, are :—

1. Medulloblastoma : vermis of cerebellum of young children.
2. Spongioblastoma multiforme : hemispheres of adults from 30 to 50 years.
3. Oligodendroglioma : frontal lobes of young men and women (calcification frequent).
4. Astrocytoma : vermis of cerebellum of children and hemispheres of adults from 30 to 50 years.

Other varieties of gliomata have been described, but these are the main types, and they only will be described.

MEDULLOBLASTOMA is a very cellular highly malignant tumour of quite young children. The growth, either wholly or in part, is removed through a vertical incision in the vermis, and radium or X-ray therapy is applied through the occipital decompression opening after the wound



FIG. 421

Spongioblastoma multiforme.

is soundly healed. Response is prompt ; but, unfortunately, recurrence is rapid and most cases are dead within two years. This tumour is notable as being the only one metastasising through the cerebrospinal fluid and producing secondary growths of the spinal cord.

SPONGIOBLASTOMA MULTIFORME (Fig. 421) is an intensely malignant rapidly growing tumour against which no surgery or radio-therapy avails. The present custom is not to operate when biopsy by brain needling reveals this tumour. If, at operation, this typical diffuse hæmorrhagic multicystic tumour is found, it is customary to remove all the tumour possible and then sew up tightly without a decompression, so that the patient will keep well for a few months and then die rapidly ; thereby the miserable *degringolade* associated with decompression is avoided. Rarely a lobectomy will accomplish a complete removal of this growth when it occurs in the frontal, temporal or occipital lobe.

OLIGODENDROGLIOMA is a less common tumour which often lends itself to complete removal of the frontal lobe, a condition not incompatible with normal existence and intelligence even when the left lobe is ablated.

**ASTROCYTOMA** is to a great extent a midline cerebellar tumour of young children. It shows clear demarcation and frequently has an associated cyst. Removal is not followed by recurrence and the results are good. The astrocytoma of the hemispheres of adults is not so clearly encapsuled, and the operative results are correspondingly worse.

### VASCULAR AND OTHER TUMOURS

**Angiomata** are a small group, and vary from huge racemose arterio-venous aneurysms through compact highly vascular tumours to small growths in the walls of large cysts. These latter occur usually in the cerebellum and, when associated with retinal angiomata and a cystic pancreas, produce the syndrome known as Lindau's disease. The treatment of these tumours is often difficult because of their vascularity, but the compact and cystic varieties can be satisfactorily removed. Large arterio-venous aneurysms are best treated by carotid ligation.

**Cerebral Aneurysms** are generally due to congenital defects in the arterial wall (Fig. 413). They are usually basal in association with the circle of Willis and produce characteristic pressure effects on the cranial nerves. These often appear suddenly because of leakage and, when in the anterior part of the circle, cause sudden blindness, complete ophthalmoplegia and facial pain (ophthalmoplegic migraine); when posterior, facial paralysis and deafness may appear.

The anterior aneurysms are well treated by carotid ligation. The posterior ones have been occasionally cured by opening them and stuffing the cavity with a muscle graft.

**Tumours of the Choroid Plexuses** are either colloid cysts or papillomata. Cases do well after removal of these lesions.

**Ependymomata** are tumours growing from the lining of the ventricles. The commonest type grows in the 4th ventricle and gives symptoms of cerebellar tumour. A large tail sometimes hangs through the foramen magnum, and thus the signs may be those of a spinal tumour. They are benign and of remarkably slow growth.

**Cholesteatomata**, "pearly tumours" or epidermoids, result from inclusion of an area of skin epithelium in the brain during embryonic development. Down the years of life the desquamation of the skin lining slowly expands the tumour till symptoms are produced. If accessible, their operative treatment is very satisfactory. The contents are scraped out and the cyst lining removed.

### SURGICAL TECHNIQUE FOR INTRACRANIAL OPERATIONS

Intracranial operations in earlier days were conducted at incredible speed and the blood, which was shed profusely, was washed away with a constant stream of saline playing over the wound. It is now realised that speed is not essential and, in most quarters, has given way to a more deliberate technique with conservation of blood and brain tissue.

**SUPRATENTORIAL OPERATIONS.**—The scalp is incised according to the situation of the tumour. In frontal and pituitary tumours the incisions used are shown in Fig. 422, and the scalp is turned down before the bone is drilled, whereas in parietal and occipital lesions (Fig. 423) the bone is turned down with the scalp flap. The skin incision is made with pressure applied

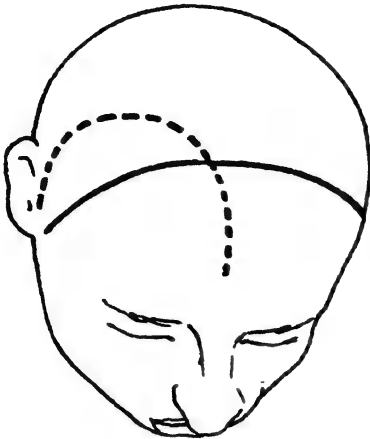


FIG. 422

Alternative incisions for frontal and pituitary operations.

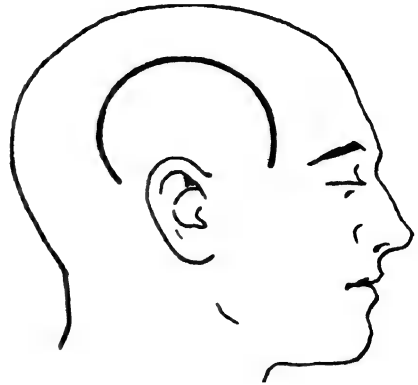


FIG. 423

Incision for temporal tumours. For parietal growths the incision is placed a little higher.

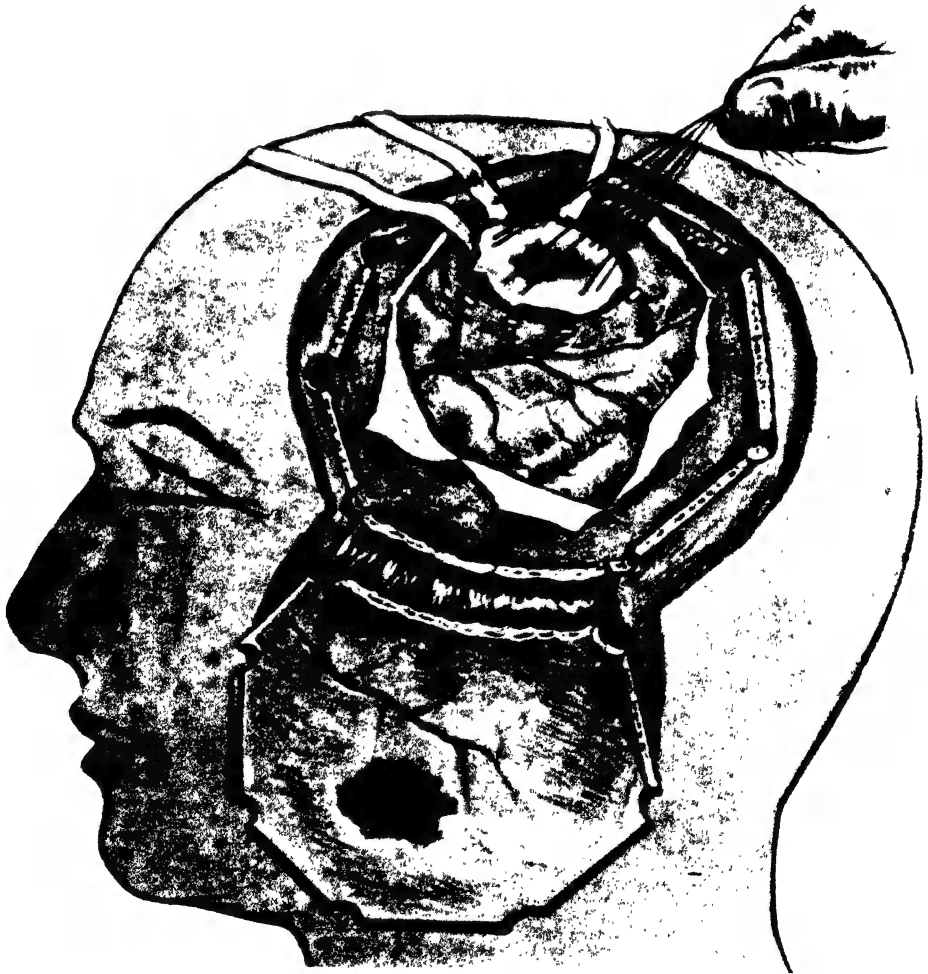


FIG. 424

Removal of meningioma by combination of traction and cotton-wool strips

on each side of the wound and the galea is seized with forceps before the pressure is released, so that when these forceps are angled back over the cut edge all oozing is checked. The pericranium is then incised with the diathermy needle and the periosteum pushed back so as to make room for the burr holes which are generally five in number and made with an electric or hand burr. These holes completed, a Demartel's guide is passed from one to the next, followed by a Stille's modification of Gigli's saw; the holes are then joined up. The bone flap is next broken back on its temporal muscle attachment and the dura exposed. The large dural vessels are tied with silk stitches and smaller bleeding points controlled by touches with the diathermy. If the brain pressure is very high, it should now be reduced by inserting a brain needle into the ventricle or the cyst, so often associated with tumours, when by withdrawal of fluid the pressure is so reduced that the dura can be safely opened without brain herniation. Sometimes fluid cannot be found and the opening of the dura is fraught with danger of damage to the brain cortex. It may be necessary to inject intravenously 100 c.c. of 15 per cent. sodium chloride in such a case and wait half an hour before rapidly incising the dura.

Once the cortex is exposed, the tumour may be seen or its presence inferred by the flattened convolutions overlying it. If seen, it is separated by gentle dissection with cotton pledgets from the surrounding brain and, by traction with silk stitches inserted through the tumour itself, is gently dislodged from its bed (Fig. 424). If the tumour is sub-cortical, its relations are ascertained by inserting a brain needle at several sites and then "uncapping" the tumour by excising a medallion of cortex over it (Fig. 425). Bleeding from the cortex is prevented by putting silk ligatures around, or silver clips on, the larger vessels and coagulating a track in the cortex before incising with the diathermy needle. The tumour is then removed by gentle cotton-wool dissection. It sometimes happens that the tumour is too large to be removed *in toto*; in these cases the centre of the tumour is "cored out" with the diathermy cutting loop, or sucked out, and the remaining shell can then be withdrawn from the surrounding brain.

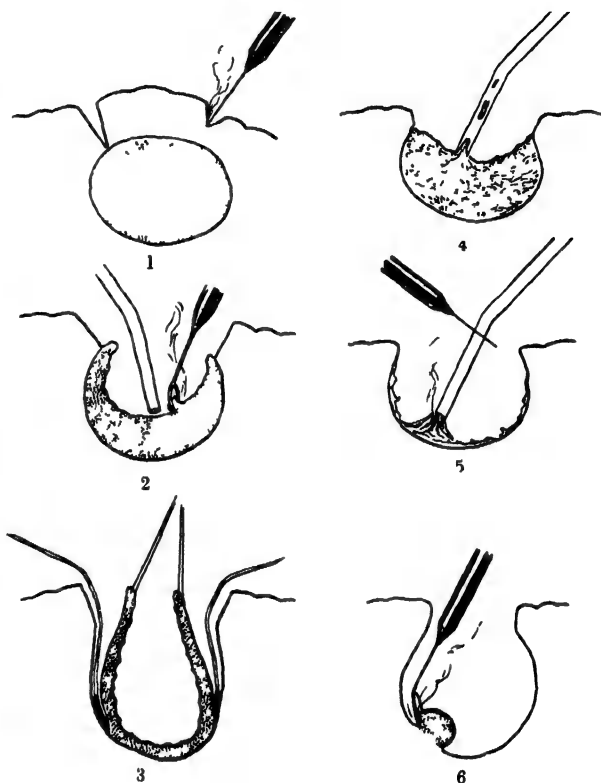


FIG. 425

Methods of removing cerebral tumours.

1, uncapping it; 2, scalloping it out with cutting diathermy loop; 3, removing capsule by traction and cotton-wool dissection; 4, sucking out the soft tumour; 5, coagulating the vascular network, using a metal sucker tube; 6, excising mural nodule from the cyst.

After removal of the tumour the bed is systemically hæmostased. The vessels are picked up with dissecting forceps and sealed with the coagulation current. Silver artery clips may also be used, and a neat way of securing difficult vessels is shown in Fig. 426, the vessels being drawn into the mouth of a metal suction tube and coagulated with the diathermy current. Muscle or fibrin grafts can also be used for stopping difficult vessels. When all is dry, the flap is returned to place and fixed with a silver wire; the muscle and galea are then sutured with fine waterproofed silk and the skin closed

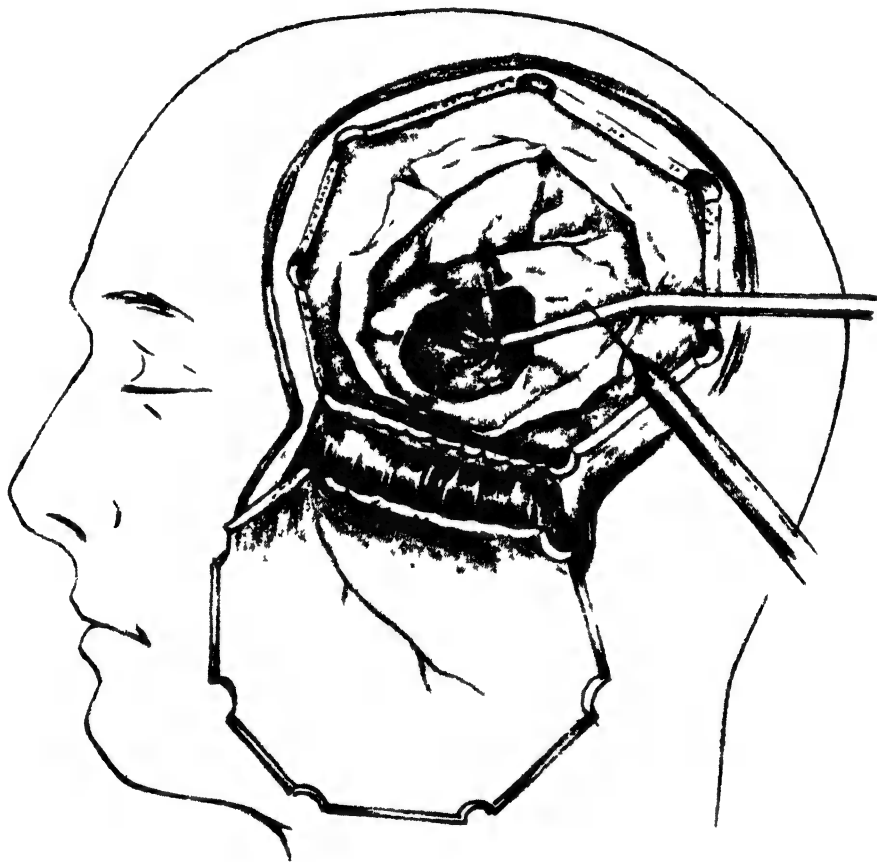


FIG. 426

Method of exposing temporal lobe tumour which has been removed with a metal sucker and vessels coagulated from time to time.

with silkworm gut stitches. Twenty-four to seventy-two hours drainage may be used into a large tumour cavity or under an extensive flap.

*After-treatment.*—The skin stitches are cut in forty-eight hours and removed in seventy-two hours; the wound does not gape because of the careful silk suturing of the galea aponeurotica. Post-operative increase of intracranial pressure can be dealt with by ventricular or lumbar punctures and the administration of hypertonic solutions. Progressive post-operative compression should be met by reopening the flap and removal of the fresh blood clot, the wound being reclosed meticulously and a small drain left in.

It is sometimes necessary to be discreet and stop the operation before complete removal of a tumour owing to blood loss or shock. In such a case the closure has to be most carefully made because pressure still exists and

herniation would be a disaster, as would even the mildest wound sepsis because of the danger of a subsequent operation in the presence of infection.

**CEREBELLAR OPERATIONS.**—The cerebellum is exposed by a curved incision (Fig. 427) in adults and a straight vertical incision in children. The muscles are stripped off the occipital bones with diathermy and raspatory, and the emissary veins plugged with wax. The bone is then drilled and nibbled away, the posterior margin of the foramen magnum and the occipital bones being removed until the lateral sinus is exposed. The posterior border of the atlas and even axis must also be excised if the cerebellum has been forced as a pressure cone through the foramen magnum. In acoustic neuroma the removal of bone is done only on one side. The dura is now incised by a number of radiating incisions; but, before doing this, pressure should be reduced by emptying the lateral ventricles, the cisterna magna and a cyst, if this is present.

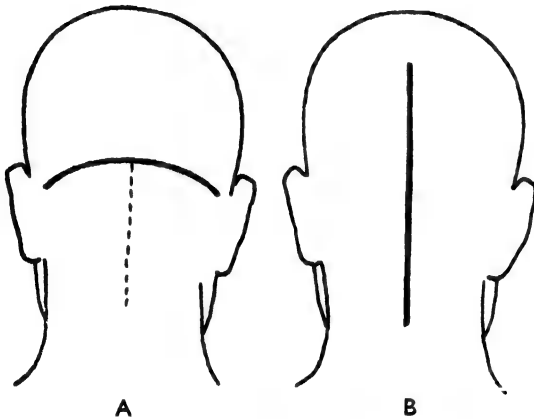


FIG. 427

Incisions for cerebellar operations.

A, for adults—dotted line incision is optional to gain access to upper vertebræ; B, for children.

When the cerebellum is exposed, the tumour is attacked, if it can be seen. It is sometimes necessary to incise the vermis or cerebellar lobe to expose the tumour, which is then removed as previously described. If a cyst is found, its wall is carefully examined and in one place a nodule will be found. This "mural nodule," if carefully removed, will prevent the recurrence of the cyst, since it is the tumour itself, an angioma or astrocytoma as a rule (Fig. 428).

After cerebellar operations, increase of pressure may be very troublesome and sometimes oedema of the medulla

occurs because of the operative disturbance. These complications can be guarded against to some extent by leaving a small catheter in the lateral ventricle for three to four days, and by being careful to remove enough bone from the atlas, and even axis, to give freedom from pressure.

**THIRD VENTRICLE OPERATIONS** are now frequently performed, the great precision of ventriculography enabling tumours here to be diagnosed. When they lie in the anterior part of the ventricle, exposure is made by excising a medallion of cortex from the right frontal lobe, and widely opening the lateral ventricle, when by enlarging the foramen of Monro the tumour can be removed. The common growth in this anterior situation is the colloid cyst arising from the choroid plexus. It is a benign lesion, and its removal effects a complete cure. The tumours in the posterior part of the ventricle are exposed by a right-sided occipital flap; the right occipital lobe is retracted outwards after emptying the ventricular fluid by needle. The tentorium and the splenium are now incised and the tumour exposed. Most neoplasms in this region derive from the pineal body and are very malignant, but an occasional embryonic tumour in this situation makes exploration worth while.

**OPERATIONS UPON THE CRANIAL NERVES.**—The optic nerves and chiasma are often surrounded by adhesions, the result of trauma, syphilis or adjacent sphenoiditis or ethmoiditis. This important disease (chiasmal arachnoiditis)

is often overlooked and complete blindness may supervene, generally with the diagnosis of retrobulbar neuritis. The arachnoiditis may take the form of fibrous bands, soft granulation tissue, cystic collections or dense encapsulating fibrous tissue. Careful stripping of the nerves from this tissue produces most gratifying results.

*The Trigeminal Nerve*, the seat of the common complaint of tic douloureux, most frequently requires operation. The injection of the

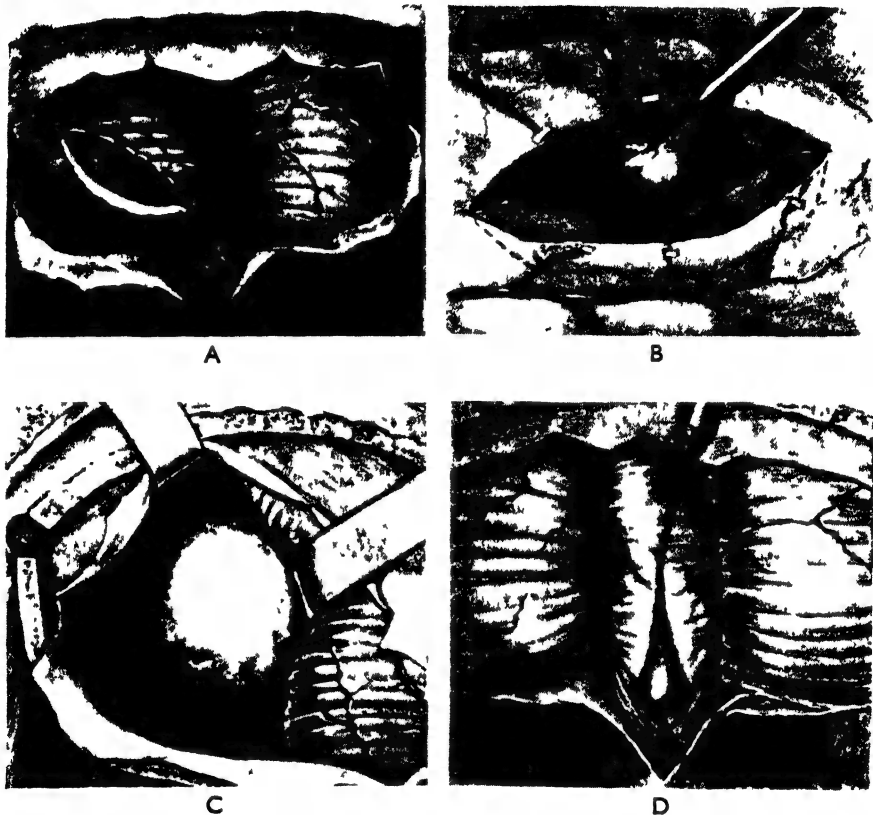


FIG. 428

Methods of approach to cerebellar tumours.

A, cerebellum freely excised to expose the tumour in one lobe ; B, the removal of mural nodule with diathermy ; C, exposure of tumour in cerebello-pontine angle. Removal of fluid from ventricles and basal cisterns gives the necessary room ; D, incision of expanded vermis to expose mid-line tumour.

nerve with alcohol is still a favoured procedure, although in many quarters it is felt that the division of the sensory route behind the ganglion is a more precise operation.

**Alcohol Injection** is best done by Hartel's route. A long needle is entered 2 cm. outside the angle of the mouth ; pointing towards the lambda it is inserted to a depth of 8 to 9 cm. when the foramen ovale is reached and signalled by a shoot of pain along the lower jaw. The needle is then advanced 1 cm. through the foramen ovale and the ganglion injected with a few drops of novocain first and then  $\frac{1}{2}$  c.c. of 90 per cent. alcohol.

**Operation.**—A temporal route (Frazier's operation) is the favoured one. The incision and bone removal are shown in Fig. 429. The dura covering the middle fossa is then elevated till the middle meningeal artery is seen



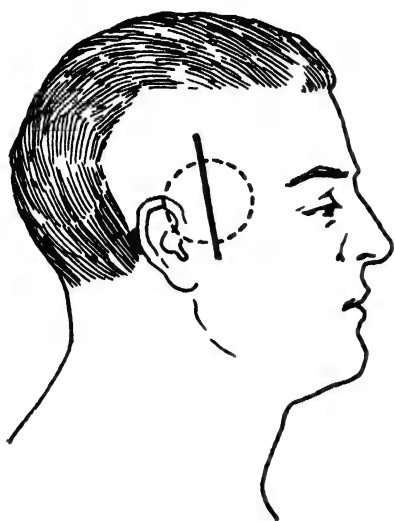


FIG. 429

Incision and area of bone removed for exposure of gasserian ganglion.

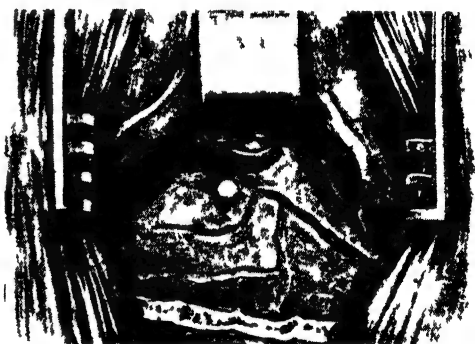
coverings and the fluid sucked away; the cerebellar lobe is lifted up, the Vth nerve visualised near the apex of the petrous and divided or crushed with a silver clip. Dandy claims that by this method the face still has some sensation left, although pain cannot be felt. Sjoquist has gone further and suggested a division of the quinto-spinal tract in the medulla and thus achieved a preservation of common sensation in the face, but with loss of pain and temperature sense—an ideal result.

The *Auditory* nerve is now frequently divided for Ménière's disease (paroxysmal aural vertigo). The nerve is divided, on the side on which tinnitus and hearing are worst, by the same cerebellar route as for the Vth nerve. If the hearing is not much impaired the vestibular (anterior) half of the nerve is divided, leaving the cochlear half intact.

The *Glossopharyngeal* nerve is sometimes divided by the cerebellar route for neuralgia of this nerve. It is also a very great help to divide this nerve, together with the fifth, in cases of severe pain in carcinoma of tongue and fauces.

emerging from its foramen spinosum. The foramen is packed with a tiny wool pack and the artery divided. The foramen ovale can then be exposed by raising the dura further and, working back from this, the ganglion is exposed in its capsule. When well cleared, the cave of Meckel is opened behind the ganglion and its sensory root is raised on a hook and divided, taking care to leave the motor root intact (Fig. 430). If the neuralgia occurs only in the lower two divisions it is wise to leave the upper one-sixth of the root so as to retain sensation of the eye and forehead, a valuable safeguard against injury to the eye as a result of corneal anæsthesia.

The posterior route is favoured by Dandy. A small disc of bone is removed behind the mastoid process and the dura opened. The basal cisterns are then emptied by tearing open the arachnoidal away. There is now plenty of room to



A



B

FIG. 430

A, exposure of gasserian ganglion (note foramen spinosum plugged with wool); B, shows preservation of ophthalmic division and the motor route.

### HERNIA CEREBRI

This term should not be applied to those protrusions of brain matter through a deliberately planned decompression opening, in which case the scalp is intact over the swelling. Hernia cerebri implies the prolapse of brain substance through an opening in both skull and scalp, the actual visible tissue consisting of inflamed cerebral matter. Such a condition must inevitably be an indication of increased intracranial tension, for if this was absent the inflammation of the prolapsed tissue would subside and the brain retire within the skull. It occurs as a result of penetrating injuries which have infected the skull as well as the brain, in which case the prolapsed tissue is largely composed of exuberant granulations rather than the brain itself. It will also be seen as a late result of formal decompressions, when either the suture line yields to increasing pressure or the skin is involved in the spread of the growth.

*Treatment* must be directed towards the underlying increased intracranial tension. In those cases in which sepsis is the main etiological factor the surface should be treated with dehydrating dressings, such as absolute alcohol, hypertonic saline or glycerin and magnesium sulphate paste. These patients are likely to suffer from various forms of epilepsy, and it may be necessary at a later date to excise the scar, free the brain from adhesions and repair the defect in both skull and scalp. If the cause of increased intracranial tension is progressive and not amenable to successful removal, no treatment is of any avail and the hernia must be regarded as a terminal manifestation. Thiersch skin grafts applied to the surface of the fungus take well. The clean epithelialised surface so obtained is a great advantage and permits further operative treatment in a clear field.

A. DICKSON WRIGHT.

## CHAPTER XLII

### THE DISEASES OF THE SPINE AND SPINAL CORD

**SURGICAL ANATOMY.**—The vertebral column consists of seven cervical, twelve dorsal, and five lumbar vertebræ, together with the fused sacral and coccygeal elements below. The intervertebral discs of elastic fibrocartilage increase the range of movement of the column as a whole and act as “dampers” to the jolts and jars to which the spine may be subjected. A considerable range of flexion and extension is possible, as are lateral bending and rotation to a lesser degree. In childhood the column presents one slight continuous curve with an anterior concavity; in adults this concavity persists in the dorsal region, but in both the cervical and the lumbar portions the curve has been changed so that the concavity faces backwards. The whole spinal column, therefore, has an S-shaped curve.

*Surface Markings.*—The following points have an important application to clinical examination :—

The cricoid cartilage is at the level of the 6th cervical body.

The spinous processes of the 7th cervical and 1st dorsal are easily palpable.

The root of the spine of the scapula corresponds to the 3rd dorsal spinous process.

The inferior angle of the scapula (at rest) is opposite the 7th dorsal spine.

The line joining the highest points of the iliac crests crosses the interval between the 3rd and 4th lumbar vertebræ.

The line joining the posterior superior iliac spines passes over the 2nd sacral spine.

The spinous processes bear varying relations to their own vertebral bodies according to the amount of slope they possess, so that

All the cervical and 1st, 2nd, and 3rd dorsal spines correspond to their own bodies.

The 4th to the 7th dorsal spines are opposite the bodies of the vertebra next below.

The 8th to the 12th dorsal spines are at the level of the lower border of the vertebra next below.

All the lumbar spines are opposite their own bodies.

The spinal cord lies within the vertebral canal and is enclosed in a prolongation of the three membranes which surround the brain. Their relative arrangement is exactly similar, and the subarachnoid space is likewise filled with cerebrospinal fluid. The dura mater ends at the level of the upper border of the 3rd sacral segment, whereas the spinal cord terminates at the level of the intervertebral disc between the 1st and 2nd lumbar vertebræ, below which level the nerve trunks of the cauda equina occupy the spinal canal.

Physiologically the spinal cord retains its developmental arrangement and is consequently composed of a number of segments, from each of which

arise a pair of spinal nerves. These nerves, having both anterior and posterior roots, leave the bony canal through the intervertebral foramina, but since the spinal cord is so much shorter than the vertebral column it must necessarily follow that the nerves run downwards in the canal before reaching their appropriate openings; further, this obliquity of their course increases as the cord passes downwards, until eventually the 3rd, 4th, and 5th lumbar and all the sacral segments are collected together in the conus medullaris, which correspond in level to the 11th and 12th dorsal and 1st and 2nd lumbar vertebræ.

The surface markings of the various spinal segments may be given somewhat roughly as follows:—

In the cervical region add one to the number of the vertebra for each cervical segment.

In the upper six dorsal add two.

In the lower six dorsal add three to the number of the vertebra concerned.

*Development.*—The spinal cord is developed very early from a median longitudinal groove in the dorsum of the embryo. This epiblastic groove deepens and on either side of it appear lateral ridges of mesoblast and epiblast. The neural groove becomes buried beneath the surface by the fusion of the lateral ridges. It is represented by the central canal of the cord, which is formed from the epiblastic inclusion, while the laminæ, spinous processes and post-vertebral muscles are developed in the mesoblast of the lateral ridges.

## CONGENITAL ANOMALIES

### SPINA BIFIDA

Spina bifida, as its name implies, is a developmental defect in which the spinous processes have not been formed owing to the failure of the laminæ to meet and fuse in the midline behind. This bony defect may, or may not, be accompanied by anomalies of development of varying degrees in the spinal cord. It occurs chiefly in the lumbar region, but is occasionally present in the cervical part of the column and, with the exception of spina bifida occulta, all varieties are recognised at birth. In many infants there will be associated congenital anomalies, some the direct result of the spinal cord lesion, such as paralytic talipes equinovarus, others unconnected with it, *e.g.*, cleft palate, imperforate anus, etc.

The *etiology* is unknown, though in a few instances a familial tendency exists. There appears to be a connection between it and hydrocephalus, for not only do the two conditions coexist but the successful closure of a meningocele may be ruined some months later by the development of an internal hydrocephalus.

*Varieties of Spina Bifida* (Fig. 431):—

**A. Spina Bifida Occulta** is the only variety in which there is no external swelling and which frequently is not recognised until the second or third decade of life. The failure of closure of the laminæ is present, but there is no gross involvement of the cord or of its membranes. On the surface some patients will show a revealing sign, such as a pilonidal sinus, nævus, subcutaneous lipoma, or hairy mole. The posterior surface of the spinal membranes may be

attached to the skin by a fibrous cord—the ligamentum reuniens. The edges of the bone defect may be palpable, but in many patients the gap is so small that an X-ray is needed for its recognition.

**B. Meningocele.**—The cord is normally developed, but there is a herniation of the membranes through the bony defect. A sac of varying size is covered with skin and contains cerebrospinal fluid.

**C. Meningomyelocele.**—A similar protrusion of membranes is present, but in this type either the cord itself or some of the spinal nerves pass in and out of the sac. This nerve tissue is either free or adherent to the wall of the sac.

**D. Syringomyelocele** is a condition in which the central canal is dilated, and the nerve tissue which occupies the sac is that lying posterior to the central canal, while the anterior part is normally situated within the vertebral canal.

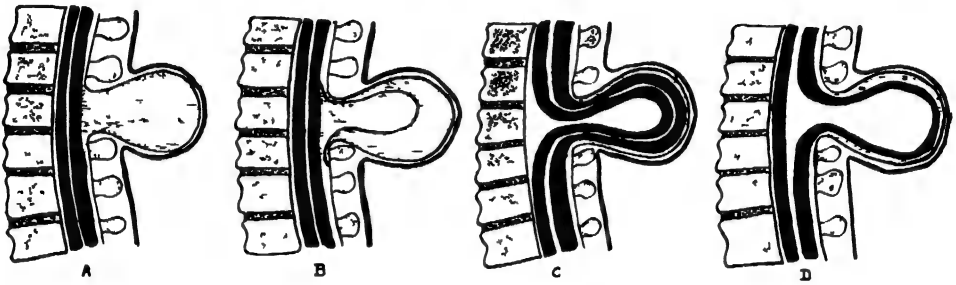


FIG. 431

Varieties of Spina Bifida.

A, meningocele; B and C, meningocele, the former having a nerve trunk and the latter the whole cord in the sac; and D, syringomyelocele

**E. Myelocele.**—This represents a total failure of invagination of the original epiblastic groove, so that the central canal opens on the surface, being surrounded by an area of neural tissue spread out fanwise and fusing at its periphery with normal skin.

It is probable that the myelocele is the commonest of this rare example of developmental anomalies, but the meningocele is most frequently seen in clinical practice.

**Clinical Signs.**—**A. Spina Bifida Occulta.**—If the skin over the defect is normal, no indication of the presence of the latter will be seen during the first decade; indeed, a great many patients of all ages are shown by X-rays to have these bone defects without symptoms. Clinically the conditions associated with it are concerned either with the feet or the urinary bladder. Mild degrees of paralytic talipes or pes cavus should be suspected of being associated with a spina bifida occulta; similarly, lack of bladder control, such as nocturnal enuresis in older children, and frequency or actual incontinence in young women, in the absence of urinary disease, can often be referred to the bifid spine. These manifestations are due to the drag of the ligamentum reuniens on the spinal coverings, and in some cases may be relieved by its removal.

**B. Myelocele (Fig. 432).**—The soft, red area in the midline of the back in the lumbar region fusing with normal skin at its periphery

and having a central orifice, from which leaks cerebrospinal fluid, cannot be mistaken. It is incompatible with life.



FIG. 432

Myelocoele The central canal may be seen opening on the surface at the apex of the defect

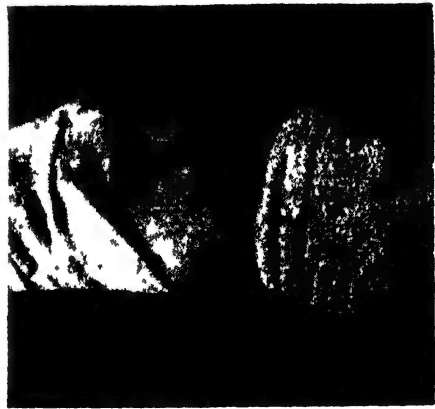
C. The other varieties all present a swelling in the midline of the back and differ only in the amount of nerve tissue contained within them (Fig. 433). The swelling increases in size when the child coughs, strains or moves about; it can also be reduced, though this is a dangerous proceeding, since it may cause convulsions. Some idea of the contents of the sac may be gained by transillumination.

A meningocele will have no associated nerve lesions, but the meningomyelocele and the syringomyelocele are invariably accompanied by spastic paralysis of the muscles of the legs, talipes or pes cavus, and interference with the function of the bowel and bladder. Although sensory loss is not marked, trophic lesions are apt to be severe.

The clinical picture will be changed immediately for the worse if the surface covering of the sac should become ulcerated. The skin



A



B

FIG. 433

Two views of a meningomyelocele in a baby aged 9 months.

is frequently thin and atrophic in these lesions, and in such cases it can be only a matter of time before either rupture or infection occurs. Death from meningitis is then inevitable.

*Treatment.*—Meningoceles and meningocele (with spinal nerves and not the whole cord) are alone amenable to surgical treatment, and then only if the skin is healthy. The sac is excised, the nerve elements replaced, the dura closed, a muscle flap turned across the gap and the skin sutured. Even when the result seems good at first, a secondary hydrocephalus is likely to develop within a few months.

#### TUMOURS OF THE SACROCOCCYGEAL REGION

A number of very rare tumours are met with in this area, arising either from the neurenteric canal or the post-anal gut. They include the sacral pilonidal sinus, the post-anal dimple, dermoid cysts and lipomata. Certain specialised tumours of great pathological but slight



FIG. 434

A sacrococcygeal tumour. Mr V. Pennell's case.

surgical interest are the chordoma and the congenital sacrococcygeal tumour; the former arises in the remnant of the notochord, while the latter is a true teratoma, being regarded by some observers as an example of an "included foetus" (Fig. 434).

#### INJURIES OF THE SPINAL CORD

Fractures, fracture-dislocations and other injuries of the vertebral column are described in full in Chap. XLVI, and here we are concerned only with those lesions of the spinal cord and cauda equina which result from injury. A perfect understanding of the clinical pictures produced by injury and disease of the spinal cord at various levels can be founded only on an exact knowledge of the anatomy and neurophysiology of the central nervous system. The student is therefore referred to the standard textbooks on these subjects; he will find that he is amply repaid for the time thus spent, because he will then appreciate that the clinical picture of all spinal cord lesions is governed exclusively by an anatomical and physiological exactitude.

#### CONCUSSION OF THE SPINAL CORD

The physical conditions of environment of the brain and spinal cord are so different that no useful purpose is served by attempting

comparisons between them. The brain is encased within the unyielding skull and is subject to variations of intracranial pressure, whereas no such conditions are present in the vertebral canal.

The pathology of spinal concussion is therefore different to that of the brain, and its occurrence is altogether denied by some neurologists. Nevertheless, a condition does occur, though very rarely, in which widespread loss of function follows an injury to the spinal column, complete recovery taking place within thirty-six hours. It is believed that the only pathological changes present in the cord are minute areas of hæmorrhage and œdema.

*The Clinical Picture* varies according to the site and severity of the injury. Usually there is a total cessation of all functions of the cord below the affected segment, combined with an advanced degree of general shock. In the upper cervical region death may be instantaneous, in the lower cervical segments all four limbs are paralysed, while lower down still the damage is confined to the lower limbs and the sphincters of the bowel and bladder. In general, even when motor loss is complete, sensation is rarely entirely interrupted and priapism never occurs. In severe cases the picture is that of a complete transverse lesion of the cord, while partial injury to this structure may be made to appear more extensive than it actually is by the concussion which accompanies it.

*Diagnosis and Treatment.*—It is evident that in the early hours after injury an exact diagnosis is impossible and no one can say whether a total transverse lesion or concussion alone is present. The early treatment therefore must be that of a total lesion, the patient being kept absolutely prone and most carefully nursed. Within forty-eight hours either complete recovery has taken place or the residual loss of motor and sensory function will define the extent of the gross injury.

### COMPRESSION OF THE SPINAL CORD

Although injuries to the spinal cord are usually of sudden and dramatic onset and their results both formidable and far-reaching, it must not be thought that every injury will produce extensive muscular paralysis, sphincter dysfunction and sensory loss. Furthermore, the spinal cord and its nerves are damaged more gradually and more insidiously by other agents than gross trauma. Before describing the grave lesions of the spinal cord associated with injuries of the vertebral column, it will be instructive to obtain a general clinical picture of the results both of gradual compression and also of sudden gross destruction of nerve tissue. The effects on the nerves, the cord itself and the circulation of the cerebrospinal fluid must be considered separately.

**A. The Spinal Nerves.**—A mild degree of pressure on the *posterior nerve roots* causes irritative symptoms in the peripheral distribution of the nerve. Sensations of tingling, pricking and “pins and needles” will be followed by pain and hyperæsthesia of the skin supplied by the nerve. Later, when pressure becomes sufficient to arrest all conduction



of nerve impulses, anæsthesia takes the place of hyperæsthesia, but the pain continues (anæsthesia dolorosa). Pressure on the *anterior roots* is rarely seen except in conjunction with other and more extensive lesions. In its early stages it leads to spasmodic twitchings and cramps, which will later be succeeded by muscular atrophy, flaccid paralysis and loss of deep reflexes. Pressure on the *spinal nerve trunk* as it traverses the intervertebral foramen will provide a picture of combined motor and sensory irritation in the early stages and paralysis and anæsthesia in the later, all of which are confined to the area of distribution of the nerve concerned.

**B. The Spinal Cord.**—1. *Gradual Compression.*—(a) Motor effects. The earliest signs will be a weakness of voluntary movement in the toes and feet, succeeded by a gradual development of spastic paraplegia with increased reflexes and the presence of an extensor plantar response and ankle clonus. In the next stage the flexor group of muscles atrophy more quickly than the extensors, the legs become still weaker and “paraplegia in extension” is established. Eventually all power of voluntary movement is lost and the joints of the lower limb are pulled into extreme flexion, with the thighs in contact with the abdominal wall—“paraplegia in flexion.”

(b) Sensory changes. At the level of the compression, pain is experienced in the distribution of the spinal segment actually compressed. These “root pains” often precede all other symptoms by many weeks and are of the greatest diagnostic importance as giving a clue to the level of the lesion. Below this a loss of sensation gradually appears simultaneously with the early muscle weakness, until finally a complete anæsthesia up to the segmental level of the compression is established. In the area of distribution of the segment immediately above that affected, a zone of hyperæsthesia may be interposed between the areas of normal and abolished sensation.

(c) Sphincter Control. In the early stages, there will be bladder irritability and some loss of control. Later the behaviour of both bladder and rectal sphincters depends on the level of the lesion. If this is above the centres in the lumbar segments of the cord, there will be retention of urine, which passes into the condition of retention with overflow, until eventually the bladder adapts itself to an involuntary emptying at definite periods, over which the patient has no control—“the automatic bladder.” There is constipation, combined with loss of control after the administration of aperients.

When the lesion is low down in the cord, true incontinence of both urine and fæces occurs.

(d) Trophic changes are marked in the area below the level of the lesion, and it may be impossible, in spite of the most skilful and loyal nursing, to prevent the occurrence of bed-sores.

2. *Sudden Compression* is accompanied by the immediate onset of total flaccid paralysis with complete sensory loss below the affected level. If the patient survives the shock, there will be retention of urine, constipation, priapism and trophic changes. The picture varies with the level of the lesion and will be described below.

**C. The Cerebrospinal Fluid.**—Should the subarachnoid space be occluded, the cerebrospinal fluid becomes stagnant and certain changes occur in it. These constitute Froin's syndrome, the fluid being yellow in colour, having a specific gravity far in excess of normal, and coagulating spontaneously on standing.

*Methods of Examination.*—A searching neurological investigation will go far to establish both the diagnosis and the level of the lesion, but it is not always possible to be sure on clinical grounds alone. Froin's syndrome and Queckenstedt's test confirm the presence or absence of a block to the circulation of the cerebrospinal fluid, while the exact level of an obstruction within the theca can be demonstrated by radiology after lipiodol injection. The diagnosis and the decision as to operation are so frequently matters of such difficulty that the closest co-operation between neurologist and neuro-surgeon is called for.

### SPINAL HÆMORRHAGE

Hæmorrhage into the vertebral canal is usually associated with injuries to the spinal cord and column, but occurs very rarely, apart from injury, in young people below the age of 20 years. Every severe injury of this region must be accompanied by intraspinal bleeding, but two types of spinal hæmorrhage are described in which the symptoms are due to the hæmorrhage itself and not to gross injuries of the cord or column.

**Hæmatorrhachis** (Extramedullary Hæmorrhage).—As a result of an injury to the spinal column which gives no signs of fracture or dislocation, bleeding may occur either between the dura and the bone or within the dural sac. It almost invariably affects the cervical region. After an initial period of spinal concussion the picture is that of spinal irritation, with root pain and muscle twitchings. Later, if sufficient blood is extravasated, paralysis and anæsthesia will follow. Since the lesion is usually cervical, there is a lower motor neurone type of flaccid paralysis and muscular atrophy in the arms and an upper motor neurone type in the legs, with exaggerated reflexes.

A completely different picture is seen when the blood trickles down inside the dura and slowly, but in increasing amount, collects in the lower part of the thecal space. This leads to Thorburn's "gravitation paraplegia," in which first the signs of irritation and then those of paralysis appear in the areas supplied by the cauda equina and spread gradually higher and higher as the bleeding continues.

*Treatment* consists in complete rest, the injection of ergotin and adrenalin and careful attention to general nursing. If the bleeding is continuing and the area and extent of the pressure increasing, a laminectomy may have to be considered, though it can rarely do good.

**Hæmatomyelia** (Intramedullary Hæmorrhage).—In certain injuries the cervical spine may be violently overflexed, so that the cervical part of the spinal cord is stretched without there being any injury of the vertebral column. In such cases one or more hæmorrhages may occur in the region of the anterior horn cells of the lower segments of the cervical cord; as a result, areas of grey matter are permanently

destroyed, the neighbouring white matter is compressed and, if the bleeding is extensive, blood will rupture out of the cord into the subdural space.

After the spinal concussion, which follows immediately upon the accident, has passed off, a flaccid paralysis (lower motor neurone type) of the muscles supplied by the damaged anterior horn cells will be found. According to the extent of the lesion, either all the muscles of the upper limb or certain groups only are affected. In many patients the small muscles of the hand are the only ones thus paralysed. In addition, the pressure of the clot on the descending tracts in the cord leads to a spastic paralysis (upper motor neurone type) of the muscles of the leg. Interference with sensation is variable and irregular, and is frequently of that type of dissociated anæsthesia met with in syringomyelia. There is usually priapism and retention of urine and, as in other lesions of the cervical region, Horner's syndrome is present, viz., contraction of the pupil, retraction of the eyeball and narrowing of the palpebral fissure.

It will be evident that the future prospects of such patients depend entirely on the extent of the hæmorrhage. In some people the damage will be so severe as to constitute a virtual total transverse division of the cord and little, if any, recovery can be expected. In the usual type of case the damaged areas in the anterior horn cells are permanently destroyed and the resultant flaccid paralysis and wasting of muscles must persist, but the other lesions are due to pressure and may confidently be expected to improve or even recover completely. The final defects are, therefore, confined to certain muscles in the arms, while the legs recover. There is always the possibility that death may occur in the early stages from respiratory failure, while months later secondary spinal cord degenerations may set in.

*Treatment* consists in absolute rest, combined with efficient nursing. Laminectomy cannot possibly serve any useful purpose in this type of lesion.

#### COMPLETE TRANSVERSE LESION OF THE CORD

As might be expected from its small size, injuries of the cord are more likely to be complete than partial, and, as no regeneration in the central nervous system is possible, such injuries are unhappily irremediable. The cord is usually crushed by the displacement of bone in fractures or fracture-dislocations of the spine, and in many cases the damage is inflicted even though the spontaneous reduction of the dislocation has occurred within a few brief moments. It is with these injuries that we are chiefly concerned in civilian practice, but complete lesions are produced by several other means, *e.g.*, gunshot wounds of the spine and cord, and those conditions in which compression of the spinal cord occurs from spinal tumours, cysts and inflammatory exudates, in the later stages of which a partial compression terminates in a complete transverse lesion.

*General Clinical Picture.*—The exact distribution of the paralyses must depend on the level of the spinal injury, and an analysis of the

various clinical pictures follows. Nevertheless, it is useful to consider the general findings before passing to a detailed neurological description. Two stages have to be discussed.

**A. The Stage of Initial Spinal Shock.**—As soon as the patient has recovered from the general physical shock of the accident there will be found below the level of the lesion total flaccid paralysis and complete anæsthesia, absolute absence of all reflexes and retention of urine. Of equal importance, though less evident in the first few days, are the trophic disturbances. The feet are cold and blue, there is some œdema of the ankles and the skin rapidly becomes dry, shiny, thin and almost transparent and tends to crack. Even the most devoted nursing will not always succeed in preventing bed-sores over prominent bony points, and the least inattention may result in large sloughing ulcers over the sacrum. The bladder muscles are paralysed, but the sphincter is in tonic spasm, so that there is retention of urine and, if this is not relieved, false incontinence—that is, retention with overflow—results. Relief of the retention adds a further element of danger, since cystitis can hardly be avoided. Should it occur it tends to be of a fulminating type, which spreads rapidly to the kidneys. To a lesser extent the bowel also gives rise to anxiety; constipation is present, and incontinence is apt to follow the use of aperients, thus increasing the risk of bed-sores. The technique of tidal drainage is described on p. 759.

**B. The Stage of Spinal Reflex Activity.**—If the patient has survived the formidable dangers outlined above for four weeks, a change will be observed. There is no abrupt transition, but a slowly progressive alteration in the muscle reactions below the level of the lesion.

The muscles remain paralysed and the limbs are without sensation, but the flexor reflexes reappear in a very special way. At first the zone of skin sufficiently receptive to initiate the reflex is confined to a small area on the foot; it then spreads to include the whole of the sole and slowly, week by week, advances up the lower extremity until it finally embraces the whole area of skin below the level of the cord lesion. Not only does the area of reception thus extend its borders, but the field of response gradually spreads both in muscle distribution and in the force of the movement. At first, feeble flexion of the toes, ankle and knee joints occurs, but later, violent contractions of all the joints of the lower extremity are accompanied by similar movements of the abdominal muscles. In time this “mass reflex” may follow the most trivial cutaneous stimulation.

During this phase the bladder may regain its power of evacuation, although this is independent of the patient’s knowledge or volition. It is consequently known as the “automatic bladder,” and after many months a patient may learn to initiate the bladder action by some quite irrational act, such as stroking the skin on the inner side of the thigh; further, he may become aware that the involuntary act of micturition is shortly about to occur. During this period the trophic disturbances become progressively less and the danger of bed-sores is diminished, until after many months the nutrition of the skin

approaches normal, and burns or minor septic conditions heal without much difficulty.

*Prognosis.*—In some patients (see “Upper Cervical Segments”) death is instantaneous; very many others do not survive the phase of spinal shock, but a certain number will live for many years. Death is usually due to pulmonary infections, renal failure from an ascending pyelonephritis, or septicæmia from sloughing wounds or bed-sores. The lower the level of the lesion the more favourable is the outlook. Providing the arms escape, no patient need feel that he or she is a hopeless burden on other people or that life holds no prospects of interest and usefulness.

*Treatment.*—**A. Prophylactic.**—It must never be forgotten that the spinal cord may be damaged not at the time of the injury but by subsequent movements. No person who has sustained the type of accident likely to injure the spine should be moved until skilled aid is at hand. He must then be gently turned on to his face with the trunk extended, and all manipulations, such as lifting him on and off a stretcher, should be done in that position.

**B. The Stage of Spinal Shock.**—Treatment is directed to the prevention of complications and consists almost entirely in devoted nursing. Attention is paid to preserving the integrity of the skin, to treatment of the bladder (see p. 759) and to overcoming constipation and gaseous distension.

**C. The Later Stage.**—The most dangerous period having been surmounted, treatment during the succeeding months is both physical and psychological. The skin, bladder and bowel still need attentive care, but no less important is the treatment of the utter despair of mind and spirit. It is doubtful if any patient can live for any length of time unless he or she can be persuaded that life is not hopelessly futile.

*The Clinical Picture at Different Levels.*—1. **THE UPPER FOUR CERVICAL SEGMENTS.**—Injuries to this area are immediately fatal owing to the paralysis of all muscles of respiration.

2. **THE LOWER CERVICAL AND 1ST DORSAL SEGMENTS.**—The phrenic nerve escapes and a purely diaphragmatic type of respiration remains. Anaesthesia reaches to the second intercostal space. The cervical sympathetic being paralysed, Horner's syndrome is present, namely, contraction of the pupil, recession of the eyeball and narrowing of the palpebral fissure. There is retention of urine, constipation and priapism, which is a turgid semi-erect condition of the penis. The extent of the muscular paralysis varies according to the level, thus: (a) at the 5th cervical segment, the arms are totally paralysed and lie beside the body; (b) at the 6th cervical segment, the deltoids, the flexors and supinators are not involved, the arm lying abducted at the shoulder, flexed at the elbow and wrist, with the forearm supinated; (c) at the 7th cervical and 1st dorsal there is the same picture, the intrinsic muscles of the hand being paralysed, the “*main d'accoucheur*” resulting.

3. **THE DORSAL SEGMENTS FROM THE 2ND TO THE 12TH.**—The lesion is now below the level of the motor supply of the upper extremity and

the arms are normal. Respiration is still partly diaphragmatic in type and is somewhat embarrassed by distension of the intestines; further, feebleness of the act of coughing tends to the retention of sputum, so that the onset of hypostatic bronchopneumonia is an ever-present danger. According to the level, all or part of the intercostal and abdominal musculature is paralysed. Anæsthesia is of the girdle type, ending in a sharply demarcated line of hyperæsthesia passing horizontally round the body. The bladder will become automatic after a time. The prognosis is poor, but in the lower segments is more favourable.

4. THE LUMBAR SEGMENTS.—The paralysis will be confined to the muscles of the lower extremity and pelvic girdle, while the upper limit of anæsthesia reaches a level between the symphysis pubis and the umbilicus. The greater part of the abdominal musculature having escaped, respiration is free and the forcefulness of coughing is unimpaired, so that there should be no anxiety with regard to pulmonary complications. Priapism is not seen at this level. According to the relationship of the injury to the bladder centre, there will be either retention of urine with overflow or true incontinence. An automatic bladder is never established in these lesions. The lower bowel is incontinent and constipation and intestinal distension are absent. The prognosis is fair.

5. THE CAUDA EQUINA may escape total injury, and the picture is apt to be somewhat complicated. Either all the muscles of the lower extremity and perineum are affected or the quadriceps and adductors of the thigh may escape. Anæsthesia is extensive, but confined to the legs. There will be incontinence of both bowel and bladder, but priapism does not occur. It must be remembered that the nerves of the cauda equina are peripheral nerves; consequently, the paralysis will be of a lower motor neurone type, and the prognosis as to both life and function is good, since peripheral nerves are capable of regeneration, and laminectomy with suture may lead to complete recovery.

#### INCOMPLETE LESIONS OF THE CORD

These injuries are not uncommon, small areas of the cord being lacerated or contused, while spinal hæmorrhage and some injury to the spine will be associated with them. In the early stages the clinical picture is indistinguishable from that of a complete division, flaccid paralysis, loss of sensation and sphincter dysfunction being present. After some days or weeks a return of function will be observed in a small part of the paralysed area. This revelation of the incompleteness of the lesion is followed by a progressive improvement, and many weeks or months may elapse before the permanent damage can be estimated. Its distribution must necessarily depend on the level of the lesion as well as its extent in the cord.

*Treatment* in the early weeks will be similar to that of a complete lesion. Later, all muscles which show signs of recovery must be energetically assisted by faradism and massage. The same general care and devoted nursing are needed. In these partial lesions the



bladder is unlikely to be permanently affected and the return of voluntary control is to be expected.

**Hemisection of the Cord.**—Rarely a penetrating wound by bullet or shell splinter effects an exact division of one lateral half of the cord. As a result the Brown-Séquard syndrome will be seen, viz.: (a) on the affected side, there is paralysis of the leg of the upper motor-neurone type, with active reflexes including a plantar extensor response, and a loss of the sense of appreciation of the position of joints and of vibration; (b) on the opposite side there is loss of all sensation of pain and temperature, but no muscle paralysis. Control of the bladder is not likely to be lost.

**Lesions involving Nerve Trunks** may occur in any part of the spinal column. Injuries of the lower lumbar vertebræ can involve only the trunks of the cauda equina, and in the other parts of the column the nerves may be compressed as they traverse the intervertebral foramina, whilst the cord itself remains undamaged. The picture is that of a lower motor neurone type of lesion and depends entirely on the nerve involved and the extent of its injury, either temporary compression or complete division. Recovery may be expected after the release of pressure and, if necessary, suture, since regeneration is possible in peripheral nerves.

**Summary of Treatment.**—Each section contains directions as to treatment, but this subject is incomplete without some indication of the value of laminectomy. So insistent is the demand that "something should be done" in these sad cases that it is important that the limitations of any operation should be clearly defined.

Laminectomy can never be of use in the following conditions: (1) spinal concussion; (2) complete division; (3) hemisection of the cord (apart from the necessary technique of wound treatment); (4) the early stage of partial lesions.

It is indicated: (1) in partial lesions, when the localised nature of the injury has been revealed and the removal of a displaced fragment of the vertebra is possible (this is sometimes essential in the cervical region when manipulation and extension fail to overcome the signs of compression); (2) when the symptoms appear to be increasing steadily during the first few days, in order to control hæmorrhage; (3) in lesions of the cauda equina to suture one or more nerve trunks; (4) after the lapse of some months, when symptoms of compression suggest the presence of callus, an arachnoid cyst or scar tissue; (5) in penetrating wounds as part of the general technique for wound treatment.

#### TRAUMATIC SPINAL NEURASTHENIA

This condition, traditionally known as *Railway Spine*, is seen in these days following many other accidents than those of rare occurrence on the railway systems of this country. The accident is usually of great violence, in which the terror and mental shock exceed the physical injury. There is frequently some direct trauma to the back, but it is rarely severe, and actual fractures of the spine are never seen. At the

time the victim will profess to be unhurt, will probably assist in the rescue of others, and return home and to work the following day. Within a week symptoms of headache, backache, inability to concentrate and a feeling of impending disaster appear. The condition is in every respect analogous to "shell shock," with its attendant excitability, nervous irritability, weakness, loss of memory, dream-racked sleep, noises in the head and many other subjective symptoms. Atypical areas of tingling in the skin or patchy anæsthesia may be present; the bladder is irritable, and there may be loss of both sexual power and desire.

Examination reveals a complete absence of any positive evidence of organic disease in either the central or the peripheral nervous system. The most effective treatment consists in the favourable settlement of all legal claims for compensation.

### DISEASES OF THE SPINAL CORD

**Transverse Myelitis** is more usually considered in textbooks of medicine. Its surgical causes are long-continued pressure on the cord by displaced bone fragments, tuberculous granulation tissue or abscess, callus following a fracture of the spine or scar tissue. It may therefore occur as a late complication of injury to the spine and cord; a localised area of nerve tissue becomes softened, and the microscope reveals the death of nerve fibrils and cells. These changes tend to spread up the cord.

*Treatment* is to remove the cause as soon as symptoms are present.

**Acute Spinal Meningitis.**—A leptomeningitis affects the membranes of the spinal cord either as a direct extension from the cranium or as a primary infection resulting from penetrating wounds or a spina bifida. In these latter cases an extension upwards into the skull is only too probable. In spinal meningitis a sudden onset with one or more rigors ushers in a clinical picture of pain and muscular rigidity, which is frequently spasmodic in type, resembling that of tetanus. When the infection spreads to the brain the prognosis is hopeless, but if it remains localised to the spine recovery is possible.

*Treatment* is directed to the relief of symptoms, but in effect little can be done. Pain is sometimes relieved by repeated lumbar puncture.

**Chronic Spinal Meningitis** (Meningitis Serosa (Circumscripta) is a pachymeningitis of a chronic type. It may follow the localisation of an acute attack, it may be associated with injury or syphilis and it has been described as a result of the intrathecal injection of lipiodol or percaine. There is a localised thickening of the cord and membranes, in which latter cerebrospinal fluid may collect under tension.

The *symptoms* point to a gradual localised compression of the cord, pain and muscular weakness being the first indications. The picture is somewhat vague and atypical. Later, a spastic paralysis of the legs develops, but the bladder and bowel are not affected until very late in the disease.

*Treatment.*—The clinical picture cannot fail to raise the suspicion of a spinal tumour and a laminectomy will be the correct procedure.



If the cerebrospinal fluid has formed a localised cyst, great improvement may follow its removal.

### TUMOURS OF THE SPINAL CORD AND ITS MENINGES

Tumours which cause symptoms of compression of the spinal cord may be classified as follows :—

1. Extradural.
  - (a) Of the bones : chondroma, osteoma, sarcoma and secondary carcinoma.
  - (b) Of the soft tissues : lipoma, angioma and sarcoma.
2. Intradural, but extramedullary.
  - (a) Benign neoplasms : lipoma, fibroma, psammoma, cavernous hæmangioma.
  - (b) Malignant neoplasms : sarcoma and endothelioma (meningioma).
  - (c) Cysts : arachnoid and hydatid.
  - (d) Inflammatory swellings : tuberculoma and gumma.
3. Intramedullary. Malignant glioma and endothelioma.

*Symptoms and Signs.*—If the student will refer to page 877 he will find set out the clinical picture of gradual compression of the spinal cord, which is so well exemplified by tumours of the spinal cord and its meninges. The symptoms are of slow onset and depend upon the site of origin of the lesion in the spinal canal and its segmental level in the cord.

In extradural tumours the pressure is likely to be exerted first upon the nerve roots of one side and then upon that half of the cord ; so that the picture will primarily be that of root pain, secondly unilateral cord pressure, and finally general cord compression.

Intradural but extramedullary swellings are the commonest of all. They give rise to a prolonged stage of root-pressure symptoms before a slowly progressive compression of the cord makes its appearance.

The intramedullary tumours give no root pain but a partial paraplegia from the outset.

*Localisation.*—The localisation of the level of a tumour is obviously of the greatest importance. The clinical picture should provide a reasonable guide, but it is not always clearly defined. Lipiodol (or neohydriol) injections into the spinal theca permit an exact localisation of the obstruction by X-rays.

*Diagnosis* is by no means easy, for syphilis, syringomyelia, disseminated sclerosis and chronic spinal myelitis may at times prove most misleading.

*Treatment.*—Every patient suspected of a spinal cord tumour should be offered operation. The extradural and extramedullary tumours are usually on the posterior or postero-lateral aspect of the cord, and can frequently be removed with success. The intramedullary types are probably not amenable to removal, but even in these the decompression brought about by laminectomy will give considerable relief for a time.

### DISEASES OF THE SPINAL COLUMN

**Acute Osteomyelitis of the Spine** occurs in children and adolescents, but is rare. Its pathology is identical with that in the long bones, the causative organism being the *S. aureus*. It involves the body or laminae of either a cervical or lumbar vertebra. It is characterised by intense pain in the back, fever and toxæmia. Owing to the danger of spread to the meninges the prognosis is very bad. Rapid destruction of bone occurs and abscesses form.

*Treatment* consists in early incision to ensure adequate drainage and immobilisation of the spine with extension. If the patient survives, one or more sequestra will need to be removed.

**Typhoid Osteitis of the Spine** is also a rare condition, occurring during convalescence from typhoid fever. The lower dorsal and lumbar vertebræ are commonly the site of the lesion. The onset is sudden, the patient complaining of pain in the back, and on examination the spine will be found to be rigid and tender and there will be a high pyrexia. The history of typhoid fever, a positive Widal reaction and the radiographic appearance of necrosis, accompanied by the formation of new bone, serve to distinguish it from osteomyelitis and tuberculous diseases of the spine.

*Treatment.*—Pus does not always form and conservative treatment with immobilisation of the spine may lead to recovery. If pus does form, it must be given adequate drainage.

**Tuberculous Disease of the Spine** is described in full in Chap. XLVIII.

SYPHILIS OF THE SPINE is fortunately one of the rarer manifestations of the acquired form of this disease in its tertiary stage. Gummatous lesions and periostitis are met with chiefly in the cervical region and simulate Pott's disease very closely. The history, the positive Wassermann reaction and the sclerosis of bone in an X-ray should lead to a correct diagnosis.

*Treatment* is by the usual specific methods.

**Arthritis of the Spine.**—Arthritis deformans affects the spinal column in a manner similar to other joints. RHEUMATOID ARTHRITIS occurs in rheumatic subjects and its clinical picture is similar in many respects to the manifestations of rheumatism elsewhere. It attacks the cervical segments most commonly and affects the muscles and ligaments, so that pain, limitation of movement and even a deformity comparable to torticollis result. The treatment is directed toward the rheumatism.

OSTEO-ARTHRITIS differs in no way from the disease in the larger joints. It is very common in both sexes after the age of forty years, especially among people whose work has entailed a great deal of stooping and exposure. It affects chiefly the lower dorsal and lumbar regions. The cartilages are thinned and fibrillated, while osteophytic outgrowths form at the margins of the upper and lower surfaces of the vertebræ. The symptoms are pain and stiffness in the back, made worse by damp, cold and unwonted exercise. In early cases manipulation of the spine under general anæsthesia may effect considerable improvement, while later treatment is directed to the underlying cause. Locally, pain can be relieved—at least temporarily—by heat and short-wave diathermy.

**Spondylitis Deformans** is a disease of uncertain etiology which leads eventually to almost complete rigidity of the spine. Although in many respects similar to simple osteo-arthritis, in some details it displays characteristic qualities. Two pathological types are described : (a) the osteo-arthritic type, in which there is marked hypertrophy and condensation of the bones, with extensive formation of osteophytes from the vertebral bodies, while the changes also affect equally the intervertebral articulations ; (b) the ligamentous type, in which the bones are rarefied and osteophytes are not formed to any extent. The intervertebral discs atrophy, and widespread ossification occurs in all the ligaments, viz. : anterior spinal, interspinous, costo-transverse, etc.

*Clinically* also, two types are differentiated : (1) The Marie-Strümpell type (spondylitis rhizomélisque) affects not only the spine, but also the root joints, i.e., shoulder and hip. There is no involvement of the central nervous system. It starts in the lumbar region and spreads upwards, affecting both sexes equally and being uncommon before 35 years of age. (2) The von Bechterew type (spondylitis heredo-traumatique) occurs in males only and at a much earlier age (after 15 years). It commences in the cervical region and spreads rapidly throughout the spine. A certain number of patients develop a local meningitis in the cervico-dorsal segment of the cord.

The *symptoms* are pain, stiffness and deformity, until finally the spine becomes completely rigid (the "poker-back" spine). The prognosis is extremely poor and, until ankylosis has occurred, pain is severe and persistent.

*Treatment* is directed to the relief of pain and the prevention of deformity.

**Osteo-arthritis of the Sacro-iliac Joint** is by no means uncommon, especially in women. The only symptom is a dull persistent aching pain either in the region of the joint behind or referred to the front in either iliac fossa. Patients frequently come for advice complaining of low abdominal pain, and when this occurs on the right side many of them are diagnosed as examples of chronic appendicitis. The early cases are relieved by a manipulation under anæsthesia, and if this fails the injection of procaine sometimes achieves immediate success.

**Sacralisation of the Lumbar Spine.**—The 5th lumbar vertebra may, either wholly or in part, take part in the formation of the solid sacrum. Usually the changes affect the transverse processes, which are greatly enlarged and may form a joint or fuse completely with the ilium or sacrum ; these changes may be unilateral or bilateral. A large number of such abnormalities are devoid of all symptoms, but in some patients the deformity leads to pressure on the 4th and 5th lumbar nerves, scoliosis or localised sacro-iliac pain.

*Treatment* is symptomatic, except in very severe cases, in which resection of the overgrown transverse process may be required.

**Epiphysitis of the Spine** (Scheuermann's disease).—The lips of the upper and lower surfaces of the vertebræ are developed from secondary centres. These occasionally undergo changes similar to those in the femoral head and tibial tubercle. Symptoms are pain, malaise and the gradual development of a kyphosis.

*Treatment* is by immobilisation for three months.

**Coccydynia.**—Pain in the bottom of the spine is a frequent condition in women, and in the absence of a fracture or dislocation is invariably a symptom of neurasthenia. There is usually a history of a minor injury to this region in the shape of a fall or blow, and the pain is described as being excruciating. It is made worse by sitting, walking and defæcation. On examination nothing abnormal will be found, but manipulation between a finger in the rectum and the thumb externally produces "agonising pain."

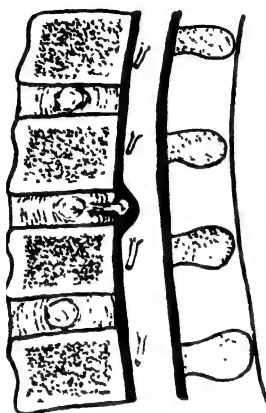


FIG. 435

Diagram illustrating prolapse of the nucleus pulposus.

*Treatment.*—If a fracture or dislocation has resulted in a forward displacement of the lower segments of the coccyx, which has not been properly reduced, the coccyx must be removed. But in all other cases operation is definitely harmful, as the pain persists. A dramatic and lasting recovery is usually obtained by the injection of procaine in front of and behind the coccyx. General treatment is directed towards the underlying neurosis.

**Prolapse of the Nucleus Pulposus** of an intervertebral disc in the lumbar region is a well recognised cause of compression of the spinal cord. The anterior surface of the cord or one or more nerves of the cauda equina suffers most from the compression (Fig. 435).

*Treatment* consists in laminectomy and removal of the prolapsed tissue.

**Growths of the Spine.**—Benign tumours of the spine are rare; they comprise chondroma and osteoma. The malignant growths are primary and secondary. Primary sarcoma is unusual. The commonest of all spinal tumours are the secondary carcinomata from the breast in the female, the prostate in the male and the thyroid, stomach and kidney in both sexes.

*The Clinical Picture* is very typical. Pain is of sudden onset, becomes increasingly severe and is uninfluenced by rest. At first localised to the vertebra concerned, it later spreads to the area supplied by the nerves emerging from the affected segments of the spine. Eventually the spinal canal is invaded and the cord compressed, so that paraplegia follows. A kyphosis is present in a certain percentage of patients and is due to collapse of the vertebral bodies. The condition is rapidly progressive and constitutes one of the more sad endings to malignant disease.

*Treatment* can be directed solely to the relief of pain, and in this connection X-rays and radium play an important part. The insertion of radon seeds is entirely justified if it results in the cessation of pain.

## DEFORMITIES OF THE SPINAL COLUMN

All types of deformity of the spine are dealt with in full in Chap. XLIX.

R. M. HANDFIELD-JONES.

## CHAPTER XLIII

### INJURIES AND DISEASES OF THE NERVES

**S**URGICAL ANATOMY.—An axis cylinder or nerve fibril is developed from a central nerve cell and its ability to transmit stimuli depends on the integrity of this connection. A number of axis cylinders are bound together by connective tissue to form a nerve fibre, bundles of which collected together constitute a nerve. The connective tissue carries blood vessels and lymphatics. In medullated nerves each axis cylinder has a medullary or myelin covering enclosed in the neurilemma or sheath of Schwann; non-medullated nerves have neurilemma but no medullary sheath.

The twelve cranial nerves emerge from the base of the brain and tend to be either purely motor or purely sensory in function. The spinal nerves arise in each spinal segment from the anterior (motor) and posterior (sensory) roots, which unite to form the main nerve trunk, this passing out between the adjacent vertebræ. Spinal nerves may remain single or unite with their neighbours to form plexuses, *e.g.*, the brachial, lumbar and sacral, but before doing so each trunk receives a branch from the sympathetic nervous system. The peripheral nerves are the result of this fusion, and consist of motor, sensory and sympathetic fibres, except in the case of a few purely sensory nerves such as the radial, internal cutaneous and long saphenous. The motor fibres which supply the voluntary muscles arise from the cells of the anterior horn. The involuntary muscles are supplied by the sympathetic system, except those which receive their innervation from certain cranial nerves, such as the vagus.

Three different types of stimuli are conveyed by sensory fibres, *viz.*, protopathic, epicritic and deep sensations.

1. Protopathic sensation is the appreciation of pain and marked differences in temperature. It is not a highly specialised sense, and the fibres concerned regenerate more quickly and completely than the others. It is tested for by sharp pin prick and with test tubes of hot and cold water.

2. Epicritic sensation is more highly specialised and consists in the appreciation and accurate localisation of the lightest touch and of small differences of temperature. It is of the greatest importance in the co-ordination of delicate muscular movements, and recovery after injury is slow and often imperfect. Wisps of cotton-wool and test tubes of water of slightly differing temperatures are used for testing it.

3. Deep sensibility and the appreciation of the position of joints and the movements of muscles are transmitted by the nerves which supply the muscles and tendons, and remain unchanged when cutaneous nerves are injured, provided that the muscles and tendons have escaped damage. Deep sensation is tested for by deep pressure, while the appreciation of position, shape and texture of objects (*stereognosis*) is analysed by asking a blindfolded patient to describe the position of a limb and the character of an object held in the hand. There is a certain amount of overlap in the

areas supplied by sensory nerves, this being more marked in protopathic than in epicritic sensation, and so the loss of sensation is smaller than the area supplied by the damaged nerve.

Sensory nerves have a nutritional influence on the tissues which they serve and trophic changes occur when a nerve is injured.

*Methods of Examination.*—When a lesion of a peripheral nerve is suspected, the nature of the motor disturbance must be investigated in addition to the tests for sensory loss detailed above. The amount of interference with motor function varies from complete paralysis to a short period of inactivity with rapid recovery, according to whether the nerve is divided or merely concussed or contused. When the voluntary control of a muscle is lost its fibres atrophy (wasting), and there is an alteration in the response to electrical stimulation known as the "**Reaction of Degeneration**," commonly called R.D. Under normal conditions, when the electrode is placed over the motor point of the muscle, contraction results from both faradic and galvanic stimulation, the response to faradism being greater than to galvanism, i.e.,  $F > G$ . Moreover, on galvanic stimulation the optimum response is obtained when the kathode is placed over the motor point and the current is closed, i.e., the kathodal closing current is greater than the anodal closing current, or  $K.C.C. > A.C.C.$  In the reaction of degeneration there is no response to faradism and a polar reversal is obtained with the galvanic current, for the anodal closing current now elicits a response with a weaker current than kathodal, i.e.,  $A.C.C. > K.C.C.$  These changes become evident from five to fifteen days after injury. Later, if the nerve is completely destroyed, the muscle fibres become replaced by fatty and fibrous tissue and lose all power to respond to either faradic or galvanic current. This change takes several months to develop and means that regeneration is no longer possible, but as long as there is any response to galvanism the possibility remains that repair of the nerve will give a return to voluntary control. The type of response to galvanism, whether brisk or sluggish, also serves as a criterion of the state of the muscle fibres.

Unless splints are carefully applied to prevent deformity the unbalanced action of the opposing groups of muscles will lead to their contracture, while the paralysed muscles may be so stretched that their chances of good functional recovery are considerably lessened. The paralysed muscles should therefore be supported in the position of relaxation.

Alterations in the blood supply, or vasomotor changes, are always present in the area supplied by a damaged nerve. The early signs are those of congestion, but this later gives place to a marked deficiency of blood supply, the skin becoming blue and cold and liable to chilblains. Trophic changes are always present. The subcutaneous fat is absorbed, and the skin becomes scaly, smooth and shiny, and is dry from absence of sweating. It is easily injured and heals very slowly. The nails become grooved and brittle.

### INJURIES TO NERVES

A nerve may be injured in many ways and in some instances may be the only structure damaged. More commonly the nerve lesion is a complication of an open wound, contusion, fracture or dislocation. It may follow manipulations to reduce these injuries, or be due to stretching by any subsequent deformity. A nerve may be completely severed or avulsed from its origin; it may be bruised, crushed, stretched, or compressed by callus or a tumour. The injury may be complete or incomplete, but a complete physiological division does

not necessarily imply anatomical division, as the nerve may appear to be intact to the naked eye when all function is lost.

**Concussion of a Nerve** is seen in penetrating wounds when the passage of a high velocity missile close to the nerve results in a temporary anæsthesia and paralysis of varying degrees of severity without any physical damage to the nerve itself. Massage and electrical treatment usually bring about a speedy recovery provided sepsis is not present.

**Contusion of a Nerve** is the result either of direct injury or of such indirect trauma as stretching and is usually accompanied by some hæmorrhage into the sheath, and in the more severe cases by tearing of a few fibres. The symptoms are tingling, a sensation of "pins and needles" in the area supplied and a slight muscular weakness. The condition is transient and complete recovery follows, though rest, massage and electrical treatment may be needed.

**Complete Division of a Nerve** is of common occurrence and is usually seen in wounds caused by broken glass, sharp instruments or gun shot. Similar injury without a superficial wound is caused by fractures and dislocations or by attempts to reduce them. In open wounds the ends of the divided nerve tend to separate widely, while in gunshot wounds a portion of the nerve may be carried away. In closed wounds the sheath is more resistant than the nerve fibres and may remain intact. These injuries are more common at the wrist, elbow and shoulder. They result in immediate paralysis and anæsthesia in the area supplied by the affected nerve. The early recognition of the nerve lesion is of the utmost importance, as immediate suture gives the best promise of complete recovery. Its possibility must always be kept in mind, and every wound, fracture or dislocation carefully examined to exclude nerve injury.

**RESULTS OF DIVISION.**—The retracted ends become involved in the scar tissue of the wound and develop a bulbous enlargement known as an "end bulb" or "traumatic neuroma," which consists of organised scar tissue and axis cylinders. The swelling is larger at the proximal end, owing to attempts at regeneration and the growth of new axis cylinders. Sepsis greatly increases the size of these end bulbs. They are the cause of pain and tenderness in amputation stumps and may prevent the patient from working or wearing an artificial limb, in which case the nerve must be exposed, its swollen part removed and the fresh-cut end crushed and ligatured.

**DEGENERATION OF A DIVIDED NERVE.**—The distal portion degenerates completely from the cut surface throughout its distribution whether an immediate suture is performed or not. The myelin breaks up into fatty particles which are absorbed, the axis cylinders disintegrate, and the neurilemma undergoes fibrosis. Eventually the distal portion becomes converted into fibrous tissue. The proximal portion degenerates only up to the next node of Ranvier, from which point regeneration takes place.

**REGENERATION.**—Axis cylinders grow out from the proximal end, seek the degenerating distal end, and if successful grow down it. These axis cylinders have no myelin sheath at first, but this is developed



later. The neurilemma is produced by both portions of the nerve. This downgrowth is slow, being estimated at 1 mm. a day, and regeneration therefore takes many weeks. Satisfactory recovery is more likely in nerves such as the musculospiral, which are largely motor and control no fine movements, whereas in mixed nerves and in those which control delicate movements full recovery is rarely complete, as, for example, in the median and ulnar nerves. Sepsis is a grave handicap, as it destroys the budding fibrils, and the resulting scar tissue prevents proper regeneration. When there is a definite gap between the cut ends the new axis cylinders grow into it and, failing to find their way into the distal portion, curl up in the end bulbs. Immediate and accurate suture, therefore, provides the only suitable conditions for satisfactory regeneration of the axis cylinders.

**SIGNS OF RECOVERY.**—When a nerve has been sutured the appearance of evidence of recovery must necessarily depend on the site of the injury; for instance, regeneration will be more rapidly achieved when the ulnar nerve is divided at the wrist than at the elbow. The first sign is a gradual return of sensation. Three months after suture of the ulnar nerve at the wrist, protopathic sensation returns, the area of anæsthesia slowly diminishing. Epicritic sensation reappears two months later and motor power begins to return after six months, but the maximum degree of recovery must not be expected until two years have elapsed. *Tinel's sign* is useful in determining how far regeneration has progressed. Pressure on the growing but yet unmyelinated axis cylinders causes painful tingling referred to the distribution of the nerve. Gentle tapping over the nerve trunk from the wound downwards will reveal a point at which this sensation is produced and so indicates the limit of regeneration. Motor power returns first to the muscles nearest to the site of injury. Electrically, the first sign of muscle power is reaction to galvanism, but faradic stimulation will rarely give a response until voluntary control has been regained. Trophic changes in the skin are slow to disappear.

The factors which influence recovery in a complete nerve injury are :—

- (1) The interval between injury and suture—the longer the interval the less favourable the prognosis ;
- (2) The nature of the nerve involved ;
- (3) The presence of sepsis ;
- (4) The relaxation of all paralysed muscles by suitable splints and maintenance of their function by galvanic stimulation.

**Treatment—Primary Suture.**—A completely divided nerve must be sutured at the earliest possible moment, and this implies that it is done during the first treatment of the wound. The cut ends are sought for, the wound being enlarged if necessary, freed and brought into apposition without tension, rotation or displacement. One tension suture of fine catgut is passed through the nerve  $\frac{1}{4}$  in. from the cut surfaces, and still finer coaptation stitches unite the margins of the sheath. If there is no tension sutures can be avoided, the ends being cemented together by encasing them in specially prepared fibrin.



Artificial coverings for the suture line are unnecessary, but the nerve should be buried beneath muscle, fat or fascia. It must be treated with great care and not stretched or compressed during the operation. For fourteen days after operation the limb is fixed on a light plaster splint in the position of maximum relaxation of the sutured nerve to avoid tension on the stitches, after which the position is altered to relax the paralysed muscles. Massage and electrical treatment are begun at once, the joints being kept free from adhesions by movement and the limb warm. The objects of subsequent treatment are to prevent contraction of the unopposed and stretching of the paralysed muscles, to keep the latter in as good condition as possible and to minimise as far as is feasible the trophic changes, so that when the new axis cylinders arrive at their terminal distribution, function can be resumed at once.

**Secondary Suture.**—If sepsis is present in the wound, primary suture should not be attempted, but the sepsis treated and the wound allowed to heal. Suture should not be considered till one month after all signs of sepsis have disappeared.<sup>1</sup> Late suture also is needed in those cases in which the nerve damage was not recognised at the time of injury. While waiting for the sepsis to subside or for a definite diagnosis, the paralysed muscles are supported in splints and stimulated by galvanism. If the galvanic response steadily improves and faradism begins to produce contractions, the nerve is recovering spontaneously and no operation is necessary.

In secondary suture certain difficulties arise in the presence of scar tissue, in the identification and freeing of the nerve ends and in overcoming the retraction sufficiently to obtain apposition without tension. Various manœuvres may have to be adopted to remedy this retraction or close the gap caused by destruction of tissue. First, the nerve must be freed for several inches above and below the wound, each branch encountered being similarly dissected. This allows considerable stretching of the nerve, and if necessary the neighbouring joints are so manipulated that still further relaxation is obtained, or in the case of the ulnar nerve at the elbow joint, its transference to the anterior aspect of the internal condyle adds still further to the relief of tension, so that apposition becomes possible in most cases. The bulb neuromata are then removed by a clean cut with a scalpel and the suturing done in the same way as in a primary operation. Every effort must be made to obtain end-to-end union without tension, for if this fails the results of alternative operations are most disappointing. The implantation of each cut end into a neighbouring healthy nerve in the hope that the fibrils will grow down it and so gain the distal end, the turning down of flaps of the nerve and the shortening of the bones of a limb have all been tried with poor results.

The treatment after secondary suture is similar to that following primary suture, but the time required is very much longer. When a joint has been flexed to obtain apposition, the limb must be splinted in that position for three weeks and then slowly and gradually

<sup>1</sup> A test for latent sepsis is the exposure of the area to radiant heat for fifteen minutes. Latent sepsis is present if hyperæmia and throbbing of the part appear during the following twenty-four hours.

straightened. Relaxation of the paralysed muscles by splinting follows, and massage and electrical treatment must be persisted in. The process is a slow one, and the prognosis depends to some extent on the interval between injury and suture, while in some patients, in spite of apparently accurate suture, no regeneration occurs. When all hope of recovery has been given up, function must be restored as far as possible by carefully planned tendon transplantations.

**Partial Division of a Nerve** may be either due to penetrating wounds, or physiological without any visible tear. The fibres which are severed retract and form a false neuroma, which may be centrally or laterally situated, according to the position of the injury. There will be paralysis of the muscles concerned, with a modified reaction of degeneration, while those supplied by the uninjured fibres remain normal. Sensory changes will also be more localised. In some cases an irritative lesion of the nerves develops, which gives rise to an intense burning pain in the skin supplied. This is known as "causalgia" and may be very severe, occurring in paroxysms which are brought on by such mild stimuli as warmth or light touch, and in severe cases even by mental stimuli such as lights and noise.

In partial lesions, after the exact extent of the damage has been determined, the nerve should be exposed if there has been no improvement after three months. The scar tissue and the neuroma are carefully excised, the cut ends trimmed and sutured, the intact portion of the nerve being disturbed as little as possible. In some cases of causalgia this will suffice, but in others the symptoms persist and are so severe that it may be necessary to resect the nerve or inject alcohol into it.

**Pressure on Nerves.**—A nerve may be compressed by scar tissue or stretched and pressed on by callus following a fracture. The symptoms of tingling, loss of sensation, muscular weakness and wasting will not be noticed for some weeks or months after the original injury. If no improvement is seen after three months observation, or if the symptoms are obviously increasing, the nerve must be exposed and freed from the contracting scar tissue, or displaced so that it can no longer be compressed. It should be buried in muscle or wrapped in amnioplastin. The nerves of the arm are sometimes injured by the pressure of tables, chairs or splints; for example, "crutch palsy" is due to pressure of a crutch in the axilla on the musculospiral nerve; "Saturday-night palsy" is due to the pressure of the back of a chair upon the brachial nerve trunks in a patient, who falls into a drunken sleep in this position; "operation-table palsy" is due to the arm being allowed to hang over the edge of the table during an operation. Tourniquets left on too long or applied over unsuitable points may cause temporary paralysis. The lesions are usually slight and transient, but careful splinting, massage and electrical treatment will be necessary in the more severe cases. Very rarely reaction of degeneration sets in and an exposure of the nerve will be needed to remove the cause of the pressure.

**Neuritis and Neuralgia.**—These conditions are of medical rather than surgical interest, but it is important in their treatment to place

the affected part at rest in a position of relaxation by suitable splints or plaster. Care must be taken to prevent deformities as a result of any paralysis which may develop, and the tone of the muscles must be maintained by massage and electrical treatment.

Neuralgia is a symptom of an irritative lesion of the nerve and this must always be searched for. When the cause cannot be determined the nerve itself will have to be treated to relieve the paroxysms of pain. Sensory nerves are treated by injection of 80 per cent. alcohol or by division or resection of their main trunks (trigeminal neuralgia, p. 868). Motor nerves cannot be treated so drastically and resort must be had to stretching.

**Growths** of nerve arise either from the nerve tissue itself or from the fibrous tissue of the sheath, and are called neuromata. The true neuroma is a rare tumour seen only in the posterior abdominal sympathetic system and may contain ganglion cells. The false neuroma is fibromatous in structure and may be single or multiple. In the single form a nodule can be felt in the line of the nerve, and can be moved transversely across but not up and down the long axis of the nerve. The tumour may be a fibrosarcoma, in which case its rapid growth, fixation to surrounding structures and interference with the function of the nerve will determine the diagnosis. *Multiple neurofibromatosis* of von Recklinghausen occurs in families and is associated with pigmentation of the skin and nodules scattered along both the subcutaneous and deep nerves. A diffuse enlargement, due to fibrous changes in the branches of a nerve or of contiguous nerves, leads to a condition known as *plexiform neuroma*.

## AFFECTIONS OF INDIVIDUAL NERVES

### SPINAL NERVES

**The Cervical Plexus** is injured in association with fractures and dislocations of the cervical vertebræ. The nerve lesion is rare and is greatly overshadowed by the vertebral injury and that of the spinal cord.

**The Phrenic Nerve** is injured by penetrating wounds or during operations. Irritation causes hiccough. When one phrenic nerve is paralysed the corresponding half of the diaphragm rises instead of moving downwards during inspiration. When both are paralysed the abdomen is sucked in during inspiration instead of being protruded.

The operation of crushing or avulsion of the nerve is sometimes practised in cases of early unilateral pulmonary tuberculosis to reduce the lung movements.

### THE NERVES OF THE UPPER EXTREMITY

#### **The Brachial Plexus**

*Anatomy.*—The brachial plexus is formed from the anterior primary divisions of the 5th, 6th, 7th, 8th cervical and the 1st dorsal nerves. The

root supply of the muscles of the upper extremity can be given as follows :—

5th Cervical . .	Rhomboids, supraspinatus and infraspinatus, deltoid, biceps, brachialis anticus and the supinators of the forearm.
6th Cervical . .	Clavicular head of pectoralis major, serratus magnus, the pronators, and the radial extensors of the wrist.
7th Cervical . .	Sternal head of pectoralis major, triceps, extensor carpi ulnaris and the extensors of the fingers.
8th Cervical . .	The flexors of the wrist and fingers.
1st Dorsal . .	The intrinsic muscles of the hand.

Injuries are caused by stretching or tearing when the arm is violently pulled away from the body, by direct contusion, by fracture or dislocation of the spine, clavicle or shoulder, by penetrating wounds and by the pressure of scar tissue, callus, tumours or a cervical rib. The injury may be complete or partial, and affect either the spinal nerves, the cords, trunks or branches of the plexus. It is important that diagnosis should include exact localisation of the injury within the plexus.

**WHOLE PLEXUS TYPE.**—These injuries are rare and are produced by violent wrenching of the arm away from the body, *e.g.*, when a workman's hand is trapped in rotating machinery and he is whirled round and round, or when a man seeking to board a fast-moving vehicle grips the handrail but fails to secure foothold. Flaccid paralysis of all the muscles of the arm, forearm and hand is present, and usually the deltoid and pectorals are affected, but only in very high lesions will the rhomboids and spinati or serratus magnus be involved. Anæsthesia is present over the whole arm except over the deltoid and inner aspect of the upper two-thirds of the arm. If the 1st dorsal nerve is injured high up, the sympathetic fibres will be torn and contraction of the pupil, enophthalmos and absence of sweating in the face and arm on the side of the lesion are present. Expectant treatment should be adopted, the arm being placed in a light metal splint, the humerus abducted to 90° and externally rotated, the elbow flexed to a right angle, the forearm supinated and the wrist and hand slightly dorsiflexed. Massage and electrical treatment are given to all the paralysed muscles for at least three months. If no improvement is seen and if the reaction of degeneration is present, operative exposure must be considered.

**UPPER ARM TYPE.**—The Erb-Duchenne palsy is by far the most common injury and is due to a lesion of either the 5th or the 6th and 7th cervical anterior primary divisions. It frequently arises from a birth injury, the head being stretched away from the shoulder during a difficult delivery. In later life, falls on the side of the head or the point of the shoulder, which violently separate them, may cause this type of lesion.

**Symptoms.**—The arm hangs uselessly by the side and is internally rotated, the elbow is extended and the palm faces backwards ("waiter's arm"). Flaccid paralysis is present in the deltoid, supraspinatus, infraspinatus, biceps, coraco-brachialis, brachialis anticus and supinators, and occasionally in the extensors of the wrist. There is a small area of anæsthesia over the outer aspect of the arm.

*Diagnosis.*—The history of a prolonged and difficult labour or of a definite injury makes diagnosis easy and, further, there is tenderness on deep pressure in the supraclavicular triangle. In adults with an incomplete lesion, pressure causes tingling in the distribution of the 5th nerve. A fractured clavicle at birth may cause difficulty, because the infant does not move the arm and the root of the neck is tender, but an X-ray and the absence of paralysis will reveal the real lesion. Acute arthritis of the shoulder and syphilitic epiphysitis of the head of the humerus give rise to a pseudoparalysis, but there is swelling around the shoulder and signs of true palsy are absent. Long-standing cases of Erb's palsy must be distinguished from anterior poliomyelitis and from muscular dystrophy of the scapulo-humeral type. Infantile paralysis will be recognised by its acute onset in a previously healthy child, while in muscular dystrophy both shoulders are affected and the scapulæ will show winging.

*Treatment.*—Erb's palsy is usually incomplete and recovery is good in every case if diagnosed at once and treated efficiently. The arm is held in a Fairbank's splint in the position already described, *i.e.*, shoulder abducted and externally rotated, elbow flexed to a right angle, forearm supinated and wrist dorsiflexed. Massage and galvanic stimulation are necessary for a long time. Failure of recovery and development of the reaction of degeneration point to a complete lesion, the results following operation for which are far from satisfactory.

**LOWER ARM TYPE.**—Klumpke's palsy is due to an injury to the 1st dorsal nerve and frequently to the 8th cervical. It may be caused by birth injuries or by the wrench sustained when a falling person attempts to save himself by grasping some support.

*Symptoms.*—Flaccid paralysis of all the intrinsic muscles of the hand results from injuries to the 1st dorsal nerve, and when the 8th cervical is also injured there will be paresis of the flexors of the wrist and fingers. The patient will be unable to adduct or abduct the fingers or to oppose the thumb. Later the wasting of the thenar, hypothenar and interossei muscles leads to a claw hand, the fingers being hyperextended at the metacarpophalangeal and flexed at the phalangeal joints. The sensation of the hand is not impaired, but anæsthesia is present over the inner aspect of the arm and forearm. Injury to the 1st dorsal nerve close to the cord involves the sympathetic fibres, causing enophthalmos, narrowing of the palpebral fissure and contraction of the pupil, which dilates either very slowly or not at all. The eye symptoms denote an injury so close to the cord that operation is hardly likely to do good, but fortunately these brachial plexus lesions are usually incomplete, as is shown by the dilatation of the pupil when cocaine is instilled.

*Diagnosis.*—The history of an injury is as a rule present, but the condition has to be distinguished from anterior poliomyelitis and from cervical rib.

*Treatment* consists in splinting to relax and support paralysed muscles, combined with prolonged massage and electrical stimulation. If the reaction of degeneration sets in, operative exposure must be

undertaken. The further the nerve lesion is from the cord the more hopeful the prognosis.

**Injuries to the Cords.**—The inner, outer and posterior cords may be injured by fractures of the clavicle, scapula or humerus, and by subsequent attempts at reduction. The injury most commonly takes the form of compression and is rarely complete.

THE INNER CORD is most frequently involved, and there results a paralysis of all the intrinsic muscles of the hand and those of the forearm supplied by the ulnar nerve, a claw-hand being produced. Anæsthesia is present over the inner aspect of the arm and forearm and the ulnar area of the hand.

THE OUTER CORD injury is rare and results in paralysis of the biceps, coraco-brachialis, brachialis anticus and those flexors of the wrist and fingers supplied by the median nerve. The intrinsic muscles of the hand derive their innervation from the inner cord and are therefore unaffected. Anæsthesia is present over the outer surface of the forearm and the median area of the hand.

THE POSTERIOR CORD is also seldom injured. It results in paralysis of the deltoid, supraspinatus, infraspinatus, triceps, teres major and minor and the muscles supplied by the musculospiral nerve. Anæsthesia is limited to a small area on the outer aspect of the arm and forearm.

*Treatment* consists in splinting to relax the muscles and prevent deformity, and in massage and galvanism, but the onset of the reaction of degeneration will determine the necessity for exploration and repair of the injured cord.

**The Long Thoracic Nerve of Bell** may be injured in the neck by penetrating wounds, blows and pressure from weights carried on the shoulder. It has been damaged in operations on the neck and axilla during the radical treatment of carcinoma of the breast. Uncomplicated injuries of this nerve cause paralysis of the serratus magnus. When it is compressed by heavy weights carried on the shoulder, the branches of the 3rd and 4th cervical nerves to the lower part of trapezius and the nerve to the rhomboids are usually involved at the same time.

*Symptoms.*—If the serratus magnus alone is paralysed little deformity is visible when the arm hangs at the side, except that the inferior angle of the scapula is more prominent than that of the opposite side. The patient cannot raise the arm forwards in front of the body above the level of the shoulder, nor can he push forward against resistance at the level of the shoulder, attempts to carry out these movements being accompanied by marked *winging of the scapula*. Similar movements can be performed below the shoulder level unless the lower part of the trapezius is also injured. In these combined nerve lesions, winging and tilting of the scapula are marked even when the arm is held at the side, and this deformity is increased immediately any pushing movement is attempted.

*Treatment.*—These injuries are usually incomplete, and support of the paralysed muscles with massage and galvanic stimulation suffice to bring about a complete recovery. With the onset of the reaction of degeneration the nerve should be exposed and sutured, and if this

fails the deformity of the scapula may be corrected by transplanting the insertion of the lower part of the pectoralis major into the inferior angle of the scapula.

**The Nerve to the Rhomboids** is rarely injured alone, but if this should happen the scapula is on a lower level than its normal fellow, its inferior angle is carried away from the mid-line and its spine lies more obliquely.

**The Suprascapular Nerve.**—Injuries to this nerve cause wasting and paralysis of the supraspinatus and infraspinatus, with weakness in abduction and external rotation of the arm. The lesion is incomplete and the usual treatment will suffice.

**The Circumflex Nerve** is injured by direct wounds or blows, fractures of the surgical neck of the humerus, dislocations of the shoulder and compression from crutches. The deltoid and teres minor are paralysed and abduction of the shoulder is lost. Marked wasting of the deltoid is present. There is a small area of anæsthesia over the posterior border of the deltoid. Lesions of the 5th cervical nerve give rise to a similar disability, but are characterised by paralysis of the spinati and a larger area of anæsthesia.

If an injury to the circumflex nerve is suspected the arm should be held in abduction to a right angle on an abduction splint and massage and electrical treatment given to the deltoid. Even when the paralysis of this muscle is complete, other muscles will in time overcome the disability and operation is never required.

**The Musculospiral Nerve** may be injured by penetrating wounds, but the damage is usually subcutaneous. The dislocated head of the humerus, old-fashioned crutches and the sharp edge of a chair over which the drunken man hangs his arm are causes of compression lesions in the axilla; in the arm itself the nerve winds round the humerus and is liable to injury in fractures of the middle and lower thirds of the shaft, and it may later be involved in the callus. A tourniquet applied too tightly or left on too long will cause damage.

The resultant paralysis involves the extensors of the wrist, thumb and fingers, causing wrist drop (Fig. 436) and pronation of the forearm. Supination of the flexed arm can still be carried out by the biceps, but the patient cannot voluntarily extend the wrist or fingers. If the lesion is high up, active extension of the elbow will be lost owing to paralysis of the triceps and anconeus. As the wrist is flexed, the grip is weak, since the extensors are unable to extend and stabilise the wrist to assist the action of the flexors. If the wrist and proximal phalanges are held in extension, the interossei and lumbricals are able to straighten the middle and distal phalanges. When the nerve is injured above the origin of the external cutaneous branch, there will be anæsthesia over a small area on the back of the radial side of the hand and thumb, but injuries below this level cause no sensory loss.

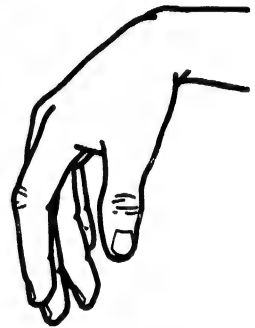


FIG. 436

The dropped wrist  
tion resulting from mus-  
culospiral nerve palsy.



The majority of musculospiral nerve injuries are incomplete, but even in complete lesions the prognosis is good. The wrist and fingers

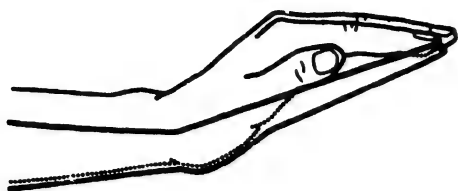


FIG. 437

The hand in a Robert Jones's long cock-up splint.

are held in hyperextension on a Robert Jones's long cock-up splint (Fig. 437) and massage and galvanic stimulation given until signs of active extension of the wrist return. If the reaction of degeneration sets in or if there are no signs of improvement in three months, the nerve must be exposed and freed. The results of operation on the

musculospiral are superior to those on all others except its counterpart in the lower limb—the external popliteal—probably because it is largely a motor nerve. When the nerve lesion is discovered in association with fractures of the humerus, operation should always be advised, not only to repair the nerve but to restore the fracture. If all treatment has proved unsuccessful, various tendon transplantations can be done to restore the extension of the wrist.

The posterior interosseous nerve is occasionally injured by posterior dislocations or fractures of the head of the radius or by operations in this region. Proper treatment of the dislocation or fracture, with support,

massage and electrotherapy to the paralysed extensors will usually achieve a good

recovery. Operation should be performed if no recovery is seen after two months.

**The Median Nerve** is commonly injured in any part of its course, more especially by incised wounds in front of the wrist and by becoming involved in fractures around the elbow. Such severe disability of the hand results that early recognition and prompt treatment are of paramount importance.

**INJURY IN FRONT OF THE WRIST.**—The abductor, opponens and outer half of the flexor brevis pollicis are paralysed, and there is marked wasting of the thenar eminence (Fig. 438) and flattening of the hand as the thumb falls back into the same plane as the fingers.

Abduction and opposition of the thumb are lost (Fig. 439).

Attempts to produce opposition of the tip of the thumb to the tip of the little finger result in flexion and adduction of the thumb, the



FIG. 438

Wasting of the thenar eminence in median nerve palsy.



FIG. 439

Diagram illustrating the movement of the thumb when the median nerve is undamaged.



patient being unable to bring the thumb forward from the plane of the fingers and swing it across the palm. Abduction to a small extent can be simulated by the extensors, but again the thumb remains in the plane of the fingers.

Anæsthesia is present throughout the anatomical distribution of the nerve. Epicritic sensation is lost over the outer half of the palm, over the palmar aspect of the index, middle and radial half of the ring fingers, and over the dorsal aspect of the distal two-thirds of these fingers. Protopathic loss is much less extensive and is confined to the distal half of the index and middle fingers (Fig. 440, A and B).

**INJURY NEAR TO ELBOW.**—The following muscles are paralysed in addition to the above, pronator radii teres, flexor carpi radialis, flexor longus pollicis, pronator quadratus, all the flexor sublimis digitorum and the outer half of the flexor profundus digitorum. The patient will also lose the power of pronation, radial deviation of the wrist and flexion of the index and middle fingers. Anæsthesia remains as before, with the addition of some loss of deep sensibility over a small part of the area.

Trophic changes are well marked in both lesions, and the

skin and nails are seriously affected. Causalgia is particularly likely to occur in partial injuries of this nerve.

**Treatment.**—Every wound in front of the wrist should be regarded as containing a severed median nerve until exploration has proved the contrary. Immediate primary suture is the best possible treatment. If the injury is unrecognised, secondary suture should be undertaken as soon as latent sepsis has disappeared. The prognosis is not altogether satisfactory, but so serious is the disability of a complete median injury that operation must be performed in every case.

**The Ulnar Nerve** is liable to injury in fractures of the internal condyle of the humerus and of the olecranon, and in dislocations of the elbow and shoulder. It may be involved at the time of the actual injury, during subsequent attempts at reduction, or compressed later by callus or scar tissue. It may also be severed in cuts in the front of the wrist or forearm, or on inner aspect of upper arm.

**INJURY OF THE WRIST.**—The muscles of the hypothenar eminence,

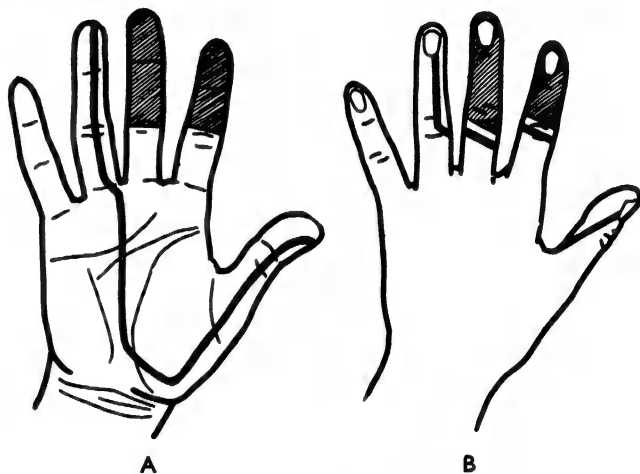


FIG. 440

A, diagram showing the extent of anæsthesia on the palmar aspect as the result of division of the median nerve at the wrist: B, the changes on the dorsal aspect of the hand. The heavily shaded area represents the loss of protopathic sensation while the whole area enclosed in the heavy black line shows the extent of the epicritic loss.

the adductor transversus and adductor obliquus pollicis, the deep or long head of the flexor brevis pollicis, the two inner lumbricals and all

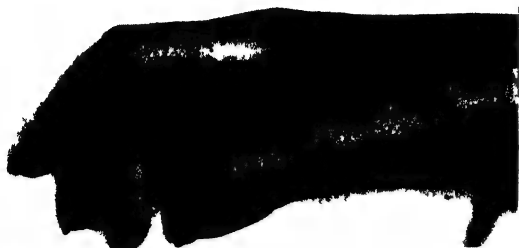


FIG. 441

Wasting of muscles between thumb and index finger in ulnar nerve palsy.

the interossei are paralysed. Marked wasting of the hypothenar muscles and in all the interosseous spaces, particularly between the thumb and index figure, ensues (Fig. 441). The following movements are lost. When the fingers are fully extended, the patient cannot abduct or adduct them. Adduction of the thumb is impossible, i.e., the thumb cannot be moved from the

abducted position into contact with the radial border of the palm. The metacarpophalangeal joints cannot be flexed or the interphalangeal joints extended. The hand is therefore held in the "claw hand" or *main-en-griffe* position (Fig. 442), with the metacarpophalangeal joints hyperextended and the phalangeal joints flexed. The "clawing" does not affect the index and middle fingers so much as the others, as their lumbrical muscles are not affected.

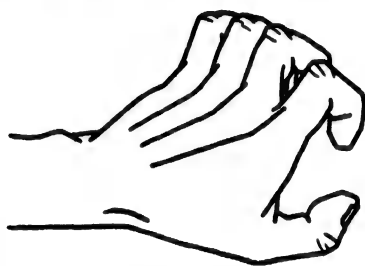


FIG. 442

The *main-en-griffe* position of the hand in lesions of the ulnar nerve.

Epicritic sensation is lost over the ulnar portion of the hand, the palmar surface of the little and the ulnar half of the

ring fingers and the dorsal surface of these two fingers from their tips half-way up them. Protopathic sensation is lost over a varying and usually very small area. Trophic changes in the anæsthetic area are well marked. When the nerve is divided above the level of origin of the dorsal cutaneous branch there is an extension of the area of epicritic loss over the dorsal area of the hand and of the ring

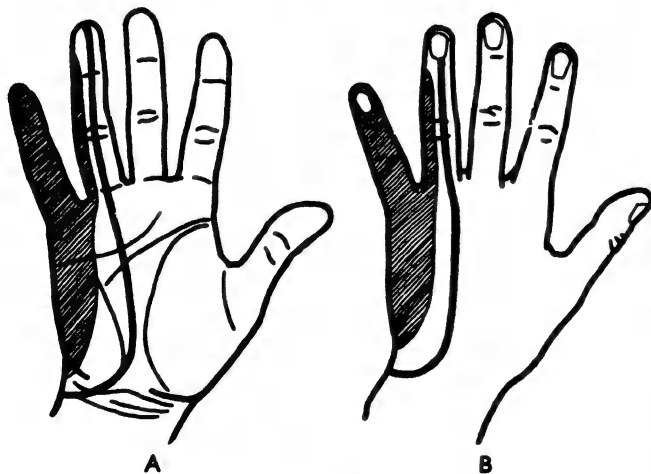


FIG. 443

Diagram showing loss of sensation after division of the ulnar nerve. A, palmar and B, dorsal aspect of the hand. Areas of shading as in Fig. 440.

and little fingers, together with a less pronounced increase in protopathic loss (Fig. 443, A and B).

**INJURY AT OR ABOVE THE ELBOW.**—The paralysis now includes the flexor carpi ulnaris and the inner half of the flexor profundus digitorum, so that further weakness in the power of flexion, of ulnar abduction of the wrist and of the grip follows. The clawing of the hand is still more marked. Loss of deep sensibility will also be present in the ulnar part of the hand.

*Treatment* is exactly similar to that for median nerve injuries and the same principles apply.

There is another injury of the ulnar nerve at the elbow which needs description, viz., **recurrent dislocation** from the groove behind the internal condyle when the elbow is flexed. If it is not treated a traumatic neuritis will be set up, and signs of incomplete physiological division follow. Operative treatment is essential, the nerve being either sutured back in the groove or preferably displaced in front of the condyle and buried in the muscles.

**The Intercostal Nerves** may be injured in fractures of the ribs, and intercostal neuralgia follows pressure on the nerve roots by neoplasms, callus, fractures of the spine, spinal caries, aneurysm or osteo-arthritis of the spine. Herpes zoster may cause intense intercostal neuralgia.

### THE NERVES OF THE LOWER EXTREMITY

*Anatomy.*—The lumbosacral plexus is formed by the anterior primary divisions of the lumbar and sacral nerves. The root supply of muscles is as follows :—

2nd and 3rd Lumbar	.	Sartorius, iliopsoas, pectineus and adductor longus.
2nd, 3rd and 4th Lumbar	.	Gracilis and adductor brevis.
3rd and 4th Lumbar	.	Obturator externus and quadriceps.
3rd, 4th and 5th Lumbar	.	Adductor magnus.
4th and 5th Lumbar and 1st Sacral	.	Glutei, semimembranosus, popliteus, quadratus femoris and gemellus inferior.
4th and 5th Lumbar and 1st and 2nd Sacral	.	Tibialis anticus, peronei.
5th Lumbar and 1st Sacral	.	Flexors of toes, tibialis posticus and abductor hallucis.
5th Lumbar and 1st and 2nd Sacral	.	Gemellus superior, obturator internus, semitendinosus and soleus.
1st and 2nd Sacral	.	Pyriformis, gastrocnemius, interossei and the two adductores hallucis.
1st, 2nd and 3rd Sacral	.	Biceps and extensors of the toes.

**The Obturator Nerve** is very rarely injured. It may be involved in dislocations of the hip and in a strangulated obturator hernia. It is more frequently divided intentionally to relieve adductor spasm in severe spastic paralysis. Complete division causes paralysis of all the adductors except part of the adductor magnus.

**The Anterior Crural Nerve** injuries are rare, except in gunshot wounds. Paralysis of the quadriceps results, with anæsthesia over the front and inner part of the thigh and on the inner aspect of the lower half of the leg around the internal malleolus. An attempt to suture the nerve should be made.

**The Great Sciatic Nerve** is commonly involved in gunshot wounds, but otherwise only a posterior dislocation of the hip will affect it. The whole nerve, its internal or external popliteal portions may be injured

the last being the most common. The injury is usually below the buttock, and so the hamstrings are not affected as their nerve of supply comes off at a higher level. Complete division leads to complete paralysis of all muscles of the leg below the knee, with absolute loss of sensation except in the area of the long saphenous nerve. Flaccid foot drop is present.

*Treatment* consists in the prevention of deformity and accurate suture of the nerve. Great care is taken to ensure that the two main divisions are in correct apposition. Prolonged massage and electrotherapy must be employed. The prognosis is unexpectedly favourable, especially in respect of the external popliteal division. If nerve suture fails, the resulting paralysis can be greatly ameliorated by suitable arthrodeses and leg irons.

SCIATICA is almost always a symptom of pressure on either the great sciatic nerve or the lumbosacral plexus. The causes are :—

1. Pressure on the nerve roots by subluxations of or osteo-arthritic changes in the spine and sacro-iliac joints, by spondylitis deformans, by malignant growths or tuberculous disease of the spine, by tumours of the cauda equina or the meninges, and by herniation backwards of the nucleus pulposus of the intervertebral discs in the lower lumbar spine.
2. Pressure on the lumbosacral plexus by the pregnant uterus, fibroids, ovarian tumours and malignant growths of the pelvic bones, prostate, rectum or uterus.
3. Pressure on the nerve trunk below the pelvis by the dislocated head of the femur, aneurysms or new growths.
4. Degenerative diseases of the spinal cord, such as tabes and syringomyelia.
5. Diseases of the hip joint.

True sciatic neuritis is unilateral, and comes on in attacks which are precipitated by exposure to damp and cold. The diagnosis of a true sciatica must *never* be made until an exhaustive examination has eliminated any cause of pressure or tension.

*Symptoms.*—Acute sciatica produces severe pain all along the course of the nerve. It may be paroxysmal or continuous, and the patient avoids any movement likely to stretch the nerve. If he walks at all, he goes on tiptoe with the knee flexed and hip slightly bent, and puts as little weight as possible on that leg. The lumbar spine and pelvis are tilted to relax tension on the nerve roots. Examination reveals tenderness on pressure over the nerve in the thigh and, later, some wasting of the muscles of the calf and of the hamstrings. Attempts to straighten the knee when the hip is flexed cause severe pain, and the deep reflexes may be diminished. The condition tends to become chronic, but an attack rarely persists for more than ten weeks.

*Treatment* consists in the discovery and elimination of the cause. The true primary sciatica is treated at first by rest in bed. Sedatives will be needed in the acute stage, and so violent is the pain in some cases that morphia is required. The limb is most comfortable if a

strapping extension is applied to the thigh to minimise movement and irritation, and also to stretch the nerve. Heat, diathermy and short-wave therapy are all useful. Later, intractable cases may be benefited by injecting saline into the nerve, oxygen into its sheath or by stretching the nerve by manipulation or operative exposure.

**The External Popliteal Nerve** may be injured in the lower part of the thigh or more commonly as it winds round the neck of the fibula. Paralysis has been known to follow the reduction of an intricate congenital dislocation of the hip of some years duration. In complete division there is paralysis of the tibialis anticus, the extensor group of the foot and the peronei. The foot is held in the position of equino-varus, and voluntary eversion and dorsiflexion are impossible. Anæsthesia commonly involves the dorsum of the foot and the lower and outer part of the leg, if the lesion is above the origin of its cutaneous branch. The nerve is frequently the site of peripheral neuritis in arsenical, lead or alcoholic poisoning.

*Treatment* consists in early suture, splinting and physiotherapy. If this fails, subastragaloid arthrodesis gives good functional results.

**The Internal Popliteal Nerve** is rarely injured, but may be involved in penetrating wounds or in the forced correction of an old-standing flexion deformity of the knee joint. Complete division paralyses all the calf muscles and all the intrinsic muscles of the foot. There is anæsthesia of the sole of the foot, of the plantar surface of the toes and of the distal part of the dorsal aspect of the four outer toes. There is a tendency to a later development of pes cavus or calcaneo-valgus.

*Treatment* consists in early suture, splinting and physiotherapy. The results are poor, but the resulting deformity is not very disabling.

Partial injuries of both internal and external popliteal nerves may cause severe causalgia.

**The Anterior and Posterior Tibial Nerves** are occasionally injured by penetrating wounds and by fractures of the tibia and fibula. The results are similar to those of injury to the popliteal nerves, but the degree of paralysis and the extent of anæsthesia is less and is determined by the level of the injury.

## THE CRANIAL NERVES

The cranial nerves are injured in fractures of the base of the skull and, after they have emerged from their respective bony foramina, by penetrating wounds or by operative mistake. In fractures the nerves may be torn completely across, when the paralysis is complete; they may be contused, in which case function will return after a period of paralysis; or there may be hæmorrhage into the sheath with a gradual loss of function. The nerves may be compressed by scar tissue, callus, gummata, tumours, aneurysms or the swelling which follows a chronic meningitis. All the cranial nerves are liable to injury, but the Vth, VIIth, XIth and XIIth are most commonly involved. Although many of these lesions are intracranial it is nevertheless the nerve trunks which are damaged and not the nerve centres, so that

the paralysis is of the lower neurone type and on the same side as the injury except in certain lesions of the optic nerve.

**Ist or Olfactory Nerve.**—Loss of smell results from lesions of this nerve produced by fractures of the cribriform plate, contusion of the anterior lobes and pressure from tumours or chronic meningitis. Tests are carried out with aromatic substances, *e.g.*, peppermint or oil-of-cloves, and not by irritant gases. The sense of taste is often impaired when smell is lost.

**IInd or Optic Nerve.**—Lesions of this nerve may be due to fractures, intracranial lesions, penetrating wounds and pressure from tumours, aneurysms or the inflammatory exudate of orbital cellulitis. The degree of loss of vision will depend on the nature of the cause and the extent and site of the damage. A pituitary tumour pressing on the optic chiasma causes bitemporal hemianopia. Pressure on the optic tract or on the occipital centre leads to hemianopia.

Optic neuritis or papillœdema is an œdema of the disc ("choked disc") and is generally caused by increased intracranial pressure. Primary optic atrophy results from pressure on the nerve or from general disease and is recognised by the clearly defined white appearance of the disc. Secondary optic atrophy follows increased intracranial pressure, the disc being grey-white and irregular in outline.

**IIIrd or Oculomotor Nerve.**—A partial paralysis of this nerve may occur in fractures involving the sphenoidal fissure or result from the pressure of hæmorrhage, aneurysm, gumma or new growth. The symptoms of a complete lesion, which is uncommon, are ptosis of the upper eyelid from paralysis of the levator palpebræ, external squint from palsy of all the recti muscles except the external, and slight downward rotation of the eye from paralysis of the inferior oblique. Diplopia or double vision is therefore present. As the constrictor of the iris is paralysed the pupil is dilated, and there is loss of accommodation and reaction to light. Exophthalmos—protusion of the eyeball—may be present to a slight degree.

When all the muscles of the eye are paralysed the condition is known as ophthalmoplegia externa and is generally due to a gumma in the floor of the third ventricle.

**IVth or Pathetic Nerve** is very rarely damaged. Its injury results in paralysis of the superior oblique, with deficient movement on looking downwards and outwards, and so causes double vision.

The *treatment* of lesions of the first four cranial nerves is the removal of the cause, should that be accessible.

**Vth or Trigeminal Nerve** or its branches may be injured in fractures of the base of the skull and compressed by hæmorrhage, aneurysm, gumma or growth. Complete division results in anæsthesia of the whole area supplied, with paralysis of the masseter, pterygoid and temporal muscles. Mastication is still possible, the opposite muscles being strong enough, but the jaw deviates towards the paralysed side owing to the action of the sound external pterygoid. Injury to the ophthalmic division affects the conjunctiva and cornea, which become insensitive to foreign bodies and so are liable to infection and ulceration.

The Vth cranial nerve is of peculiar surgical importance because of the frequency with which it is affected by irritative lesions.

**TRIGEMINAL NEURALGIA** may be due to pressure or to an irritation from infective processes of the teeth, buccal cavity, nose, nasal sinuses and middle ear, and possibly to a syphilitic neuritis. Only in the absence of all these causes of irritation can true trigeminal neuralgia or tic douloureux be diagnosed. This disease affects middle-aged people, especially women, and is one of the most painful conditions known. It is always unilateral and attacks the maxillary and mandibular divisions. The pain comes on in paroxysms, which at first are moderate in degree and are separated by considerable intervals of weeks or months, but later the attacks become more frequent and more severe. They are brought on by trifling stimuli, such as eating, washing or a puff of wind. The skin of the affected area of the face and the mucous membrane of the tongue and cheek become hypersensitive. During the attack the muscles of the jaws, and even of the neck, twitch. Eventually the pain is so severe that the patient is left completely prostrated and terrified lest any stimulus should precipitate another attack. The ophthalmic division is much less affected than the other branches, although profuse lachrymation may be present in the attacks.

*Treatment.*—Every possible source of irritation must be carefully excluded and the general health of the patient improved as far as possible. The pain in the early stages will be controlled by analgesics, but in the later attacks only morphia can give relief. Two methods of operative treatment are possible, alcohol injection and section of the sensory route behind the Gasserian ganglion. Injection should always be given a trial first and yields a high percentage of successes. A description of the two procedures is given on p. 868.

Neuralgia in some of the terminal branches of the Vth nerve occurs independently of a true tic douloureux, the supra-orbital and inferior dental nerves being sometimes affected. If no cause can be found, the nerve should be divided and a small portion removed.

**VIth or Abducent Nerve** is injured in penetrating wounds or fractures of the skull and is compressed by a gumma, aneurysm or tumour. Its paralysis results in internal strabismus and diplopia.

**VIIth or Facial Nerve.**—Lesions of this nerve result in facial paralysis and are due to disease, injury or compression of (a) the cerebral motor cortex, corona radiata and corpus striatum, (b) the nucleus in the floor of the fourth ventricle, or (c) the trunk of the nerve. Lesions above the nucleus cause paralysis of the upper neurone type, on the opposite side without the reaction of degeneration, and in these cases the lower half of the face is chiefly involved and emotional expression is not interfered with as much as voluntary movement. Subcortical lesions are usually merely a part of more widespread damage, from which a hemiplegia develops. The lesions of the nerve trunk are caused by hæmorrhage, thrombosis, aneurysm, abscess, gumma or new growth. In the nuclear and trunk affections the whole face is paralysed on the same side, the muscles atrophy rapidly,



reaction of degeneration is present and emotional expression and voluntary movement are equally lost.

**LESIONS OF THE NERVE TRUNK.**—The nerve is commonly damaged in fractures of the base of the skull, which involve the temporal bone, and it may be compressed by an acoustic nerve tumour in the internal auditory meatus. As the nerve passes through the aqueduct of Fallopius it may be involved in inflammatory diseases of the middle ear, in tuberculous foci and in malignant disease of this region; here also the nerve may be injured in operations for mastoid disease. Outside the skull the nerve may be involved in infections or growths of the parotid gland or it may be damaged during operations.

**COMPLETE FACIAL PALSY** is characterised by marked asymmetry, the affected side of the face being smooth and expressionless and the natural folds absent. The palpebral fissure is widened and the lower eyelid with its punctum lachrymale droops away from the eyeball, so that tears run down over the cheek. Voluntary and emotional movements are entirely lost and all attempts at smiling cause the face to be drawn to the opposite side. The conjunctival reflex is lost and corneal ulceration ensues. Owing to the paralysis of the orbicularis oris the patient cannot show his teeth or whistle, and pronunciation of the labials is imperfect. Paralysis of the buccinator allows food to collect between the cheek and the teeth. When the lesion is in the petrosal region the auditory nerve may also be involved. The chorda tympani may be damaged, resulting in loss of taste in the anterior part of that side of the tongue with diminished salivary secretion, or the petrosal nerve may be injured, with paralysis of that side of the soft palate.

**Treatment.**—The cause must be sought for and removed or eliminated if possible. Division of the nerve during operations should be remedied by immediate suture. The muscles of the face should be treated by electrical stimulation. If the nerve injury is due to fracture and compression by blood clot inside the temporal bone, if no sign of improvement occurs and if the reaction of degeneration has set in, the nerve should be decompressed by removal of the walls of its bony canal. If the injury is inaccessible, or if the cause cannot be removed and the reaction of degeneration is present, the operation of facio-hypoglossal anastomosis should be performed. The results are distinctly good and facial movements slowly return. The patient moves the tongue and the face together, but in time is able to dissociate the two movements. If all these procedures fail, certain plastic operations may succeed in improving the deformity of the face, even if active movements are still absent.

**BELL'S PALSY** is a condition of facial palsy due to a neuritis of rheumatic nature brought on by exposure to cold and draughts. It generally recovers in a few weeks with the administration of potassium iodide and sodium salicylate accompanied by massage and electrical treatment.

**FACIAL TIC (Histrionic Spasm)** is a painless clonic spasm of the facial muscles affecting either the whole of one side of the face, including the platysma, or certain muscles only, such as the orbicularis oris



It may be due to an irritative lesion of the brain, or may be reflex from irritation of the Vth nerve endings. The more severe forms are usually due to irritation in the petrous bone, and these may cause the patient much distress.

*Treatment* consists in removal of the cause if possible, the administration of antispasmodic medicines, counter-irritation over the nerve and improvement of the general health. The severe cases have been treated by division of the nerve followed by a facio-hypoglossal anastomosis.

**VIIIth or Auditory Nerve.**—Nerve deafness results from division of the nerve in fractures of the base of the skull, or from compression by tumours either of the nerve itself or of the cerebello-pontine angle. Irritation of the nerve causes tinnitus, vertigo and nystagmus. Tumours of the nerve or in its neighbourhood can be successfully removed.

**IXth or Glossopharyngeal Nerve.**—This nerve, lesions of which cause difficulty in swallowing (dysphagia) and in speaking (dysarthria), is very rarely damaged.

**Xth or Vagus Nerve** may be injured within the skull, in the neck or in the thorax. Intracranial conditions, such as tumours, gummata or meningitis compress the nerve, which is also injured by fractures of the skull. In the neck it may be divided or dragged on during operations, involved in new growths or malignant cervical glands or injured in penetrating wounds. Aneurysms and neoplasms may cause pressure lesions in the thorax.

If one vagus nerve is divided completely, immediate inhibition of the heart and of respiration will result, but this is usually temporary. Prolonged irritation of the nerve, however, may cause permanent cessation of the heart action and of respiration. If the site of injury is above the origin of the recurrent laryngeal nerve, there will be acceleration of the pulse rate, with palpitation and slowing of the respiration rate, with fear of suffocation, in addition to the signs of recurrent laryngeal palsy. In lesions below the origin of the recurrent laryngeal branch, these symptoms will be much less severe.

Paralysis of half of the palate will result if the injury is above the origin of the branches to the pharyngeal plexus.

THE SUPERIOR LARYNGEAL NERVE may be damaged during operations, when there will be hoarseness from paralysis of the cricothyroid muscle, and loss of sensation in the larynx, which will permit the aspiration of solid particles into the lungs.

THE RECURRENT LARYNGEAL NERVE, which is the motor nerve to the laryngeal muscles, may be damaged during operations, especially on the thyroid gland. The left nerve may be pressed on by aneurysms of the aortic arch, and the right by aneurysms of the subclavian artery. Malignant disease at the root of the neck frequently involves these nerves, especially the left. Incomplete lesions result in an abductor paralysis of the vocal cord on the affected side with hoarseness of the voice, while in complete lesions both abductors and adductors are affected and the cord takes up a midway position. These conditions can only be diagnosed by laryngoscopic methods. Bilateral

abduction palsy leads to stridor and urgent dyspnoea. Aphonia results from bilateral adductor palsy, but this condition is usually functional.

Division of the vagus or of one of its branches should be treated by immediate suture, if recognised at the time.

**XIth or Spinal Accessory Nerve** is involved in intracranial disease, in fractures of the base of the skull, or in inflamed or malignant glands of the anterior triangle, and it is particularly liable to injury during operations in the upper part of the neck. The spinal accessory nerve supplies the trapezius and the sternomastoid, but these muscles are also supplied by branches of the cervical plexus, so that injury to the spinal accessory does not result in complete paralysis unless these other nerves are damaged at the same time. The sternomastoid and the upper part of the trapezius undergo marked wasting. The shoulder droops and the scapula is rotated so that the inferior angle approaches the midline, while the superior angle forms a prominence under the wasted muscles. In injuries in the posterior triangle the branches of the third and fourth cervical are likely to be affected and the trapezius is completely paralysed, when the scapula rotates even more markedly. When the arm has been abducted to a right angle by the deltoid, the patient cannot raise it above the head. There is some winging of the scapula, but this can be controlled by the serratus magnus in the action of raising the arm in front of the body.

Division of the spinal accessory nerve should be treated by immediate suture if recognised at the time. Late suture has not been attended with much success.

**XIIth or Hypoglossal Nerve.**—Lesions of this nerve result from intracranial disease, fractures of the base of the skull, aneurysms, new growths, gummata and operations in the neck. The half of the tongue on that side is paralysed, its muscles undergo wasting and it becomes flaccid and wrinkled. When the tongue is protruded the muscles on the unaffected side push it over to the paralysed side. The disability in mastication, swallowing and speaking is not permanent.

R. Y. PATON.

## THE AUTONOMIC NERVOUS SYSTEM

The surgery of the autonomic nervous system has passed beyond its adolescent stage and many of the problems previously awaiting solution have been settled; our knowledge of its successes and limitations is more clearly defined.

*Surgical Anatomy.*—Although surgery of this system is largely that of the sympathetic side, yet the full value of this branch of surgery cannot be appreciated unless the influence of the parasympathetic system is carefully studied. Interest centres mainly upon the cervical and lumbar ganglia and the sacral plexus.

*The Sympathetic System* consists of the thoracolumbar outflow from cells situated in the intermediolateral cell column of the spinal cord in all the thoracic and upper two lumbar segments. These spinal filaments are medullated and are known as preganglionic; they connect with cells in

the outlying ganglia, whence non-medullated postganglionic fibres are distributed to their varying destinations. There is some evidence of a higher centre in the posterior region of the hypothalamus.

In the neck there are three ganglia—superior, middle and inferior—through which fibres pass to the upper extremity, head and neck. There are ten to twelve ganglia in the thorax, four in the lumbar region and four to five sacral.

*The Parasympathetic System* or craniosacral outflow has a much more limited field of origin, arising from cells in the midbrain, bulb and sacral region of the cord. Their medullated preganglionic fibres are of considerable length as they end in ganglia in or close to the viscera which they innervate. The tectal outflow arises in the nucleus of the third nerve in which it leaves the brain; the bulbar outflow is concerned with the facial, glossopharyngeal and vagus nerves; the sacral filaments arise in the 1st, 2nd, 3rd and 4th sacral segments.

*Physiology.*—Generally speaking the sympathetic system is vasoconstrictor; in hollow viscera it closes sphincters and relaxes the remainder of the muscle of the organ concerned. It also produces glandular activity, dilatation of the pupils and increase in the pulse rate. Parasympathetic nerves are vasodilator and in hollow viscera relax sphincters and activate the peristaltic musculature. These effects are due to a chemical secretion produced by and acting upon the synapses, that liberated by sympathetic fibres being adrenalin, that by the parasympathetic acetylcholine.

It will be readily understood that in internal organs these two divisions of the autonomic nervous system must be closely co-ordinated and their proper function demands perfect timing. Imbalance between them is likely to lead to various clinical syndromes calling for surgical relief.

## INDICATIONS FOR AUTONOMIC SURGERY

These can be classified as follows :—

### I. *Diseases of the Cardiovascular System.*

#### A. Of extremities.

1. Raynaud's disease.
2. Thrombo-angiitis obliterans.
3. Acrocyanosis.
4. Erythromelalgia.
5. Erythrocyanosis.
6. Cervical rib.
7. Poliomyelitis.
8. Chronic ulcers.
9. Arterial spasm.
10. Threatened gangrene.

#### B. Of head.

1. Retinitis pigmentosa.
2. Ménière's syndrome.
3. Vertigo.

#### C. Angina pectoris.

#### D. Essential hypertension.

II. *Visceral Disease.*

1. Plummer-Vinson syndrome.
2. Cardiospasm.
3. Idiopathic dilatation of colon.
4. Renal sympatheticotonus.
5. Cord bladder.

III. *Relief of Intractable Pain.*

1. Inoperable visceral growths.
2. Painful amputation stumps.
3. Painful ulcers.
4. Causalgia.

IV. *Hyperidrosis.*V. *Intractable Dysmenorrhœa.***CARDIOVASCULAR DISEASE**

Pain in and pallor of peripheral structures, *e.g.*, fingers and toes are an indication of some embarrassment of the arterial supply to the parts. In one group of such diseases the changes in the vessel wall are degenerative, and fibrosis and calcification lead to loss of elasticity and narrowing of the lumen; in other conditions, however, the arterial wall is not diseased but is in a state of spasm, the calibre of the vessel being so reduced that peripheral ischæmia results. It must be obvious that no operation upon the autonomic system can have any beneficial effect after the arterial wall is permanently damaged by organic changes. It is in the vasospastic diseases and the earliest stages of arterial degeneration that sympathectomy serves a very useful purpose.

*Tests of Suitability.*—It is right, therefore, that we should inquire if any reliable tests are available to differentiate spasm from organic narrowing. A number of such tests have been worked out, each designed to demonstrate vasodilatation consequent upon temporary sympathetic paralysis. This is measured by the rise in skin temperature as recorded by a sensitive thermometer (thermocouple or oscillograph). Two methods are generally practised; first, the induction of reflex vasodilatation by immersing one limb in hot water and measuring the temperature of the other; and second, by paralysing vasoconstrictor fibres by injecting 2 per cent. novocain into the stellate ganglion, ulnar nerve at elbow or median at wrist, while the sciatic or external popliteal can be similarly treated. With maximum dilatation the skin temperature should rise to 36° C.; if under the condition of these tests it fails to reach 30° C. then spasm is slight, organic constriction marked and sympathetic surgery is unlikely to achieve a favourable result.

**A. In the Extremities.**—RAYNAUD'S DISEASE has been described on p. 175. In the upper limb the sympathetic trunk is divided below the 3rd thoracic ganglion with section of the white rami of 2nd and 3rd thoracic nerves. That of the first thoracic must be preserved lest Horner's syndrome develop. To prevent regeneration the upper end is dissected up and stitched into the stump of scalenus anticus. In the lower limb a lumbar ganglionectomy will be required.

**THROMBO-ANGIITIS OBLITERANS** is described on p. 265. Sympathectomy should be performed only if tests reveal a marked spasmodic element. In all cases operation should be bilateral. Prognosis is far from good.

In **ACROCYANOSIS** the pain, tenderness, swelling and cyanosis of the hands, together with any trophic changes, are usually greatly improved by sympathectomy.

In **ERYTHROMELALGIA** redness and pain appear to present a picture quite opposed to Raynaud's disease, nevertheless lumbar sympathectomy often gives relief. In erythrocyanosis a plum-coloured change is seen in the legs in women, chiefly upon the antero-lateral surfaces. Later nodular patches appear in the subcutaneous tissues and these break down to form indolent ulcers. For these later manifestations lumbar sympathectomy gives brilliant results.

**CERVICAL RIB.**—Certain cases of cervical rib and of the so-called scalene syndrome (p. 356) do not gain relief from rib removal or scalene section. Such patients may improve after cervical sympathectomy.

**POLIOMYELITIS AND CHRONIC ULCERS** both show improvement from sympathetic surgery.

**ARTERIAL SPASM AND THREATENED GANGRENE.**—Apart from thrombo-angiitis obliterans ischæmia and threatened gangrene are usually due to organic disease and not to spasm. Sympathectomy cannot be considered unless tests show a marked degree of spasm.

**B. Of the Head.**—Resection of the upper cervical ganglion has been performed in an attempt to relieve those most difficult conditions, retinitis pigmentosa, Ménière's syndrome and vertigo. In none can the results be claimed as encouraging.

**C. Angina Pectoris.**—Much work is being done on this subject. Although improvement can be expected from removal of the stellate and upper four thoracic ganglia, the patient's condition is not conducive to extensive and dangerous surgical procedures, especially when paravertebral injections may achieve a similar result.

**D. Essential Hypertension.**—Provided that organic cardiac and renal disease can be excluded in patients below the age of 50 years, fair results may be anticipated after section of the splanchnic nerves in the thorax and of the coeliac and upper two lumbar ganglia upon both sides. It is too early to assess the true merits of this drastic procedure.

### VISCERAL DISEASE

**Plummer-Vinson Syndrome.**—This disease of women—dysphagia, anæmia and atrophy of the mucous membranes of the tongue and pharynx—has been relieved in certain cases by removal of the upper cervical ganglion.

**Cardiospasm.**—Although section of the left gastric artery has been recommended, the periarterial sympathectomy thus performed gives most discouraging results. Regular dilatation is more satisfactory.

**Idiopathic Dilatation of the Colon.**—Since Telford's discovery that spinal anæsthesia cures this condition, lumbar ganglionectomy is no longer indicated.

**Renal Sympatheticotonus.**—Certain cases of hydronephrosis in which no cause can be demonstrated are believed to be due to achalasia. Only such cases as show a marked acceleration in emptying time of the pelvis after intramuscular injection of eserine (gr.  $\frac{1}{100}$ ) are suitable for sympathetic denervation. This is performed by a meticulous stripping of the renal pedicle.

**Cord Bladder.**—Retention with overflow or large quantities of residual urine without organic obstruction are sometimes due to loss of power in the parasympathetic. These conditions are associated with changes in the sacral segments of the cord, often so small as to yield no clinical evidence or, at most, a small area of anæsthesia around the anal orifice. Such patients are greatly improved by resection of the presacral nerve.

No good results can be expected from this operation in gross lesions of the cord.

### INTRACTABLE PAIN

**Inoperable Visceral Growths.**—Apart from angina pectoris and dysmenorrhœa, sympathetic surgery holds out little hope of relief to sufferers from inoperable internal growths.

**Painful Amputation Stumps and Ulcers.**—In both these conditions appropriate sympathectomy often leads to dramatic improvement.

**Causalgia** results from an incomplete division of nerves or their involvement in dense scar tissue. As a last resort, after all local measures have been tried and failed, sympathectomy sometimes brings relief.

### HYPERIDROSIS

Sweating hardly seems to justify operation, but excessive sweating may lead to loss of employment and acute mental distress. Sympathectomy is completely successful in curing this embarrassing condition.

### INTRACTABLE DYSMENORRHOEA

The position of sympathetic surgery in this disease is discussed in Chap. XXXIX. Suffice it to say here that presacral neurectomy is to be considered only when other measures have failed.

R. M. HANDFIELD-JONES.

## CHAPTER XLIV

### INJURIES OF BONES AND JOINTS

**SURGICAL ANATOMY—Bones.**—The bones of the skeleton are divided for purposes of classification into long bones, short bones, flat bones and irregular bones. The long bones are found in the limbs and consist of a shaft and two articular ends. The shaft is a hollow cylinder enclosing a space, the medullary cavity. The walls of the shaft are formed of compact bone lined by a few scattered trabeculæ of cancellous bone, which towards the ends become more numerous and the medullary cavity correspondingly smaller. The articular ends are expanded, and consist of cancellous bone enclosed in a thin compact layer and capped on their articulating surface with hyaline cartilage. The medullary cavity contains yellow marrow, a fatty tissue with few cells; the interstices of the cancellous tissue are filled with red marrow, consisting of blood spaces and groups of large hæmatopoietic cells.

During the period of growth, the articular ends are separated from the shaft by a plate of cartilage, the epiphyseal cartilage. The articular end is then called the epiphysis, the shaft the diaphysis, and the part of the shaft next to the epiphyseal cartilage the metaphysis. Growth in length takes place entirely at the metaphysis, growth in thickness is due to the laying down of new bone on the outside of the shaft by a layer of osteoblasts on its surface. The growing bone is more vascular than that of an adult, and contains a greater proportion of organic material; it can be bent considerably without breaking, and may be broken in part of its thickness only; when broken completely, the ends tend to be jagged and serrated. The metaphysis, where the blood supply is abundant and new bone is being laid down, is the weakest part.

Bone is covered by periosteum, a fibrous limiting membrane which is firmly adherent at the epiphyseal line and at the point of insertion of tendons or ligaments, but is elsewhere loosely attached. The periosteum of a growing bone is more vascular and more easily stripped than that of an adult bone, and when raised carries with it the superficial layer of osteoblasts.

The blood supply of the superficial layers of compact bone is carried by small vessels in the periosteum derived from those in the neighbourhood. The shaft is supplied by one or more nutrient arteries, which enter through foramina that are constant for each bone, and divide into two main branches, whose terminations reach the metaphyses. The articular ends receive their supply from a vascular ring, the circulus arteriosus of Lexer, which surrounds the bone at the level of the epiphyseal cartilage and gives off branches both to the epiphysis and to the metaphysis.

The short, flat and irregular bones consist of cancellous tissue enclosed by a layer of compact bone that is thin, except where it transmits stress. The cancellous tissue of flat bones receives the special name of *diploë*.

**Joints.**—The joints of the body vary considerably in size, strength and in the movements they permit. The capsule is a strong fibrous structure enclosing the joint space, and attached to the bones taking part in the

joint, being blended at the point of attachment with their periosteum. It is reinforced by ligaments, which in some cases are thickened parts of its wall, in others separate bands outside its wall, and in others lie inside the joint cavity. As a rule the portion of the bone included in the joint corresponds to the epiphysis, but in some joints the metaphysis is also intra-articular. That portion of the bone which is in contact with the other bones forming the joint is covered by a layer of hyaline cartilage; the remainder is covered by synovial membrane. The synovial membrane is a vascular tissue lined by a layer of flattened cells, and covers the inner aspect of the capsule, any intra-articular ligaments or plates of fibro-cartilage, and the bones as far as the edge of the articular cartilage. Whenever the articular ends of the bone do not accurately fill the space enclosed by the capsule, the interstices are filled either with fatty pads covered by synovial membrane, or, as in the knee, temporo-mandibular, and acromio-clavicular joints, by plates of fibro-cartilage similarly covered. The synovial membrane secretes the synovial fluid, consisting largely of mucin. In a healthy joint this is small in amount, the fluid produced being absorbed by the lymphatics; such absorption depends upon the movements of the limb and the maintenance of intracapsular tension by the surrounding muscles. The blood supply of the synovial membrane is abundant, and is derived from the *circulus arteriosus*.

## INJURIES OF BONE

**Contusions** of bone are caused by the application of force insufficient to cause fracture. Two types are described.

1. **SUBPERIOSTEAL HÆMATOMA** is due to a blow upon an exposed bone such as the skull or the tibia. The periosteum is raised, carrying with it a layer of osteoblasts, and blood collects between it and the bone. Such a hæmatoma may be absorbed, become encysted or be converted into bone.

2. **CANCELLOUS HÆMATOMA** is due to a twisting or bending force, and occurs at the metaphysis of a growing bone. Such hæmatomata usually pass unrecognised, but they may become infected by blood-borne organisms, leading to osteomyelitis or they may cause abnormalities in epiphysial growth.

**Fractures.**—A fracture is a sudden and violent solution of continuity in a bone. Fractures are divided into pathological fractures, where weakening of the bone is the dominant factor and trauma is slight or absent, and traumatic fractures due to the application of violence or strain to a healthy bone.

**PATHOLOGICAL FRACTURES** may be due to :—

1. Congenital diseases of the skeleton. *Osteogenesis imperfecta* leads to fractures during intra-uterine life and early childhood, but the tendency disappears after puberty. *Fragilitas ossium* is a disease, often familial, in which the liability to fracture appears after birth and continues into adult life.
2. Metabolic diseases affecting the skeleton. Scurvy rickets in infancy, renal rickets in adolescence, and osteomalacia in adult life, all lead to a disappearance of calcium from the



bones that makes them liable to fracture. Adenomata of the parathyroid give rise to generalised fibrocystic disease of bone, with similar results. The common form of rickets does not predispose to fracture.

3. General bone diseases of unknown origin—osteitis deformans, and myelomatosis.
4. Atrophy due to old age or wasting diseases.
5. Nervous diseases; general diseases such as tabes, general paralysis of the insane or syringomyelia; lower motor neurone lesions such as infantile paralysis.
6. Localised atrophy from pressure.
7. Inflammatory diseases, especially localised gumma.
8. Innocent new growths; chondroma, giant-cell tumour, or localised fibrocystic disease, *i.e.*, single cysts.
9. Malignant new growths: primary sarcoma, or bone metastases of carcinoma arising elsewhere.

Many pathological fractures unite readily, others slowly, while some never join.

TRAUMATIC FRACTURES are caused by the application to a healthy bone of violence sufficient to break it. Such violence may be *external*, either *direct*, applied to the bone at the point of injury, or *indirect*—bending, twisting or compression strains—usually applied in the long axis of the bone; or *muscular*, the sudden and inco-ordinated contraction of some powerful muscle or group of muscles.

### CLASSIFICATION OF FRACTURES

#### A. ACCORDING TO THE NATURE OF THE INJURY TO THE BONE

Fractures are divisible into two main groups, incomplete and complete.

1. **Incomplete Fractures.**—The term should be restricted to those which do not involve the whole thickness of a bone; it is often extended to include fissured fractures which pass right across the bone, but in which there is no separation.

- (a) *Fissured Fractures* occur chiefly where the shell of compact bone is thin, that is, in flat bones and the articular ends of the larger long bones.
- (b) *Greenstick Fractures* are seen in the long bones of children and are caused by indirect violence. The bone is broken transversely on the convexity of a curve, and bent or compressed on the concavity (Fig. 444).
- (c) *Cancellous Fractures* are caused by direct or indirect violence applied to the short and irregular cancellous bones and the articular ends of long bones, and by indirect violence acting on the metaphysis of a growing bone.
- (d) *Depressed Fractures* are usually seen in the skull, but may occur in other flat bones or in large cancellous bones. They are due to the application of direct violence over a small area.

## 2. Complete Fractures.

(a) *Single Fractures* are those in which the bone is broken into two fragments. They may be transverse, vertical, oblique or spiral.

(b) *Comminuted Fractures* in which the bone is broken into several pieces.

3. **Impacted Fractures** are complete fractures, in which one fragment has been driven into the other, producing some degree of interlocking.



FIG. 444

Anteroposterior and lateral views of a greenstick fracture of the radius.

Impacted fractures are usually seen in adults near the articular ends of long bones, the shaft being driven into the cancellous tissue of the end.

4. **Separation of Epiphyses** occur during childhood and adolescence. In the majority the injury is not a pure separation, the line of cleavage passing into the metaphysis, juxta-epiphysial fracture being a more accurate description.

5. **Complicated Fractures** are those in which other important structures, such as vessels or nerves, are also damaged.

6. **Fracture Dislocations**, in which the fracture is into or near a joint and accompanied by a dislocation of that joint, e.g., Pott's fracture.

## B. ACCORDING TO THE NATURE OF THE VIOLENCE CAUSING THE FRACTURE

1. **Closed or Simple Fractures** are those in which there is no communication between the surface and the broken ends of the fragments.

2. **Open or Compound Fractures** are those in which a wound on the surface communicates with the site of fracture; the presence of a

wound does not make the fracture an open one unless it leads to the broken ends of the bone. Direct open fractures are those in which the wound of the soft parts lies over the site of injury to the bone; they are usually caused by direct violence, and the soft parts are lacerated and soiled by dirt or clothing. Indirect open fractures are due to perforation of the skin by the end of one of the fragments; they are commonly caused by indirect violence, and the opening is small, not necessarily contaminated, and after reduction of the displacement may lie at some distance from the site of fracture.

### REPAIR OF FRACTURES

When a bone is broken, the site of fracture is filled with blood clot, the extent of which depends on the separation of the fragments and the degree of laceration of the surrounding tissues. The clot is first converted into granulation tissue, indistinguishable from that replacing an ordinary hæmatoma. Osteoblasts derived from the bone fragments then invade the granulation tissue, and lay down calcium in the intercellular substance. Osteoblasts are most numerous under the periosteum, less in the endosteum, and scanty in the compact bone; therefore, calcium is laid down in the outer part of the callus first, next in the inner part, and lastly in the middle zone. As calcium is deposited the tissue assumes a granular appearance under the microscope and stains deeply with hæmatoxylin. This calcified repair tissue is called **callus**, and the three zones are named external, intermediate and internal callus (Fig. 445). Calcification commences about the tenth day. After three weeks it is sufficient to throw a shadow on an X-ray film, and the callus can be felt clinically as a firm rounded mass at the site of fracture; at this time the bone can be bent, but the fragments cannot be displaced without some force. After four to eight weeks, the time depending upon the bone and the age of the patient, the callus is sufficiently firm to prevent any movement, and union is said to have occurred.

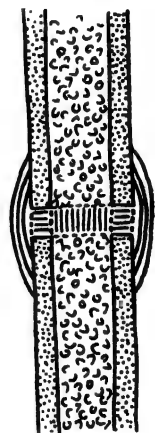


FIG. 445

Diagram showing the three types of callus as described in the text.

At the time of union the fragments cannot be displaced without some violence, but the fresh callus may be deformed if it is subjected to a more gradual strain. The repair is made strong and permanent by the gradual replacement of callus by true bone, a process called consolidation in the clinical, ossification in the pathological sense. The calcified connective tissue is absorbed by osteoclasts, and true bone arranged in Haversian systems is laid down round the blood vessels by osteoblasts. At the same time the callus surrounding the shaft, and that filling the space of the medullary cavity between the fragments, is removed, so that the bone finally resumes a shape and structure approximating to its former state. If union has occurred in a position of deformity, the new bone is laid down in buttresses that

tend to correct the mechanical weakness inherent in that deformity. The process of consolidation requires roughly twice the time necessary for union in any bone. It is complete when the site of fracture is no longer palpable or tender, and when an X-ray shows trabeculation across the site of union.

Large amounts of callus are laid down when there is a large hæmatoma at the fracture line, when the fragments are comminuted, and when movement takes place at the site of fracture during union; excessive callus formation is common in childhood, especially in fractures near the elbow. Little callus is formed when there is scant blood clot and little movement of the fracture, as in partial fractures, and fractures of bones that are immobilised by others, such as those of the skull and pelvis.

**Delayed Union** may be due to local or general causes. Among the first are the conditions described above as leading to diminished callus formation, wide separation of the fragments, the interposition of soft parts, infection of a compound fracture, or poor blood supply to one or both fragments. Among general causes may be mentioned diseases of the skeleton, especially rickets, osteitis deformans and syphilis, and systemic diseases leading to deficiency in all processes of repair. Union is said to be delayed if it has not occurred in two months; if the delay exceeds a year, there is said to be non-union.

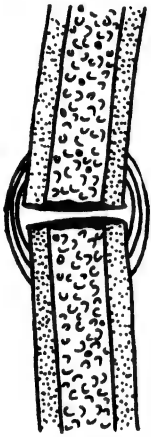


FIG. 446

Diagram illustrating the formation of a false joint.

A special warning is needed to emphasise the danger of skeletal traction, which has become so popular a method of treatment. Over-extension of the limb with wide separation of the fragments is very likely to occur unless careful watch is kept on the fracture during the early part of treatment. At the present time this over-extension during skeletal traction is the commonest cause of delayed union.

**Non-union** is of two types. In one the ends of the bone are united by repair tissue, which, however, is not converted into callus. The fragments are little altered in shape or structure, but in time tend to become dense and avascular on their free surfaces. The term *fibrous union* is often employed to denote non-union that nevertheless gives satisfactory function; if the fractured bone is one not subjected to compression or angulation and if the space between the fragments is small and the band of fibrous tissue firm, such a result will give little disability.

In the other type of non-union a false joint, or *pseudarthrosis* (Fig. 446), develops between the fragments. The ends become smooth, dense and avascular, and between them a bursal cavity lined by fibrous tissue develops. Masses of bone tend to grow round such a pseudarthrosis, forming visible or palpable tumours. The formation of a false joint is usually due to excessive movement during the process of union.

**Malunion** is union in a position of deformity with shortening,

angulation, rotation or lateral displacement at the site of fracture. Malunion, by disturbing the relationship of the joints above and below the fracture to each other, interferes with their function and, if left uncorrected, may lead in time to osteo-arthritic changes.

### DIAGNOSIS OF FRACTURES

#### *Symptoms and Signs of Fracture.*

1. **History.**—The nature of the accident will often indicate whether a fracture is likely and its probable site. The patient usually knows that he has broken a bone.

2. **Interference with Function.**—In all fractures, there is some loss of function in the injured part; this is less in partial and impacted fractures, and in those of one bone in the leg or forearm.

3. **Pain** is present at the site of fracture and is increased by movement. Pain in a bone at some distance from the point of application of violence is especially suggestive of fracture.

4. **Swelling** round the fracture occurs early and is due to the effusion of blood. Such swelling is usually visible, but in a deep-seated bone may be appreciable only by palpation. Later, the swelling is increased by œdema, which in some fractures, notably in those of the lower end of the humerus and the leg and ankle, may be extreme.

5. **Deformity**, or alteration of the normal alignment of the parts, is present except in partial fracture, or in fracture of one bone of two. The deformity may be one of shortening, lengthening, angulation, rotation or lateral displacement, or a combination of two or more of these.

6. **Abnormal Mobility.**—When a bone is broken, movement becomes possible in a place where there is no joint. Such abnormal movement may be obvious when the patient attempts to use the limb, or may only be discovered on handling the part. It is absent in partial and impacted fractures, and in those of one bone of two, or of a bone otherwise supported.

7. **Tenderness** is present at the site of fracture, and may be elicited by pressure at this spot, or by compressing the injured bone at some distant point.

8. **Crepitus** is produced when the fragments are rubbed together. It may occur when the patient attempts to use the part, or may only be noticed upon handling: *no attempt should be made to produce crepitus* if it is not noticed during the examination necessary to establish a diagnosis. The crepitus of fracture is a coarse grating that is easily felt and may be heard. A softer crepitus is found in osteo-arthritis, pleurisy, tenosynovitis and surgical emphysema.

9. **Absence of Transmitted Movements.**—The humerus, radius and femur are deeply placed in the upper part of their shafts, but the head can in each case be felt. In fracture, movements of rotation applied to the shaft are not translated to the head.

Of the above symptoms and signs only four—deformity, crepitus, abnormal mobility, and absence of transmitted movement, are absolute evidence of fracture.

**X-ray Examination.**—Skiagrams in two planes must be taken of every fracture at the earliest opportunity in order to confirm the diagnosis and demonstrate the position of the fragments. Further skiagrams should be taken after reduction to show whether the position is satisfactory. During the course of treatment it may be advisable in many cases to check position and estimate the progress of repair by other examinations. Once the fracture has been reduced and apparatus applied, it is inadvisable to risk disturbing the position for purposes of examination; such confirmatory X-rays must in most cases be taken through the splints and, if the patient is being treated by traction, with a portable apparatus brought to the bedside. After union, a final skiagram is advisable to decide whether consolidation is complete.

The treatment of a fracture is always a responsible task, and its anxiety is increased by the fact that the majority of legal actions for negligence are concerned with these injuries. Skiagrams are available to the patient as well as to the doctor, and it is important that these should be taken at the suggestion of the attendant rather than the patient in all cases where fracture is known, suspected or even possible. The fact that X-rays have been taken both before and after reduction of the fracture is accepted in law as proof that the patient has been treated with reasonable care and skill, even if the result is poor.

## TREATMENT OF FRACTURES

The general principles of fracture treatment will be considered under the following headings :—

1. The restoration of the fragments to their correct position ;
2. Maintenance of correct position till union has taken place ;
3. Protection of the bone after union from stresses likely to produce deformity till consolidation is satisfactory ; and
4. Preservation of the function of neighbouring muscles and joints during treatment.

### THE RESTORATION OF FRAGMENTS TO CORRECT POSITION

The replacement of a fractured bone in correct position is called "reduction" or "setting." A fracture should be reduced as soon as circumstances permit, because complete replacement is more easily attained before the deformity is fixed by spasm and swelling has obscured the outlines of the parts, and because early reduction places the injured parts at rest and therefore lessens shock. Reduction may be performed by one of the following methods :—

**A. Manipulation.**—The fragments are replaced by carefully controlled manual force, the exact manœuvre to be employed depending upon the site of fracture and the displacement. In most cases the distal fragment is brought into alignment with the proximal.

Manipulation may be performed without anæsthesia if the fracture is easily reduced, as when there is a displacement of angulation only, and if the patient is not unduly apprehensive. Anæsthesia is usually

preferable. Gas is suitable where the fracture can be reduced and splints applied within a few minutes. For most cases gas allows too short a time and gives insufficient relaxation, and local or general anæsthesia should be employed. Local anæsthesia is very suitable for the reduction of a fracture, since no elaborate technique is required ; 2 per cent. novocain is used, the injection of from 10 to 40 c.c. into the space between the fragments producing anæsthesia appearing in a few minutes and lasting for one or two hours.

**B. Gradual Traction.**—Certain fractures, notably oblique or very comminuted fractures of the long bones, may be reduced by a pull in the axis of the limb, but become redisplaced when the pull is relaxed. In such cases traction is employed both for the reduction and subsequent fixation. Gradual traction is also necessary for the reduction of fractures that are first seen after the lapse of some days, when muscle spasm, infiltration of soft tissues, and early changes of repair round the fragments make replacement by manipulation impossible.

**C. Operation or Open Reduction** is necessary when replacement by other means has failed or is impossible. The chief indications for open reduction are the interposition of soft parts between the ends, gross displacement of a detached fragment, wide separation in traction fractures, failure to secure reduction by manipulation because of the shape of the bone ends, or severe concomitant damage to nearby important structures.

### THE FIXATION OF FRACTURES

A fractured bone must be retained in correct position after reduction till union has occurred. Such fixation may be external, by splints ; or internal, by screws, wires, plates or pegs controlling the bone itself.

**Splints** fall into two main categories, those for fixation and those for traction. The first are used in fractures which, when reduced, show little tendency to redisplacement and can be retained, either by simple immobilisation and protection, or by maintenance of the limb in some special position ; the latter are necessary in those fractures which tend to redisplacement after reduction.

**FIXATION SPLINTS.**—The following comprise the splints commonly employed for the treatment of such fractures as can be maintained in position after reduction by fixation alone.

(a) *Gutter Splints.*—These are strips of wood or metal, hollowed out on the side that is placed in contact with the limb, and made in assorted sizes, ranging from 6 by  $1\frac{1}{2}$  in. to 18 by  $2\frac{3}{4}$  in. Wooden gutter splints are only suitable for splinting one segment of a limb. Metal gutter splints may be bent opposite a joint, or twisted in their long axis, and have therefore a greater range of application ; they may be made of iron or aluminium, and are usually sold with a covering of felt glued to the concave side. Aluminium splints have the advantage that they are translucent to X-rays. Two special varieties of gutter splints are Gooch splinting, consisting of parallel wood laths glued to a canvas backing, which is sold in sheets and is useful for an emergency outfit, since gutter splints of any size and shape can be cut from it,



and Cramer's skeleton wire splints, made from galvanised wire in the form of a ladder, which can either be applied singly or used to form more complex splints by fastening two or more together by twists of wire or strapping.

(b) *Plaster of Paris* is an anhydrous sulphate of lime. When dry it is an amorphous white powder; when mixed with water it "sets" in from seven to ten minutes to form a homogeneous white mass, possessing considerable strength. For the purpose of making splints, some form of fibrous material is always incorporated in the plaster of Paris, to give it the necessary tensile strength. In practice, plaster bandages are used for all surgical purposes. These are made from "book muslin," into the meshes of which plaster of Paris of the fine variety known as "dental" plaster is rubbed; the bandages are made in widths varying from 2 to 8 in., and are rolled very loosely, so that when immersed in water they quickly become saturated.

*The Plaster Case* is a complete splint, enveloping a limb or segment of a limb, made by applying plaster bandages in quick succession and rubbing in each layer as it is put on. Since plaster expands slightly in setting, the case may be applied directly to the skin, where there is no risk of the limb swelling afterwards. Alternatively, a layer of dressmakers' wool is put over the limb, either as a complete covering or over subcutaneous bony prominences, before the plaster is applied. Seven or eight layers of plaster bandage form a case of sufficient strength, but extra layers should be incorporated at points of special stress.

The plaster case affords a completeness and security of fixation that is unequalled by any form of stock splint, since it is made to fit the individual limb. It can be varied in many ways; windows may be cut to allow access to wounds or faradic stimulation of muscles, or larger spaces may be left and bridged by struts of malleable iron. The case may be bisected, either immediately or after some weeks, and removed as required for dressings or massage. A plaster case should rarely be used in the treatment of a fracture by traction, since the pressure of such a rigid material, however well padded, is very prone to produce sores.

Plaster gutter splints are made by unrolling a soaked plaster bandage, and folding the layers backwards and forwards over each other on a surface of wood or glass, rubbing the whole together. The resulting slab is applied to the limb wet, and held with turns of gauze bandage. When set, the splint is trimmed, if necessary, and bound round the edges with adhesive strapping.

(c) *Special Fixation Splints*, designed each for some particular part of the body, are numerous, and each hospital favours particular patterns.

**TRACTION SPLINTS.**—When the line of a fracture is such that, after complete reduction, there is no mechanical security, fixation must be obtained either by operation or by continuous traction. Many different splints may be used for traction. The majority consist of a metal skeleton enclosing the limb, having at its proximal end some padded surface bearing against part of the patient's body, and at its



distal end, which lies beyond the limb, a point for fixation of the traction cord.

1. *Thomas's Knee Splint* for the lower limb consists of two iron side bars united at the lower end by a broad W to which the extension cord is tied, and welded at their upper end to an iron ring set obliquely. The ring is padded with felt covered by basil leather, and is designed to bear on the tuber ischii (Fig. 447).

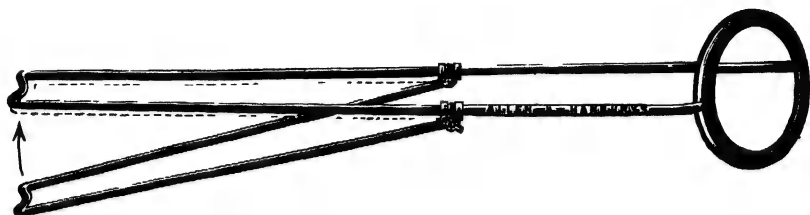


FIG. 447

Thomas's knee splint with flexion piece. (Allen & Hanburys.)

A knee flexion attachment is commonly used with the Thomas's splint. This is a replica of the distal half of the splint, and is fixed to the side bars by small metal clamps bearing hinges, so that the leg and foot, which rest in it, can be set at different angles to the thigh, or knee movements practised.

2. *Thomas's Arm Splint* is like the leg splint, but made of lighter material. The ring is set at right angles to the side bars, and may be hinged.

3. *Robert Jones's Humerus Splint* consists of a ring encircling the shoulder, the lower part resting in the axilla being padded and covered with leather. Side bars, springing from the highest and lowest points of the ring, are carried down on the outer and inner sides of the arm to a point 4 or 5 in. below the elbow, where they are bent in a W for the attachment of extension cords. They are brought up again to the level of the forearm, and extended to the wrist where they are united in a broad U (Fig. 448).

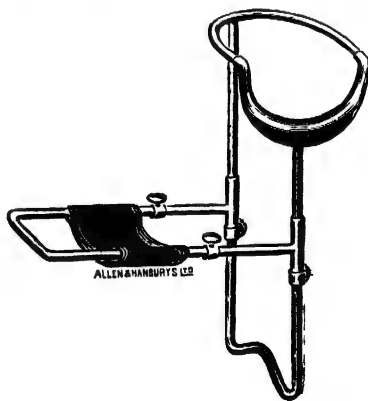


FIG. 448

Robert Jones's humerus splint slightly modified. (Allen & Hanburys.)

Many other splints are used for traction, and it is often possible, especially in the arm, to apply traction effectively without the use of any splint.

**METHODS OF APPLYING TRACTION TO A LIMB.**—The pull may be taken from the surface of the limb, either by taking advantage of the contour of the part, or by means of adhesive substances fixed to the skin; or it may be taken from the bone. The first method is known as surface traction, the second as skeletal traction.

*Surface Traction.*—When there is a change in the diameter of a limb, such as occurs at a joint, traction may be made from a band

which encircles the limb above its widest part. Thus a padded loop may be fixed at the wrist or ankle from which traction is made on the hand or foot. The arm or leg may be pulled upon by flexing the elbow or knee, and attaching the extension apparatus to a band passing round the forearm or calf. These methods can usually be employed for short periods only, and are therefore of limited value.

Traction by adhesives is more commonly employed, the substance commonly used being zinc oxide strapping.

*Application of Strapping Extension to the Leg.*—A piece of 3-ply wood, 3 in. by  $3\frac{1}{2}$  in., with a hole bored in the centre, is used as a spreader. This should lie about three fingers breadth below the sole of the

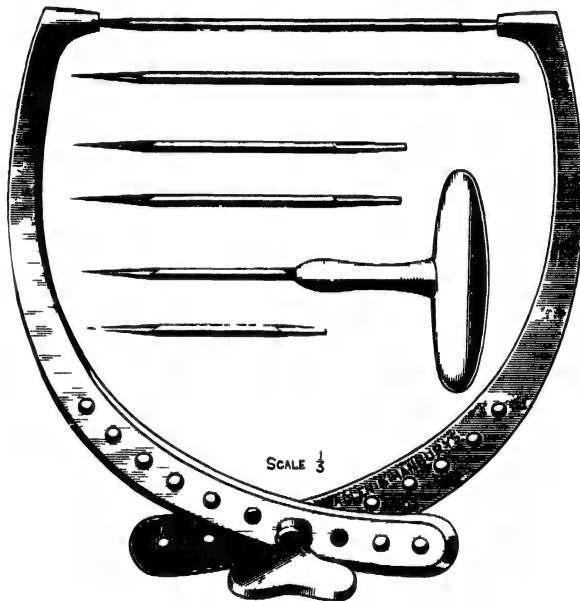


FIG. 449

Steinmann's pin apparatus, showing pins, introducer and caliper. (Allen & Hanbury's.)

foot, and a piece of  $2\frac{1}{2}$ -in. strapping should be cut of such a length that, with its centre over the spreader, the two ends will extend on each side as far as the knee, or in the case of high fractures of the femur, to the middle of the thigh. That portion of the strapping which extends from the spreader to just above the malleoli on each side should be rendered non-adhesive by fastening strips of calico bandage or strapping over its sticky surface. The limb is shaved, the spreader is held below the foot, with its hole in the plane of the malleoli, and the two bands of strapping are pulled tight and pressed against the skin in the central axis of the limb on each side. The skin bands are kept in position by further sections of strapping which encircle the limb but do not quite meet; alternatively, a continuous wrapping of elastoplast, an adhesive material which, being resilient, does not constrict the limb or fall into folds, may be applied. A strong cord, knotted at its proximal end, is passed through the hole in the spreader and

passed over a pulley to take the weights. Finally, pads of wool are placed between the malleoli and the side bands, and the foot and leg are covered with turns of flannel bandage.

*Skeletal Traction.*—Several types of apparatus are used for making traction directly on a bone. In each case some part of the instrument must be introduced through punctures in the skin. The bone may be pulled by points pressed against its surface, by a band passing over a prominence such as the os calcis, or by a pin or wire passing through its substance. Traction may be made from the distal end of the fractured bone, or the proximal end of the bone immediately distal, in which case the force is transmitted through the articular ligaments.

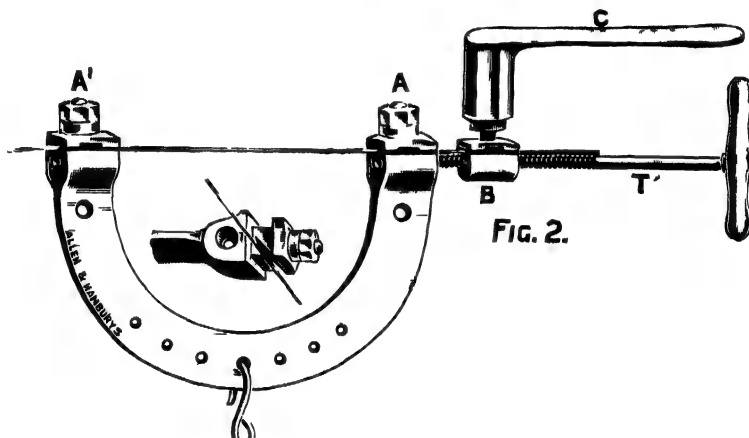
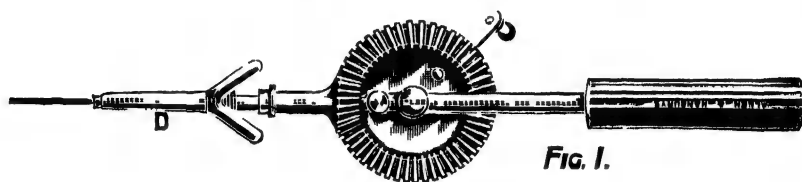


FIG 450

Apparatus for the insertion of Kirschner's wire. (Allen & Hanburys)

In general, it may be said that pins or wires passed through the bone are most satisfactory, since they cannot slip, and they allow not only traction but correction of angulation and rotation; and that, in most cases, traction through a distal bone is the safest method, since it avoids the risk of infection in the neighbouring joint.

Pins for traction are made of plated or rustless steel, sufficiently stout to be rigid, and are pointed at one end and square at the other (Fig. 449). They are driven through the bone, either by taps with a hammer, or by screwing them in with a handle slipped over the squared end. A caliper, with cupped jaws to fit over the projecting points, is used for traction.

Kirschner has introduced the use of steel piano wire, 19 gauge for the upper limb and 17 for the lower limb, in place of pins (Fig. 450). The small puncture is almost painless and reduces the risk of infection.

On the other hand, wire needs more elaborate apparatus for its introduction, and must be held under tension in special calipers to make it sufficiently rigid. Except where traction must be made on the lower end of the femur or the olecranon, the pin is equally good.

*Comparison of Surface and Skeletal Traction Methods.*—Surface traction is easily applied, simple and safe. It is very effective where a sufficient area of skin is available, that is, in fractures near the trunk. On the other hand, the apparatus is liable to slip and produce sores on the skin. It is of little use when the area of skin available is small, either because the fracture is a distal one or because the skin is already lacerated, and when it is necessary to use powerful extension, as in old-standing fractures.

Skeletal traction acts directly on the bone, and a given force applied



FIG. 451

Pearson's modification of the balkan beam, allowing suspension as well as extension.

(Allen & Hanburys.)

thus is as effective as twice the amount applied to the skin. It allows not only extension, but a considerable amount of direct control of the lower fragment. It can be used with equal success in distal fractures, and in the presence of lacerated wounds, provided that no septic place is near the point selected for traction. The method must be applied under conditions of strict asepsis, and is therefore unsuitable where such do not prevail. When these precautions are observed, and when traction is applied distal to a joint, the risks of infection are excessively small.

*The Use of Traction Methods in Fracture Treatment.*—Traction

upon any part of the body must be opposed by an equal force of counter-traction. This counter-traction is obtained from some fixed point on the patient's body, usually the axilla or chest wall in the upper limb, and the tuber ischii in the lower limb, against which the proximal padded end of the splint is pressed. Extension is then made either by fixed traction, that is by pulling on the limb to the required extent and tying the cord to the distal end of the splint, or by spring traction, the cord being attached to a spring or elastic band fixed to the splint. Alternatively, weight traction and counter-traction may be used. The extending force is a weight tied to the end of the extension cord, which passes over a pulley; the counter force is the weight of the patient's body, which is made effective by tilting the foot of the bed.

Fixed or spring traction with counter-traction against the body has the advantage that the patient can be moved with the apparatus in position. It is used for the transport of cases of fracture, and for the treatment of certain fractures of the upper limb where there is no great displacement. Weight traction necessitates treatment in

bed. It has an advantage over the other methods that the extending force is accurately known, and that it cannot vary during treatment, provided the pulley is at some distance from the splint and nearly frictionless. It also avoids the pressure of the splint upon the body, and is therefore preferable where powerful or prolonged extension is necessary.

When the patient is confined to bed, that is, in all cases treated by weight extension and in many where fixed extension is used, it is advisable to suspend the splint from a fracture frame (Fig. 451). This consists of an overhead bar joining two uprights which are attached to the bed frame, or stand on the floor. The proximal and distal ends of the splint are suspended from this bar by cords passing over pulleys, carrying weights which exactly counterbalance the mass of the splint and enclosed limb. With the injured part thus "floating" the patient can move about in bed or be raised for nursing without disturbing the fixation of his fracture.

#### TREATMENT OF FRACTURES DURING CONSOLIDATION

During the time that intervenes between union and consolidation, the callus that bridges the site of fracture may be rebroken by violence of moderate degree, or deformed by weight bearing or muscular action. Some form of protection is therefore necessary during this period. In the upper limb it is only necessary to protect the bone from accidental injury; in children this precaution is specially advisable. In the lower limb a walking caliper, which relieves the bone of the weight of the body but allows the use of muscles and joints, should be ordered in all cases of fracture of the long bones and worn for two to four months after union. Following abduction fractures at the ankle it is sufficient to divert the weight to the outer side by a leather wedge on the inner side of the sole of the boot. Fractures of the spine should be supported for from three to six months after the erect position is resumed by a special frame of steel and leather.

#### PRESERVATION OF THE FUNCTION OF MUSCLES AND JOINTS DURING TREATMENT

The ultimate restoration of function in a fractured limb can only be attained if the mechanical basis of movement is completely restored; that is, if the bone is united in correct length and alignment, the joints being in their proper relationship to the stresses transmitted along the limb, and the contour of the surfaces about which muscles play accurately reproduced. Function will, however, be restored more quickly if the muscles and joints are exercised during the period of union and consolidation. Where the claims of splinting and movement are contradictory, splinting must take precedence. But in deciding the method of treatment in any case, the use of muscles and joints must always be taken into consideration. When it is possible to move the limb without risk of disturbing the position of the fracture, supervised voluntary movements should be ordered after the third or fourth day. In a Colles' fracture, reduced and immobilised in plaster, the

patient should be encouraged to exercise and use the thumb and fingers, elbow and shoulder from the very beginning. When the limb must be immobilised, it is often possible to remove part of the splint, or one half of a sectioned plaster case, to allow access to the limb for physical treatment. Even when direct access is considered unwise, use of the part on its protective apparatus can be encouraged; thus, in a fracture of the tibia and fibula correctly reduced, walking may be allowed in a plaster case applied directly to the skin after the third week, and long before this, of course, on a walking iron. In fractures treated by traction, the apparatus should be so arranged that massage may be applied to the injured segment, and the joints moved; in this respect skeletal traction has manifest advantages over surface traction.

### OPERATIVE TREATMENT OF FRACTURES

Operation is employed in the treatment of fractures when other methods have failed or are clearly unsuitable. It should in any case only be undertaken by a surgeon conversant with the "no touch" orthopædic technique in all its details, and under conditions where these can be rigorously applied. With recent advances in fracture surgery, especially the introduction of local anæsthesia and of skeletal traction, the indications for operation have been considerably reduced. Operation may be required for reduction, for fixation or for both. Operative reduction is required when the shape of the bone-ends is such that they can be fitted together only by separation and exact dovetailing, where small fragments are widely displaced, and where soft parts intervene between the fragments and cannot be disengaged by manipulation. Operative fixation is necessary when the fracture cannot be kept in position after reduction, by position or pressure alone—in traction fractures, in some fractures near a joint where a small fragment is angulated by the pull of a powerful muscle, and occasionally in oblique fractures where skeletal traction has failed to give satisfactory reposition. When the fragments tend to separate, wire, silk, or kangaroo tendon are used for fixation; when they tend to angulate, plates of metal, beef-bone or living bone, fixed by metal or bone screws, are employed.

### TREATMENT OF DELAYED UNION, NON-UNION AND MALUNION

**Delayed Union.**—A search should first be made for the cause of the delay, whether this lies in the general health of the patient, or in some local error such as malposition of the fragments, sepsis in a compound fracture, or insufficient immobilisation. Any fault discovered should be corrected, and it should be remembered that this lies more often in the treatment than in the condition of the patient.

Attention should be paid to improving the general health of the patient, and especially to providing in the food those factors that are essential to bone repair. The diet should include liberal amounts of fresh vegetables and animal fats. Calcium in the form of calcium

gluconate, and preparations such as irradiated ergosterol, which contain vitamin D, should also be administered. Fresh air and sunlight stimulate all reparative processes.

The most important local treatment is encouragement of function in the fractured segment, while retaining the reduced position of the fragments. In the lower limb, walking in a weight-relieving caliper should be prescribed; in the upper, use of the limb in a close-fitting plaster case or leather support.

If in spite of long-continued correct treatment there is still inadequate union, ten to forty holes, according to the size of the bone, may be drilled in the indolent repair tissue and the adjacent bone-ends through two or three skin punctures. The fracture is then again immobilised for a further prolonged period, while exercises to preserve the function of the limb without disturbing the fragments are perseveringly carried out.

**Non-union.**—If the gap between the fragments is moderate, and there is no pseudarthrosis, the above methods should be given a trial. If they fail, and in any case where a wide gap or a false joint separates the bone-ends, union can only be obtained by operation. In some situations, such as the upper end of the radius or the lower end of the ulna, the disability caused by non-union may be insufficient to justify operative interference. An operation for non-union demands the exposure of the bone-ends, the removal of all scar tissue and sclerosed and avascular callus till healthy bleeding bone is exposed, and the placing of healthy bone surfaces in firm contact with each other. Where shortening of the limb does not produce great disability, as in the case of fractures of the humerus, the freshened ends of the bone may be wired or screwed together. In other cases the gap left after freshening the ends must be bridged by an autogenous bone graft, which is usually cut from the subcutaneous surface of the tibia.

**Malunion.**—The treatment of malunion varies with the nature of the deformity, the stage of repair and the age of the patient. In the old the risks of non-union after correction, and the less urgent need for perfect position, should be taken into consideration before active measures are adopted.

Deformities of angulation may be corrected during the period of repair by bending the callus, either forcibly under anæsthesia, or gradually by bands attached to the side bars of a traction splint or by a series of plaster cases. After union, and during the period of consolidation, the bone must be refractured and splinted as for a recent fracture in correct position. At a still later period, alignment should be corrected by dividing the bone with an osteotome at a point immediately above or below the fracture, and fixing the limb in correct position.

Deformities of shortening, lateral displacement, or rotation can only be satisfactorily corrected by operation. The site of fracture must be exposed, callus chiselled away, the original fracture planes exposed, and the ends brought into correct relationship and the position maintained by the appropriate form of splinting or skeletal traction.

Even small degrees of malposition are of serious import in fractures near or involving joints, where the contour of the joint surfaces and their alignment in relation to the transmission of force along the limb must be correct if function is to be preserved. In recent cases showing malunion, the fracture should be reconstructed and the fragments replaced by open operation. After repair has taken place in the deformed position a new articulating surface will have been formed by repair tissue; if the movement in the joint is free and painless, but the alignment unsatisfactory, the latter should be corrected by osteotomy; if the new joint is painful, arthrodesis provides the only remedy.

### TREATMENT OF COMPOUND FRACTURES

The essential difference between a compound and a simple fracture is that in the former micro-organisms have access to the site of injury, which in the latter is sterile. In indirect compound fractures the wound is usually clean-cut and little contaminated. In direct compound fractures the soft tissues are lacerated, often to a severe degree, and pieces of cloth, earth, grease, and other foreign matter are carried into the depths of the wound; the bone is usually contaminated, and fragments may be extruded or even lie loose in the clothing. In road accidents the soiling may be extreme, and anaerobic organisms such as the bacilli of gas gangrene and tetanus may be present.

Bone, being unable to undergo the ordinary changes of inflammation, has little resistance to bacterial invasion. If infection becomes established in a compound fracture, loose fragments die, and the ends of the shaft and the larger fragments undergo a septic osteitis, leading to the death and subsequent separation as sequestra of portions of their substance. Since the element of tension in a closed space does not exist, a true osteomyelitis, such as occurs in an uninjured bone due to infection by the blood stream, is uncommon, but may arise, leading to wide necrosis of the bone, metastatic abscesses, or death from pyæmia. Apart from its effects upon the injured bone, sepsis in a compound fracture leads to the formation of granulation tissue in place of callus, so that non-union is common and delayed union the rule, while the new bone that is thrown out is weak and porous.

The most important point in the treatment of a compound fracture is therefore the prevention or elimination of sepsis. Immediately after injury the micro-organisms in the wound are in the foreign matter or upon the surface of the tissues, and the wound is said to be contaminated; after twenty-four hours they have multiplied and established their hold, and the wound is infected.

In the case of an indirect compound fracture, with a small puncture wound, it is sufficient to shave and cleanse the skin round the wound, apply an antiseptic dressing, correct the position of the fracture, and immobilise the limb on a splint. The dressing should be changed twice daily till it is clear that no infection has arisen. If a spike of bone is protruding from the wound it should be carefully cleaned before it is reduced by manipulation.

In direct compound fractures operation is always essential. The



technique is that for all wounds as laid down in Chap. VII, p. 124. All loose pieces of bone are removed and soiled bone surfaces carefully trimmed with sharp bone-cutting forceps. The fracture is reduced and the limb immobilised in plaster.

The recent advances made in sulphonamide therapy have made it possible for much earlier operative interference in fractures which show signs of delayed union. Here again, very careful surgical technique is essential, and bone grafting or plating can be performed with much less risk of stirring up latent infection.

## INJURIES OF JOINTS

Joints, like bones, may be injured by *direct violence* applied to the joint itself, by *indirect violence*—strains of rotation, lateral bending, or compression applied to the limb as a whole, or by *muscular violence*. Direct violence usually gives rise to penetrating wounds or contusions, indirect and muscular violence to sprains, internal derangements, and dislocations.

**Penetrating Wounds** of joints are usually caused by sharp objects—knives, nails, cutting tools or broken glass. They may also be caused by motor accidents or bullets, and in these cases are usually accompanied by injury to the bones forming the joint. Penetrating wounds commonly involve the larger and more exposed joints, the knee more often than any other. The joint is swollen, and glairy synovial fluid may be recognised in the discharge from the wound. Their chief importance is that they present a path by which infection may enter the joint.

**Contusions** are the result of a blow over the joint. The overlying structures, capsule, and synovial membrane, are bruised or torn in varying degrees. Synovial fluid, usually mixed with blood, is poured out, distending the joint cavity. Local bruising and swelling are seen at the point of injury, which is also tender on pressure and painful during movements which stretch the damaged structures. Another type of contusion is caused by strains of compression, which jar the opposed articular surfaces against each other. Such injuries are often accompanied by sprains of ligaments, but the chief damage is sustained by the articular cartilage, which becomes swollen and opaque at the points of impact. There is effusion into the joint, but no evidence of injury on the surface.

**Sprains** are caused by indirect violence, which forces the joint in some direction beyond its normal limits. The capsule is stretched or torn, and ligaments are ruptured, partly or completely, or in some cases detached at their insertions with a superficial flake of bone. While the damage falls chiefly on the fibrous structures, the synovial membrane is usually lacerated to some extent.

The joint becomes distended with fluid, and signs of local injury, swelling and bruising, appear over the damaged ligaments. Tenderness on pressure and pain on movement are accurately localised to the point of injury. Voluntary movements of the joint are impaired, and,

when important ligaments have been torn across, abnormal mobility may be found.

**Internal Derangements**, widely interpreted, include the tearing of intra-articular ligaments. The term is usually limited to the displacement of some intra-articular structure between the opposing surfaces of the bones forming the joint, causing mechanical locking. Since the only structures which can be so displaced in a normal joint are the intra-articular menisci and folds of synovial membrane, these injuries are limited to the knee, sterno-clavicular and temporo-mandibular joints.

During some extreme active or passive movement, a sudden pain, often accompanied by the sensation of a "click," is felt in the joint. The joint is found to be swollen, and its movements in some directions are arrested by a firm but resilient block. By some manipulation the impacted body can usually be dislodged, often with a recognisable snap, restoring immediately the full range of movement.

**Loose Bodies** which develop in a joint may be caused by pieces of bone or cartilage which have been separated as a result of trauma, or they may have been extruded into the joint as a result of the condition known as osteochondritis dessicans. This condition, which occurs most commonly in the knee joint, and more particularly on the inter-condylar aspect of the internal condyle, is due to an avascular necrosis; some cases may be associated with trauma, but this is not a necessary factor of the condition. Osteochondritis dessicans may affect several joints in the same patient; for instance, both elbows or both knees, and in some cases both the elbows and the knees have been involved. The presence of a loose body in the knee joint gives rise to locking, and the patient may even be aware of its presence and may indicate its position. X-ray examination is essential in determining the exact diagnosis and the appropriate treatment.

**Dislocations.**—A dislocation is a complete disjunction of the articulating surfaces forming a joint. Usually the capsule is torn, and the articular end of one of the bones has left the capsule through the rent and lies among the surrounding tissues. Dislocations are as a rule caused by indirect violence, and are more common in those joints whose security depends rather on muscular support than on the shape of bones or the strength of ligaments. They are seen in the middle ages of life; in childhood a similar injury will cause separation of an epiphysis, and in old age a juxta-articular fracture.

A dislocation produces notable deformity of the joint itself, usually a flattening where one bone is absent from its normal situation, and a prominence where it lies in an abnormal one, and of the whole region, due to the part distal to the joint being out of its normal alignment. In addition there is swelling, pain, loss of function and limitation of movement in directions that are specific for each dislocation.

**SUBLUXATION** or partial dislocation may also occur. In this the articular surfaces have lost their normal relationship, but are still in contact. Stretching or tearing of ligaments is necessary before subluxation can occur, but neither articular surface is extruded from the capsule.

**RECURRENT DISLOCATION.**—When the mechanism on which the security of a joint depends is imperfectly reconstituted after reduction, dislocation is liable to recur with only moderate violence. Recurrent dislocations of this type are only common in the shoulder and patella, and will be discussed with these regions.

**PATHOLOGICAL DISLOCATION.**—A joint may become dislocated without pain, or indeed any symptoms, when the articular surfaces are completely or partly eroded, and the capsule and ligaments softened or destroyed by disease. These dislocations are primarily due to disease of the joint, the factor of trauma being slight or absent; they cannot therefore be discussed appropriately in the present context.

**The Repair of Joint Injuries.**—The capsule and ligaments consist of bundles of fibrous tissue containing a few elastic fibres. When they are torn, the space is filled with blood clot, which is replaced, first by granulation tissue, later by connective tissue. If this connective tissue bridge is short and firm the injured part will regain its former strength, but if the gap between the ends is wide, or if the repair tissue is stretched during organisation by movement, faulty position or distension of the joint with fluid, permanent weakness will result.

The synovial membrane is richly supplied with blood vessels. These dilate in response to injury, so that the membrane becomes swollen and hyperæmic, and a large amount of synovial fluid, richer in fibrin than the normal secretion and containing blood from the torn vessels, is poured into the joint. The damage to the synovia is made good by the ordinary process of repair; if it is moderate, the membrane will regain its normal appearance and structure; if considerable, an excess of fibrous tissue will be formed leading to adhesions limiting movement, or in the case of free synovial folds, to permanent thickening which may cause internal derangement of the joint. Synovial fluid is absorbed by the subsynovial lymphatics, and this absorption is aided by muscular movements and intracapsular tension; it is hindered by complete immobility and by wasting of muscles surrounding the joint.

The hyaline cartilage covering articular surfaces has no recognisable blood supply except at its periphery, and possesses no power of repair by its own tissue. When bruised the cartilage first swells and becomes opaque, while later its surface layers are disintegrated and cast off. Cartilage so thinned becomes worn away by subsequent use of the joint, leading to osteo-arthritis. Cuts in hyaline cartilage become partly filled in by ordinary fibrous tissue. A severe blow on an exposed surface may lead to the death of the injured portion of cartilage, which later becomes separated from the surrounding healthy portions by aseptic necrosis, and is finally extruded into the joint as a loose body.

The interarticular menisci consist of fibro-cartilage covered on their free surfaces by synovial membrane. Tears are repaired by connective tissue. If a torn meniscus is replaced early and not disturbed during the process of healing, a close approximation to the normal may be reproduced; if, however, it is not replaced, or is injured again before repair is complete, it will become loose or deformed, and liable to be jammed between the articulating surfaces during movement.

## PRINCIPLES OF TREATMENT IN JOINT INJURIES

**Penetrating Wounds.**—A penetrating wound of a joint presents the problem of infection or potential infection. The synovial fluid which is poured out in response to any injury has the active bactericidal power of any inflammatory exudate, and can deal with an invasion by organisms small in number or of low virulence. When this mechanism fails, the infection becomes one in a closed space. The fluid then serves as a culture medium and becomes purulent, and, till evacuated, prevents the secretion of fresh active fluid; the synovial membrane is transformed into granulation tissue. A joint so infected is almost necessarily destroyed as a joint; further, toxæmia, pyæmia and death may follow.

The *treatment* of a penetrating joint wound is therefore similar to that of a compound fracture—excision of the track, and of all soiled and lacerated tissues. If penetration of the synovial membrane is doubtful, no probing or instrumental exploration, which may enter an unopened cavity and carry infection from the surface to the deeper parts, should be undertaken. The wound in the skin and capsule is first trimmed and the synovial layer inspected. If an opening is present, which will be indicated by the escape of fluid, its edges should be excised. The joint cavity may then be inspected, any blood clot or foreign matter washed out, and the interior well insufflated with sulphanilamide powder. The synovial membrane and capsule are closed with interrupted catgut sutures, the skin wound is partly closed, and a rubber or gauze drain led down to the capsule. A full intensive course of the appropriate sulphonamide drug should be administered by the mouth. The limb is immobilised in a splint or plaster till it is evident that infection has not appeared in the joint.

**Non-penetrating Injuries.**—Two conflicting claims must be reconciled. On the one hand, torn ligaments and intra-articular cartilages require complete relief from strain over a long period. On the other hand, function is necessary for the preservation of movement, the avoidance of adhesions, the prevention of muscle wasting, and the absorption of fluid, which, if allowed to remain, will stretch the torn and even the intact ligaments, and injure the periarticular muscles.

In all cases complete rest and pressure with a firm bandage is advisable for the first forty-eight hours after injury, in order to arrest the effusion and hæmorrhage from torn structures. In the case of contusions and minor sprains, the use of the joint should be encouraged after this, only such movements as stretch the injured parts being discouraged or prevented by apparatus. Firm pressure should be applied by strapping or a crêpe bandage while swelling persists, and massage may be given.

When important ligaments have been torn, more complete immobilisation is necessary in the early stages, and more positive measures to relieve strain afterwards, which should be continued in the case of the joints of the lower limb for from four to six months. The first demands splints or a plaster case, the position for fixation depending upon the ligament which is injured, and the most useful position for

the joint should ankylosis or limitation of movement result. Function can often be encouraged even though the joint is completely immobilised, thus in the case of dislocation of the knee with rupture of all the important ligaments, the whole limb must be immobilised in a plaster case, but holes may be cut for daily faradic stimulation of the quadriceps and walking should be encouraged. Later, a joint may be used more freely, but relieved of strain in certain directions; thus after rupture of the internal lateral ligament of the knee, a jointed steel and leather case may be worn which will allow anteroposterior but prevent lateral or rotary movement, and in slighter injuries the heel of the boot may be raised on the inner side to prevent abduction. Internal derangement should be corrected at once by suitable manipulation; thereafter the treatment follows the same lines as that for torn ligaments—maintenance of function combined with relief of strain.

Dislocation should be reduced immediately. Early movement is very desirable to prevent the formation of adhesions, but the extent and direction of this movement will be governed by the joint involved and by the extent of damage to the ligaments and adjacent structures.

A. E. PORRITT.

R. Y. PATON.

## CHAPTER XLV

### INJURIES OF THE UPPER LIMB

#### THE CLAVICLE

**F**RACTURES of the *shaft* of the clavicle are usually caused by indirect violence, *e.g.*, blows or falls on the point of the shoulder, which compress the bone in its long axis. The bone breaks about the middle, or at the junction of the middle and outer thirds; the line of fracture is either transverse or oblique downwards and inwards, often with some comminution (Fig. 452). The displacement is constant, the outer fragment is carried downwards, inwards and forwards by the weight of the shoulder and the pull of the pectoral muscles, and overlaps the inner, which retains its level or is slightly raised by the sternomastoid attachment, by about half an inch. The classical signs of fracture—loss of function in the arm and pain, swelling,



FIG. 452

Diagram illustrating the two common positions of fracture of the clavicle.

tenderness and crepitus at the site of fracture—are present. The appearance of the patient, with swelling due to the projecting inner fragment at the base of the posterior triangle, and the shoulder on the injured side at a lower level, or the elbow held up by the

opposite hand, the head being inclined to the same side, is usually unmistakable.

The reduction and fixation of fractures of the clavicle present certain difficulties. Reduction can be effected by the standard method of bringing the distal fragment into line with the proximal one, that is, by pulling the shoulder, to which the outer end is attached, outwards, backwards and upwards. General anaesthesia, whilst facilitating reduction, makes the satisfactory application of retentive apparatus almost impossible. Local anaesthesia should therefore be used for any early case. By strong traction on the shoulder, followed by downward pressure on the inner fragment, it is often possible in the case of a transverse, or nearly transverse, fracture to obtain complete and stable reduction; it is then sufficient to apply two or three bands of strapping across the shoulder from front to back, crossing over the fracture, and place the arm in a sling.

When reduction is incomplete, or if complete is unstable, the shoulder must be held in the corrected position until union has commenced. The methods of immobilisation of the clavicle should permit use of the shoulder and arm. The figure-of-eight bandage controls the fracture and permits early movements, but must be carefully

supervised and reapplied every two or three days. The patient sits on a low stool and the shoulder is braced well backwards and upwards. A large pad of wool is placed in each axilla, extending well up in front. The bandage, which must be at least 4 in. wide, is passed over the shoulder, under the axilla and crossing over to the other shoulder, which is similarly bandaged (Fig. 453). Where the turns of bandage cross between the shoulder blades a few stitches can be put in to hold them together. The arm is supported in a sling and early movements are started.

In the "Newmarket" method the patient sits on a low stool and the arm on the injured side is abducted to a right angle, externally

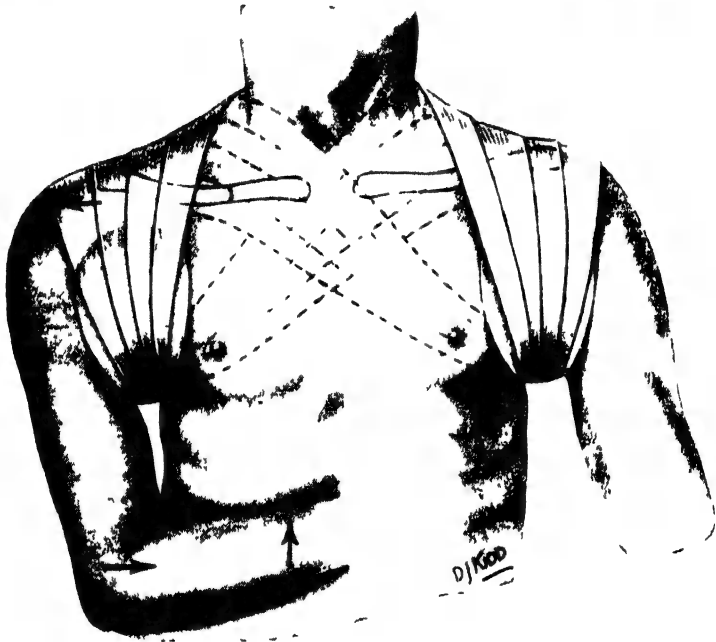


FIG. 453

The bandage pulls the outer fragment backwards and upwards, and the weight of the arm over the axillary pad maintains distraction from the midline. (Watson-Jones.)

rotated and extended strongly backwards. A wide roll of gamgee is passed under the axilla and over the point of the shoulder, and an adhesive felt pad is placed over the inner fragment. A length of 3-in. wide adhesive strapping is then passed from the umbilical level vertically upwards over the inner fragment and straight down the back to a corresponding level—the pull firmly holding down the fragment. The second strap is passed a short distance further in, overlapping the first. The third piece of strapping starts nearer the umbilicus, crossing the others obliquely towards the point of the shoulder round into and through the axilla, protected by the gamgee and then up over the point of the shoulder again and down obliquely across the back. A fourth piece of strapping is run round the body at the umbilical level to fix the ends of the other pieces. The arm is then dropped into a sling, and the patient is able to start using the limb immediately.

In girls where a noticeable deformity must be avoided it is often advisable to treat by complete rest in bed. No pillow is allowed and a vertical sandbag is placed between the shoulder blades. In some cases extension in the abducted position may be necessary to overcome overlap and deformity.

The retentive apparatus should be kept in position for three weeks. Fractures of the clavicle unite rapidly and the result as regards function is nearly always good, even if some deformity persists. Callus, which may form an unsightly swelling after union, rarely remains visible after a year.

The shaft is sometimes injured by direct violence, in which case the subclavian vessels or brachial plexus may be damaged by displaced fragments. Operation is necessary in such cases.

Fractures of the *outer end* are caused by direct violence (Fig. 452).

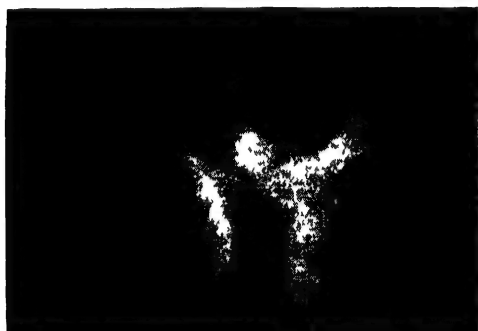


FIG. 454

An X-ray showing a dislocation of the acromio-clavicular joint.

The ligaments which bind this portion to the coracoid and acromion processes of the scapula usually prevent displacement, unless the line of fracture passes outside the trapezoid ligament, in which case the displacement is that of an acromio-clavicular dislocation. A sling till pain and bruising have disappeared is sufficient treatment. Fractures of the *inner end* present similar features. They are due to direct violence, and if the rhomboid ligament is uninjured they are

accompanied by little displacement, while if it is torn the injury resembles a dislocation.

**Dislocations of the Clavicle—A. THE OUTER END.**—Dislocations of the acromio-clavicular joint are caused by falls on the point of the shoulder, forcing the acromion process downwards. In most cases the acromio-clavicular ligaments only are torn and the outer end of the clavicle rides upwards, but as it is not entirely displaced from contact the condition is really one of subluxation. If the coraco-clavicular ligaments are torn in addition, complete dislocation with wide displacement will follow (Fig. 454).

The joint is swollen and painful, while movements, especially of abduction, are restricted. The deformity is obvious, the end of the clavicle forming a visible prominence which can readily be pushed downwards into its normal position. Reduction is therefore a matter of simplicity, but retention is more difficult. A pad of wool is placed in the axilla and the wrist is supported by the collar-and-cuff method with the forearm horizontal. A thick pad of adhesive felt is placed over the outer half of the clavicle and another pad is placed below the elbow over the olecranon and epicondyles. With the humerus pushed fully up, broad pieces of strong inelastic strapping are passed round the pads, pulling them as tightly together as possible



(Fig. 455). Several strips are required and will have to be reinforced by others frequently, as the weight of the limb stretches the strapping. In subluxations the position must be maintained for at least three weeks ; in dislocations, at least six weeks. It is usual for some displacement to persist in spite of the most careful treatment, but there is seldom any permanent disability. A large number of jockeys and acrobats are carrying on their occupation with old unreduced dislocations.

Dislocations, other than the superior one described, are very rare.

**B. THE INNER END.**—The clavicle may be displaced forwards, backwards or upwards in relation to the manubrium, the first being the most common. Forward and upward dislocations are caused by indirect violence, the shoulder being forced downwards and backwards and the inner end of the clavicle levered out of position, the first rib acting as the fulcrum. The rhomboid ligament is usually intact in forward dislocations but torn in upward ones. Backward dislocations are due to direct violence on the inner end of the clavicle.

The displacement is obvious, the head of the bone forming a visible prominence in the forward and upward varieties, and its absence being equally noticeable in the backward type. Pain and inability to use the arm are the main complaints, but a backward dislocation may cause urgent symptoms by pressure on the trachea, cesophagus or subclavian vessels. Reduction is effected by pulling the shoulder outwards and backwards and pressing on the head of the bone. The shoulders are then held back by the figure-of-eight bandage with large axillary pads as for a fractured clavicle, and a pad is fixed over the joint with strapping. Such methods fail to maintain complete reduction, but the resulting disability is slight.



FIG. 455

Correct method of strapping an acromioclavicular dislocation. (Watson-Jones)

## THE SCAPULA

**The Body** of the scapula, lying upon the resilient thorax and covered by muscles, is well protected and is fractured only by direct violence of considerable degree. Fractures take the form of irregular fissures, involving chiefly the infraspinous fossa, but often extending across the scapular spine ; there is seldom any gross displacement of the fragments. Pain, increased by movement, swelling of the whole scapular region, bruising and crepitus are present. If the spinous process is broken, the line of fracture can be felt and the fragments may be moved independently.

The scapula is immobilised by bands of strapping round the chest and over the shoulder, crossing over the affected bone. The arm is

placed in a sling. Massage may be started on the fourth day and movements on the tenth.

**The Neck** is fractured by blows or falls on the shoulder. The fracture passes from the scapular notch to the axillary border, separating the glenoid and coracoid processes with the capsule of the shoulder joint from the body. If the coraco-acromial and coracoclavicular ligaments remain intact, there is little displacement, the fracture is suspected only owing to pain and loss of function, and its presence is definitely established by X-rays. If these ligaments are torn the outer fragment is displaced downwards by the weight of the arm, causing flattening of the shoulder and prominence of the acromion. This appearance at first sight suggests dislocation of the shoulder, but its clinical signs are absent, while the deformity is reduced merely by supporting the elbow and recurs when this support is relaxed; further, the coracoid can be felt to move with the shoulder.

When there is no displacement the arm is placed in a sling and massage and movements started early. In patients under 45, where displacement has occurred, weight traction with the shoulder abducted to a right angle should be applied for at least four weeks.

**The Glenoid** may be fractured in dislocation of the shoulder, a small chip being detached from the lower or anterior margin. It should be suspected when the reduction of a dislocated shoulder cannot be maintained, and is a common cause of recurrent dislocation. It can be established only by X-rays.

**The Coracoid** may be broken by the recoil of a gun, by the head of the humerus or by muscular action. In the uncommon event of displacement the arm should be strapped across the chest in a position of flexion, to relax the pull of the muscles attached to the process.

**The Acromion** is broken by direct violence applied to the shoulder. There is usually little displacement, but voluntary abduction causes pain. The arm should be fixed on an abduction splint for four weeks.

### INJURIES TO THE SHOULDER JOINT

**Dislocations of the Shoulder.**—The shoulder has a wider range of movement and depends less for its security upon the factors of mechanical coaptation and ligamentous protection than any other joint. It is thus more often dislocated. The great majority of these injuries are due to indirect violence such as falls on the outstretched hand or arm, which force the humerus into hyperabduction till the neck is thrust against the acromion. Further abduction forces the head of the bone through the lowest and weakest part of the capsule, so that it comes to lie below the glenoid rim on the long head of the triceps. Rarely the arm may remain in this hyperabducted position with the head below the glenoid, the “*luxatio erecta*”; but usually it falls downwards (subglenoid dislocation) or, the coraco-humeral ligaments becoming tense, the head of the humerus is pulled upwards and forwards deep to the subscapularis to lie under the coracoid (subcoracoid dislocation) (Fig. 456) or still further forwards under the clavicle on the inner side of the coracoid (subclavicular dislocation).

Less commonly it passes backwards to lie beneath the spine of the scapula (subspinous dislocation). The head of the humerus can lie in the subglenoid position with the arm at the side only if the coracohumeral ligaments have been torn. The five types of dislocation occur in this order of frequency: (1) subcoracoid, (2) subclavicular, (3) subspinous, (4) subglenoid and (5) luxatio erecta. All but the first variety are rare.

A patient with a dislocated shoulder complains of pain, inability to use the arm and sometimes numbness in the fingers. On removing the clothing an obvious deformity is seen; the shoulder is flattened and a sharp angle at the edge of the acromion replaces the usual rounded contour. An abnormal prominence appears where the head of the humerus lies under the muscles, the elbow is held away from the side of the chest, and the line of the upper arm is seen to lead not to the position of the



FIG. 456

Subcoracoid dislocation of the shoulder. Note the avulsion of the greater tuberosity.



FIG. 457

Subcoracoid dislocation of the shoulder. Note the flattening of the contour of the shoulder and the projection of the elbow from the side. (Watson-Jones.)

glenoid but further inwards (Fig. 457). Several tests may be used to confirm the diagnosis. A ruler laid along the arm can be made to touch the external epicondyle and the acromion. The measurement between these two points is less than on the sound side, while that taken round the axilla from a point over the upper surface of the acromion is increased. The anterior fold of the axilla is lower than the posterior. The head of the humerus can be felt in its displaced position; the elbow cannot be brought against the chest-wall. Careful examination must be made for nerve involvement as in one in seven dislocations there is paralysis as a result of traction injury of the branches of the brachial plexus. In addition, before reduction is attempted an X-ray examination must be made, if possible, to

exclude any complicating fracture.

*Treatment.*—**Kocher's Method** of reduction is simple and usually succeeds in uncomplicated subcoracoid dislocations. It depends upon the stretching of the subscapularis muscle, spasm of which is the chief obstacle to replacement. There are three steps. Firstly, with the arm to the side and the elbow at right angles, the forearm is used as



FIG. 458



FIG. 459



FIG. 460



FIG. 461

Reduction of dislocation of the shoulder. Traction is applied. The limb is gently and slowly externally rotated. The elbow is brought forward in front of the trunk and the shoulder is then internally rotated. (Watson-Jones.)

a lever by which the arm is slowly and steadily rotated outwards, until after several minutes a position of full external rotation is reached. Secondly, the elbow is slowly brought forwards and upwards across the chest towards the opposite shoulder, the arm still being fully everted. Thirdly, the forearm is smartly rotated inwards, bringing the hand on to the opposite shoulder. Reduction may occur at any part of this third stage (Figs. 458 to 461).

When Kocher's method has failed reduction by extension should be performed. The patient lies on a couch. While an assistant makes counter-traction by means of a roller-towel round the chest, the surgeon abducts the arm to a right angle and pulls steadily in its long axis until the muscles are felt to relax and the head of the humerus comes to lie opposite the glenoid. One hand is then placed in the axilla to guide the head of the bone while the other, still making traction, rotates the arm first inwards and then outwards till reduction takes place. When no assistant is available, counter-traction may be made by the unshod foot against the chest-wall, but the foot should never be placed in the axilla.

While early and uncomplicated dislocations may often be reduced without an anæsthetic, general anæsthesia should usually be employed; not only is this kinder, but the muscular relaxation so produced removes the chief obstacle to reduction.

*After-treatment.*—The arm is supported by the collar-and-cuff method with a good pad of wool in the axilla and the limb is bandaged to the trunk for a fortnight. A sling only is worn for a further week, but active movements of the elbow and shoulder are started. Passive movement must on no account be used.

*Complications.*—Fracture of the upper end of the humerus or the scapula may accompany dislocation of the shoulder. The only common fracture in this connection is that of the greater tuberosity of the humerus, which is torn off during the dislocation and remains attached to the capsule (Fig. 456). Fractures of the anatomical neck and partial fractures of the head are seen in the aged and of the surgical neck in adults, while the glenoid rim or coracoid may also be broken. A fracture may be suspected when any unusual difficulty is encountered in reduction, and is certain when in addition crepitus is felt during manipulation. Confirmation and recognition of the exact injury depend on X-rays. Extension should be used to reduce a dislocation complicated by fracture. It will succeed in those involving the greater tuberosity and the intracapsular portions of the head. When it fails, reposition by operation should be employed immediately.

Injury to nerves in the axilla is an infrequent but serious complication of shoulder dislocations. The commonest lesion is of the circumflex nerve alone, but the posterior or inner cord or even the whole plexus may be damaged. The nerves are in most cases bruised or stretched only, and partial or complete recovery may be expected if the paralysed muscles are treated by relaxation. Operative repair is unpractical.

**UNREDUCED DISLOCATION.**—A dislocation of the shoulder can usually be reduced by manipulation, often with difficulty, up to six weeks from the time of injury, and reduction should therefore be attempted up to this time. After eight weeks the head of the humerus is so fixed in its new position, and the capsule, muscles and even the nerves and vessels have been so altered by the processes of repair, that the attempt is dangerous as well as useless. Three alternative procedures can then be considered; open reduction, excision of the head of the humerus or physiotherapy to improve function in the false joint which has

formed. The method selected will depend upon the interval that has elapsed since injury, the age of the patient and the amount of disability that is present. It must be emphasised that an unreduced dislocation is often compatible with surprisingly good function, and equally that late operation can hardly be expected to produce a first-rate result.

**RECURRENT DISLOCATIONS** are usually seen in either healthy young athletes, workmen or epileptics. After the first injury dislocation is reproduced by very slight violence and even by muscular action, thus

debarring the patient from any strenuous activity. These recurrent dislocations are often the result of direct violence, such as a fall on the back of the shoulder. The capsule is torn from its attachments to the rim of the glenoid, the margin of which may be fractured. These injuries can be treated only by operation. Many methods are in use; of these that advocated by Nicola is the simplest and is effective in most cases. The tendon of the long head of biceps is exposed and divided 1 in. below the bicipital groove after stay sutures have been inserted above and below this level. A narrow tunnel is drilled from the lower end of the bicipital groove up to the articular surface of the humerus emerging into the joint at least  $\frac{1}{2}$  in. from the articular margin. The proximal portion of the tendon is guided down through this tunnel and resutured to the distal portion taut, and to the sides of the bicipital groove, while the humerus is in the position of right-angle abduction and internal rotation. Alternatively, the method advocated by Bankart should be used. It consists of repair of the anterior and inferior part of the capsule with reconstitution of the inferior lip of the glenoid, to which the repaired capsule is firmly sutured with salmon gut.

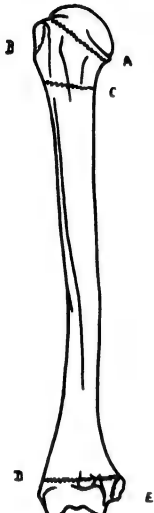


FIG. 462

Diagram illustrating the common sites for fracture of the upper and lower ends of the humerus.

A, fracture of the anatomical neck; B, fracture of the greater tuberosity; C, fracture of the surgical neck; D, supracondylar fracture; E, fracture of the inner epicondyle.

**CONTUSIONS OF THE SHOULDER JOINT.**—Falls on the point of the shoulder often lead to bruising of the articular cartilage of the head of the humerus and the glenoid. Pain, effusion and limitation of movement result and yield slowly to physiotherapy. Osteo-arthritic changes are prone to supervene later.

## THE HUMERUS

**The Upper End.**—**FRACTURES OF THE HEAD AND ANATOMICAL NECK** (Fig. 462, A) are rare except in the aged. They are usually caused by direct violence applied to the shoulder, but they may complicate dislocation. If the line of fracture is entirely intracapsular, the fragments, consisting of part or the whole of the head, are frequently detached and lie free in the joint.

There is little deformity, the outstanding symptoms being loss of function and swelling in the region of the joint. The absence of deformity distinguishes this fracture from a dislocation, and when the arm is examined crepitus is noticed in the region of the head. The exact nature of the injury can be determined only by X-ray.

Since such small fragments, if totally detached, can neither be replaced nor retained in position, they must be removed by operation. If their position is satisfactory the limb should be kept in a sling for a few days, massage being applied to the shoulder. At the end of a week moderate abduction may be obtained by a pad between the arm and the chest, and after three weeks active movements at the shoulder may be started.

FRACTURES OF THE SURGICAL NECK are frequently seen in adults (Fig. 462, c). The bone is broken either by direct violence or by falls

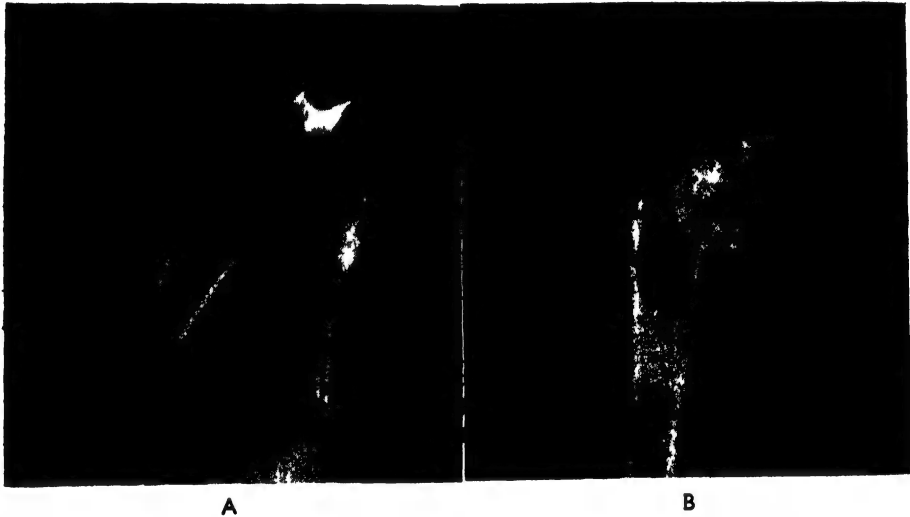


FIG. 463

Fracture of the surgical neck (abduction type): A, before; B, after reduction.

on the elbow or hand. The line of fracture is just above the insertion of pectoralis major and is usually transverse, but may be oblique or comminuted. In many cases there is little displacement, the fragments being retained by the tendinous fibres which surround the neck. Displacement, when it occurs, results either in the *adduction fracture* where the fracture line is high and there is angulation outwards at the fracture level with usually impaction on the inner side, or in the *abduction fracture* (Fig. 463) where there is angulation inwards at the fracture level, with or without impaction of the fragments on the outer side. Much bruising and swelling is present, and the patient cannot use the arm. In abduction fractures an external depression may be noticed at the fracture level and the elbow is held away from the chest-wall. If there is no impaction, it will be noticed that the head of the bone does not follow movement of the shaft, the upper end of which may be felt in the axilla. Shortening may be demonstrated by the tape-measure.

*Treatment.*—For this purpose fractures of the surgical neck must be divided into three classes. In the first group, which includes the majority of these fractures, displacement is minimal and impaction common. Hence no reduction is required and the arm is immobilised by bandaging it to the chest, a pad of wool having been placed in the axilla. At first an ordinary sling is also applied, but this should be discarded as frequently as possible after the first few days to allow active movements of the shoulder, elbow, forearm, wrist and hand. The bandage, which in well-impacted fractures can be omitted, is discarded after ten or twelve days and the arm is supported by a sling only. Active shoulder movements are started as soon as the patient can be persuaded to carry them out. No joint tends to become stiff more quickly or remain stiff longer than the shoulder; so the earlier the patient starts voluntary movements the sooner and fuller will be the recovery. Frequently repeated short periods of voluntary movements give much quicker and better results than prolonged periods twice daily.

In adduction fractures the treatment depends upon the age of the patient and the degree of angulation. In children the deformity must be connected by traction and abduction and the limb immobilised for three weeks in an abduction frame. In patients over 50 it is inadvisable to immobilise the limb for three or four weeks as the resulting stiffness will prove a grave obstacle to recovery of function. A sling with early active movements will ensure a good recovery. In other patients and where the angulation is marked, 30 degrees or over, traction and immobilisation in an abduction frame for three to four weeks is necessary.

In abduction fractures, impaction of the outer margin of the shaft and the outer part of the head with only slight angular deformity is the most common of all shoulder injuries. There is no need to attempt to reduce the deformity. A sling only is necessary with early movements of fingers, wrist and elbow, followed by active movement of the shoulder after ten days. In unimpacted fractures reduction of the displacement can be obtained by adducting the shaft across the chest and by a hand in the axilla pushing the upper end outwards until the fractured surfaces engage. The limb is then brought to the side and immobilised for three weeks by a sling, with axillary pad and bandage round the chest.

SEPARATION OF THE UPPER HUMERAL EPIPHYSIS is caused by accidents similar to those responsible for fracture of the surgical neck, and occasionally by forcible traction on the arm. The injury is usually a juxta-epiphyseal fracture, a triangular fragment of shaft being detached with the epiphysis. When separation is complete, the displacement of the fragments and the physical signs resemble those of fracture of the surgical neck, except that the crepitus is the soft grating of cartilage. In many cases the separation is partial and the injury can be recognised only by an X-ray.

*Treatment* follows the lines advocated for fracture of the surgical neck in adults. General anæsthesia is preferable to local for reduction, which must be complete owing to the disturbance of growth which follows malposition. It is sometimes impossible to obtain satisfactory reduction by manipulation and open operation is then necessary. After



replacement the arm is retained by a plaster spica in that position which keeps the fragments in alignment ; internal methods of fixation should be avoided.

**FRACTURES OF THE GREATER TUBEROSITY** (Fig. 462, B) may be due to direct violence or to avulsion. Avulsion fractures may occur as an isolated injury or may be a complication of dislocation of the shoulder. Where there is very slight or no displacement, a sling should be used for a fortnight and active movements must be started at once and practised regularly. No passive movements should be attempted.

Where the tuberosity is definitely separated, it is essential, in order to avoid grave disability, that the humerus should be abducted to a right angle and externally rotated at least 60 degrees so that the humerus is brought up to the detached fragment. This position is maintained in an abduction splint or plaster spica for six or eight weeks until union is sound. Active exercises are then started and persisted in for a long time, as full functional recovery is slow and takes six months or more. In dislocations the tuberosity is widely displaced, but returns to position when the dislocation has been reduced.

**The Shaft.**—Fractures of the shaft of the humerus are usually due to direct violence and may involve it at any level ; they are transverse (Fig. 464) or comminuted. Fractures due to indirect violence favour the junction of the middle and lower thirds and are usually oblique. The symptoms and signs are obvious and the diagnosis unmistakable. Those above the deltoid insertion tend to show an outward displacement of the lower fragment due to the pull of that muscle, and those below that level the reverse deformity, but the displacement depends in the main on the violence causing the injury.

*Treatment* varies with the type of displacement. Transverse fractures may show angulation without other deformity or there may be a considerable overlap. Oblique fractures have the most displacement owing to the fact that their sharp extremities become fixed in the triceps muscle. End-to-end transverse fractures require simple fixation by a light plaster. A long plaster slab is applied from just below the axilla, down the inner aspect of the arm, round the point of the elbow (flexed to a right angle) and up the outer aspect to the shoulder (Fig. 465). This plaster is fixed firmly by a bandage. A posterior slab from the shoulder down to the back of the wrist may be added. The forearm is supported by a sling ; while in

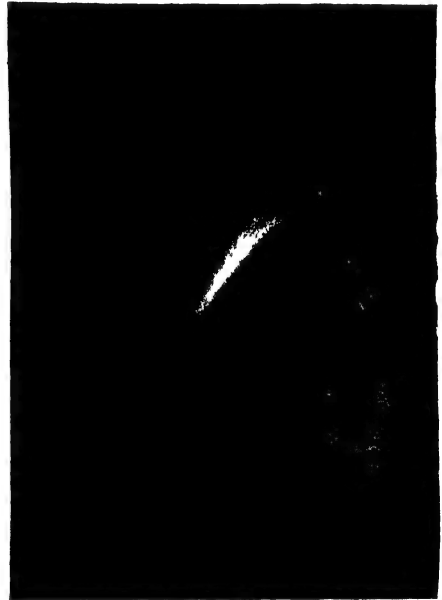


FIG. 464

Fracture through the middle of the shaft of the humerus.

some cases where the fracture is above the middle of the humerus the plastered limb must be supported in an abduction frame—60 degrees abduction and 30 degrees forward flexion being the optimum position. Transverse fractures heal more slowly than oblique fractures and may require prolonged immobilisation. Oblique fractures, if there is much displacement, should be manipulated into position and put in a plaster as described above. They usually heal very quickly. Traction is usually unnecessary as slight lateral displacements in no way prevent



FIG. 465

Plaster slab and sling for oblique fractures of the shaft of the humerus. Fractures with slow union require the immobilisation of a frame (inset). (Watson-Jones)

a good cosmetic and functional result. The oblique fractures are kept in plaster for five to six weeks, after which active exercises are started.

While the majority of these fractures unite readily, delayed union is more common than in any other bone, owing to the weight of the arm drawing the fragments apart and possibly to ill-judged extension. Prolonged immobilisation in a shoulder spica plaster down to and including the forearm should be tried for several months before bone grafting is attempted. The most serious *complication* is injury to one of the main nerves, usually the musculospiral where it lies in its groove at the back of the humerus. The nerve may be concussed, contused, torn across, or compressed by splints or callus. As the injury is usually

temporary, eight weeks should be allowed to elapse before exposing the nerve. If no recovery is apparent or if the signs are increasing, operation should be undertaken, when the nerve can be freed and sutured if necessary.

**The Lower End.**—Fractures of the lower end are most commonly seen in children below the age of 10 years and are caused by a fall on the outstretched hand or by violence applied directly to the elbow from a fall. The first type gives rise to a supracondylar fracture and the latter to intercondylar or T-shaped fractures, or to fractures of a part of the bone only.

**FRACTURES INVOLVING THE WHOLE LOWER END.**—A. *Supracondylar Fractures* (Fig. 462, D) are due to a force transmuted along the forearm, which pushes the lower end of the humerus backwards. The line of fracture is roughly transverse at or slightly above the level of the olecranon fossa (Fig. 466). The lower fragment is pulled backwards and thereafter carried upwards behind the shaft by the action of the triceps, and then tilted forwards by the weight of the forearm. The shaft projects forwards into the substance of the brachialis anticus. Less commonly the fracture may be partial or complete without displacement.

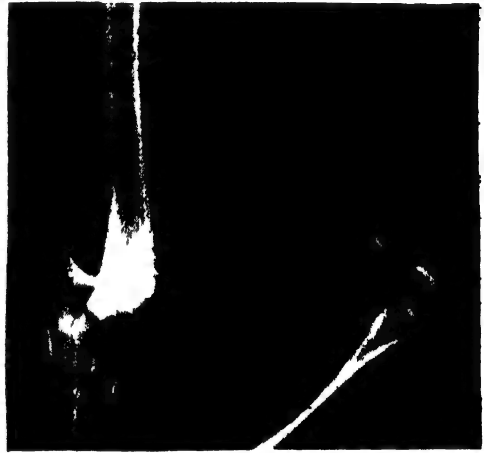


FIG. 466

Supracondylar fracture of the lower end of the humerus in a child showing the classical displacement.

In a typical case the forearm is held at an angle of 135 degrees with the arm, all voluntary movements are lost and the wrist is supported by the other hand. The projection of the shaft in the antecubital fossa and of the lower end posteriorly are obvious before swelling and bruising have appeared. On examination it will be seen that the epicondyles and olecranon maintain their normal relationship, and that the measurement from the external epicondyle to the styloid process of the radius is unaltered, but that the distance from the acromion to the external epicondyle is less than on the sound side. These signs serve to distinguish the injury from a dislocation of the elbow. Later the bony landmarks are obscured by swelling, but crepitus may be detected. In fractures without displacement the measurements are unaltered, but the swelling and bruising should suffice to suggest an injury to the bone. An X-ray must always be taken.

**B. Intercondylar or T-shaped Fractures** are more often due to violence applied to the point of the elbow and the backward displacement of the lower fragment is not therefore so common. This is a transverse fracture at or about the olecranon fossa, from which a vertical fissure runs into the joint. The lower end of the shaft is often forced into this fissure, thus separating the fragments. The clinical picture is similar

to that of supracondylar fracture, but the soft parts usually show evidence of injury and bruising is extensive and early. The condylar region is broadened and crepitus occurs with every movement.

C. *Separation of the Lower Humeral Epiphysis* may occur before the age of 6 years, but is a very rare injury. The displacement is similar to that in supracondylar fracture.

*Treatment of Supracondylar Fractures.*—Early reduction of these fractures is particularly important, since swelling appears rapidly and interferes considerably with replacement of the fragments. Careful examination for nerve injuries must always be made before attempting reduction. A general anæsthetic is always necessary. The fragments are first disengaged by gently extending the elbow and traction is then made on the forearm till shortening is overcome. The lower fragment is then forced forward into line with the upper and the elbow is gradually flexed. When reduction has been effectually performed the fracture feels reasonably secure, the bony points round the joint are in their normal relative positions, elbow movements are free and the carrying angle is equal to that on the other side. A posterior plaster slab is applied from the axillary level down to the wrist with the elbow flexed to 120 degrees (Figs. 467 to 470). If this amount of flexion causes obliteration of the radial pulse the amount of flexion must be reduced until the pulse can be felt. The wrist is supported by a collar-and-cuff sling. The circulation must be watched most carefully for the first forty-eight hours and the splints removed if the hand becomes swollen or cold, the pulse at the wrist faint, or if there is much pain in the forearm or hand. The *after-treatment* is divided into four periods of ten days each; during the first the arm is kept in the plaster case; during the second it is suspended from the neck by a sling tied round the wrist, and the angle of flexion at the elbow is increased daily to a maximum; in the third the arm is still kept suspended in full flexion, but relaxed several times a day for voluntary movements; and during the fourth active exercises are encouraged in the house, but the sling is worn out of doors.

In many cases complete reposition cannot be obtained by one manipulation, and further manipulations under general anæsthesia will be necessary. Until the general alignment is reasonably good this treatment should be adopted, and the moulding which follows use will ensure an ultimate result which is in every way excellent. If the position after several attempts at reduction remains unsatisfactory, it is advisable to let the fracture consolidate, wait for six months or a year until the range of movement shows no signs of further improvement and then perform a supracondylar osteotomy. The after-treatment is similar to that for a fracture reduced by manipulation.

T-shaped fractures should be reduced in a similar way to the above, but after the general alignment has been restored it is necessary to bring the condylar fragments together by lateral compression. Traction, combined with manipulation and lateral compression of the fragments, followed by a posterior plaster slab with the elbow flexed to about 30 degrees short of a right angle gives much better results than open operation with attempts at internal fixation. In some cases traction



FIG. 467



FIG. 468



FIG. 469



FIG. 470

**Reduction of supracondylar fracture**

Traction is applied and while it is maintained the elbow is flexed to 45 degrees above the right angle. Lateral displacement is corrected by direct pressure, and a posterior plaster slab is applied. (Watson-Jones)

on the fully extended elbow, combined with lateral compression of the fragments, followed by immobilisation in plaster in the extended position, gives good reduction, the plaster being removed after four weeks. It is not advisable to apply traction by ice-tong callipers on the condyles or by a pin or wire through the olecranon as these methods increase the stiffness in the elbow joint.

**PARTIAL FRACTURES OF THE LOWER END.**—Fractures of the condyles are caused by direct violence and by falls on the hand, the external being broken more frequently than the internal. In children between the ages of 5 and 15, the separated fragment of the external condyle includes the capitellum and an adjacent portion of the trochlea together with a part of the metaphysis above the capitellum and external condyle, carrying the external lateral ligament and the common extensor origin. Usually the displacement is slight, but sometimes the

triangular fragment is displaced outwards with marked rotation. Where there is no displacement, the elbow should be kept flexed for three weeks, followed by active exercises. If displacement is present the fragment may be manipulated back into position; should this be unsuccessful open operation is necessary, the fragment being guided back into position and sutured with catgut. Nails, screws or pegs are unnecessary and should not be used.



FIG. 471

Fracture of the internal condyle which has been drawn into the joint.

rarely much displacement. In the latter the condyle is snapped by the pull of the internal lateral ligament and is displaced downwards; it is sometimes drawn into the joint (Fig. 471). The ulnar nerve is frequently involved in this injury or it may become involved later in scar or callus formation. Open operation gives the most satisfactory result as it permits examination of the ulnar nerve in addition to dealing with the displaced epicondyle. The epicondyle is sutured by two catgut sutures back into position and the ulnar nerve transposed to the front of the joint if necessary.

*Fractures of the Capitellum* are rare injuries caused by direct violence. The fragment usually lies loose in the joint and may be considerably displaced, but exact recognition of the fracture is possible only by X-ray. The loose fragment should be replaced by operation, and the arm splinted in a position of nearly full elbow flexion; in late cases it may be necessary to remove the detached piece of bone.

*Fractures of the Internal Condyle* may be caused by direct or indirect violence. The former are often comminuted, but there is

## INJURIES OF THE ELBOW JOINT

**Dislocations.**—The bones of the forearm may be dislocated posteriorly, anteriorly or laterally on the humerus. *Posterior dislocation*, the only common one (Fig. 472), is caused by falls on the outstretched hand in a manner similar to that responsible for the supracondylar fractures. Dislocation, however, occurs chiefly in older children or adults. There is much pain and all movements are limited. The diagnosis presents no difficulty if the case is seen before the appearance of swelling. The olecranon projects behind the lower end of the humerus, and its distance from the two epicondyles is increased. Above the olecranon a hollow can be felt in which the slack triceps tendon is made out. On moving the elbow considerable resistance is encountered, but no crepitus felt unless the coronoid process is fractured. The distance from the lateral epicondyle to the radial styloid process is reduced, but that from the acromion to the lateral epicondyle unaltered, signs which serve to distinguish a dislocation from a supracondylar fracture. When the elbow is swollen these points cannot always be established, but the lower level of the backward projection and the absence of crepitus should favour the diagnosis of dislocation.

Reduction of a backward dislocation is usually very easy; the lateral ligaments are torn and the forearm bones slip into position with a dull snap when traction is made on the forearm with the elbow flexed to a right angle. It is important to bear in mind that the dislocation is always associated with some degree of injury to the brachialis anticus insertion into the coronoid process. This results in stripping of the periosteum (or occasionally a fracture of the coronoid process) with later ossification in the subperiosteal hæmatoma. The dislocation should therefore be reduced very gently and the elbow rested for three weeks in full flexion by a collar-and-cuff sling. X-ray examination after reduction is essential to confirm that reduction is complete and also to exclude fractures of the adjacent bones. The fingers, wrist and shoulder are exercised while the arm remains in the sling and after the sling is removed active exercises are started and will gradually, but sometimes slowly, restore full movement to the elbow. Massage, passive or forcible movements of the elbow are absolutely contraindicated, as they will only cause increase of pain and stiffness, with the possibility of the development of myositis

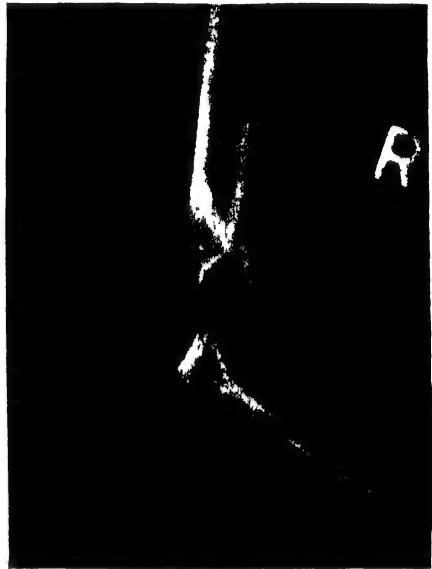


FIG. 472

Posterior dislocation of the elbow joint.

ossificans. If the coronoid process is fractured a posterior plaster slab should be used in addition to the collar and cuff.

*Anterior Dislocation* is rare and is usually accompanied by fracture of the olecranon. Replacement is easy, but the displacement is prone to recur unless the olecranon fracture is secured by operation.

*Lateral Dislocation* is also rare and easily reduced. The after-treatment is that of posterior dislocation.

*Dislocation of the Radius Alone* is nearly always anterior and usually accompanies fracture of the upper third of the ulna. The injury is as a rule due to a fall on some projection, which forces the head of the radius and lower fragment of the ulna forwards, but it is occasionally seen, without ulnar fracture, after falls on the hand. The orbicular ligament is torn, and the head of the radius lies in the supracapitellar fossa. The head can be felt in this situation and a hollow appears at its normal position. The forearm is held at an angle of 130 degrees and pronated, and movements of flexion, pronation and supination are considerably limited.

The dislocation can be reduced with little difficulty by traction on the forearm accompanied by pressure over the radial head, but recurs immediately unless the elbow is kept fully flexed. The arm should therefore be fixed with the elbow in a position of full flexion and supination, and kept in this position for four weeks. Extension beyond a right angle should not be attempted till the sixth week.

*Dislocation of Head of Radius with Fracture of the Upper Third of Ulna.*—In one type the head of the radius is dislocated forwards with forward angulation of the ulna. The displacement is reduced by traction on the limb, manipulating the ulna straight and flexing the elbow to a right angle while pressing the head of the radius back into position. A plaster is applied from the upper arm down to and, including the hand, in full supination with the elbow flexed to a right angle. In the other type the head of the radius is dislocated backwards with backward angulation of the ulna. Reduction is effected by traction with the elbow fully extended, the head of the radius being pushed forwards and the ulna straightened by pressure from behind. A plaster is applied with the elbow fully extended, from the axilla down to the supinated hand. As fractures of upper third of the ulna often heal very slowly, the plaster immobilisation may have to be continued for more than the normal four or five weeks.

*Subluxation of the Head of the Radius (Pulled Elbow).*—This injury is seen in young children who have been lifted by the wrist. The head of the radius is pulled partly out of the orbicular ligament, which becomes folded in the radio-humeral space. There is considerable pain with limitation of flexion and rotary movements. Reduction is readily effected by flexing the elbow while the forearm is alternately pronated and supinated. The elbow should be kept flexed after reduction for fourteen days by suspending the wrist from the neck with a loop of bandage.

*Tennis Elbow* is a form of sprain which occurs, as the name suggests, most commonly in tennis players, but is also seen in fly-fishers, painters, workmen who use hammers, and indeed in any occupation or



sport where quick elbow movements are carried out while the hand is grasping some implement. The characteristic symptom is pain in the region of the external epicondyle, which occurs whenever the hand is clenched; in severe examples the pain is brought on by any grasping movements, so that a tea cup may be dropped involuntarily. Upon examination a tender spot is found over, or just in front of, the external lateral ligament.

It appears that tennis elbow may take two forms. In one there is an injury to the common origin of the extensors of the wrist and fingers; in the other an arthritis, traumatic or infective, limited to the radio-humeral joint and causing swelling and tenderness of the synovial fringe which separates the bones forming this joint. The first type yields, in its early stages, to physiotherapy, and later may be cured by manipulation. The second demands rest and the removal of any focus of infection, or if the trouble persists, excision of the synovial fringe may bring about a cure.

#### COMPLICATIONS OF INJURIES IN THE REGION OF THE ELBOW JOINT

1. **Injury to Nerves.**—Of the nerves round the elbow, the ulnar is most commonly injured. In dislocations and traction fractures of the internal epicondyle it may be torn or crushed between the joint surfaces, as it may in attempts at reduction of dislocation or of supracondylar fractures; in supracondylar fractures it may be stretched over callus during the repair period; and in fractures leading to a valgus deformity tension on the nerve in its groove will lead to an ulnar palsy, often many years after the accident. Crushing and tearing of the nerve should be treated by suture and transplantation to the front of the joint; compression or late palsy is relieved by transplantation alone, the nerve being laid in a bed prepared in the substance of the common flexor origin. The median and musculospiral nerves have also been damaged in injuries in the region of the elbow joint.

2. **Ischæmic Paralysis (Volkmann's Paralysis).**—Ischæmic paralysis is due to injury or obstruction of the vessels at the bend of the elbow. It is most commonly seen following the supracondylar fractures of childhood, but it may occur in partial fractures or in dislocations. The vessels (especially veins) may be injured at the time of fracture or during reduction; or they may be compressed by bone fragments, œdema or blood clot during the next few hours, or still later by splints or bandages. The muscular fibres of the forearm muscles, especially the flexor group, undergo necrosis and are replaced later by fibrous tissue which contracts, causing a fixed and claw-like hand.

The possibility of injury to vessels should be borne in mind when the case is first seen. Later the limb must be carefully watched during the first forty-eight hours for evidence of impairment of circulation; should the patient complain of pain, the pulse become feeble, or the hand blue and cold, the bandages should be removed immediately and the limb fixed loosely in such a position that the circulation is

unhampered. When the obstruction is due to swelling round the fracture, necrosis of muscle may often be averted by one or more incisions through the deep fascia.

Once the condition is established the ultimate function of the wrist and hand will depend upon the amount of undamaged muscle tissue. At an early stage, intensive physiotherapy combined with passive stretching of the shortened flexors and maintenance in an over-corrected position will often achieve more than might be expected. The old "claw-hand" type of case can only be improved by operation. That of removal of equal lengths of the shafts of radius and ulna has been displaced by the muscle-slide operation of Max Page, in which the internal epicondyle of the humerus with its attached common flexor origin is detached and allowed after forcible overcorrection of the deformity to take up a new position in the forearm.

### 3. Limitation of Movement.—In children limitation of move-

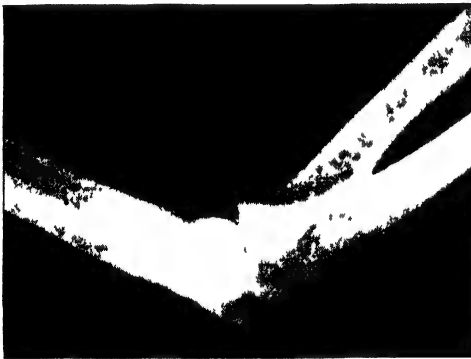


FIG. 473

Well-marked myositis ossificans in the brachialis anticus.

ment after injuries to the elbow is usually due to excess callus formation. This complication is especially liable to occur if early passive or forced movements are practised; masses of bone are thrown out, particularly in front of the joint, forming a mechanical block to flexion. When the insertion of the brachialis anticus into the coronoid process has been damaged, osteoblasts may be liberated into the muscle tissue and an increasing bony mass develop (Fig. 473), which if unchecked and untreated will result in grave restriction of movement. If the development of such a condition is suspected, absolute rest for a long period is essential and the new bone will gradually be absorbed and disappear, with a good functional result. Operative attempts at removal of this bone only stimulate further bone formation and are therefore contraindicated. On this account rest for three weeks is advisable in all cases of severe injury, the elbow being kept fully flexed, and thereafter only active movements should be encouraged, with no massage or passive movements.

In adults overproduction of bone is not so common, while, on the other hand, intra- and peri-articular adhesions may limit movements unless these latter are practised early. Limitation is usually in the direction of flexion and supination, and such injuries are therefore treated in the flexed and supinated position, but early voluntary movements should be allowed when they do not risk the position of a fracture.

## THE ULNA

**Fractures of the Olecranon** may be caused by direct or muscular violence. Those due to the former are either transverse, through the

middle of the sigmoid fossa, or comminuted, and the degree of separation of the fragments varies considerably. Those due to muscular violence occur near the tip, and separation is the rule. In either case the line of fracture enters the joint. There is pain and bruising and the joint is filled with blood-stained fluid and clot. Voluntary movements of extension cannot be performed. The gap between the fragments can be felt in fractures with separation, and crepitus will usually be detected on movement.

When there is little or no separation, it must be remembered that the triceps tendon insertion has probably been damaged as well. The elbow should therefore be immobilised for a fortnight in full extension by an anterior plaster cast extending from the axilla down to the wrist. The elbow is then gently flexed to a right angle and a sling is worn, active exercises being then started. In fractures with separation, reduction can be effected either by manipulation or by open operation. In manipulation the elbow is fully extended and the olecranon is manipulated forwards and downwards into position, the arm being immobilised in full extension by an anterior plaster case. The reduction is checked by X-ray examination, and if there is no gap the plaster is kept on for five weeks, after which voluntary movements to increase flexion gradually are started. In elderly patients this is the best treatment. In younger patients and where X-ray examination reveals that a gap still persists in the olecranon, open operation is necessary. With the patient lying prone with the arm abducted and fully extended on an arm table, it is easy to get very accurate apposition of the fragments. They are then sutured together with strong catgut or silk by a figure-of-eight suture and the triceps tendon is also repaired. A light posterior plaster slab is applied for four weeks, with the elbow flexed nearly to a right angle.

Where the olecranon fragment is small, it may be excised by careful dissection, but the triceps tendon must be strongly and accurately repaired. Immobilisation for ten days followed by active movements will lead to a good functional result.

**Fractures of the Coronoid Process** occur as complications of backward dislocation of the elbow and favour redislocation. When such an injury is present the elbow should be kept fully flexed for three weeks before movements are allowed.

**Fractures of the Shaft** are usually due to direct violence, such as falls on to a projecting surface or blows on the upraised arm. Pain and bruising are marked and the fracture is sometimes compound, but there is commonly little displacement when the radius is intact.

In cases without overlap the general alignment of the arm should be corrected and a light plaster case applied. When the ends are separated they should be replaced by manipulation under anaesthesia. In many cases operative reduction is required, but after such reduction internal fixation should not be needed.

### THE RADIUS

**Fractures of the Head** are usually due to indirect violence applied along the length of the bone, such as falls on the hand. The head is

splintered against the capitellum and, in the commonest type, the anterior and outer margin of the head is depressed or slightly detached. The head may be split by irregular cracks, etc., without displacement, the fragments being held together by the intact orbicular ligament, or there may be comminuted fractures of the whole radial head with separation of the fragments. The pieces may be completely detached and lie loose in the joint cavity.

The head of the radius may also be broken by direct violence, in which case deformity and separation of fragments are more common.

The chief *symptoms* are pain over the site of fracture and limitation of rotary movements and flexion at the elbow. Swelling appears locally, tenderness is present on pressure, and irregularity of the bone may be felt when the forearm is rotated. When a fragment lies in the joint the limitation of flexion is more marked.

Cases without displacement should be treated as sprains of the elbow in a sling, voluntary movements being started after the third or fourth day. By this means adhesions are prevented and the callus is moulded by the orbicular ligament. In the majority of such cases perfect function will result even when some slight deformity remains. When a portion of the margin of the head is depressed or where the separation of the fragment is only slight, conservative treatment in full extension will give excellent results if the orbicular ligament is intact. As the radius has been driven upwards at the time of the injury it must be pulled down and an anterior plaster slab applied with elbow fully extended and the forearm supinated. When, however, the fragment is much separated and tilted, excision of the fragment alone, if it is small, or of the head of the radius when the fragment is large or the bone is comminuted, must be carried out early. Limitation of extension is the chief disability following these fractures, but this disability can be avoided in most cases by treatment with the elbow fully extended. In children excision of the head of the radius should be avoided if at all possible and conservative treatment adopted, as the epiphysial line will be removed and so the growth of the bone will be seriously interfered with and this will later cause disability in the inferior radio-ulnar joint.

**The Shaft** may be broken by direct violence or by falls on the hand. In the majority of cases when the ulna is intact there is little separation of the fragments, owing to the muscular attachments which ensheath the bone, and the only deformity is some loss of the natural curve. When the ends are separated the displacement depends upon the nature of the violence and the pull of the muscles, particularly the pronators. When the radius is fractured with angulation or overriding with the ulna intact, there is always a disturbance of the normal relationship in the inferior radio-ulnar joint. In fractures above the middle of the shaft, the upper fragment is flexed and supinated by the biceps and supinator brevis, the lower pronated; in fractures below this point both fragments tend to be drawn towards the ulna. The most obvious symptoms of fracture of the shaft are loss of grasping and rotary movements, and pain and tenderness at the site of injury. Pain is produced at this point by pressure on the bone throughout its

length, but crepitus may not be obtained owing to the depth of the bone, intervention of muscles and absence of gross separation. It is often difficult to establish the presence of a fracture of the shaft on clinical grounds alone.

In cases without displacement the general alignment of the forearm should be corrected and a plaster case applied with the elbow at right angles and the forearm midway between pronation and supination ; such a plaster should include upper arm and hand. When displacement is present the deformity must be reduced under anæsthesia by traction and manipulation. With properly applied counter-traction on the upper arm and strong continued traction on the thumb and fingers the fragments can usually be made to engage in the fully reduced position. If reduction by this method is unsatisfactory, open operation under careful aseptic technique is essential. The fragments can usually be made to engage firmly and no internal fixation is necessary. Plaster is applied from the axilla down to the metacarpal heads, and the circulation in the fingers and their movement must be carefully observed for the first twenty-four hours for signs of ischæmia. After reduction a plaster case should be applied. In fractures of the upper third of the bone the elbow is flexed and the forearm fully supinated ; in those of the lower half the midway position between supination and pronation is adopted. The plaster must remain on for at least four weeks.

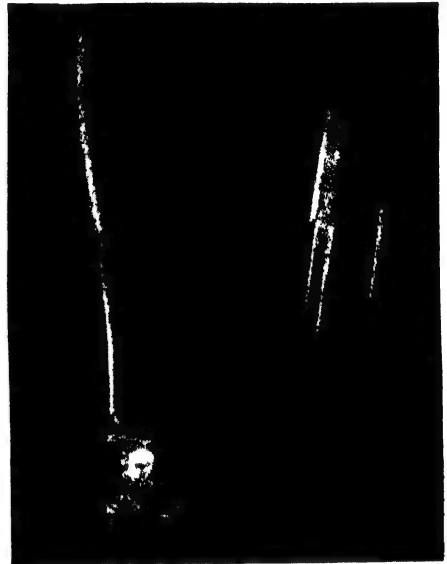


FIG. 474

Fractures of the shafts of both ulna and radius.

### THE RADIUS AND ULNA

Fractures of the shafts of both bones are commoner than those of either bone alone. In adults they are usually caused by direct violence ; in children they may also be due to this cause, but can follow a fall on the hand. The level of the fractures is about the middle of the forearm (Fig. 474), the radial fracture, however, being usually at a lower level than that of the ulna. In children the fractures are frequently of the greenstick variety. In many cases the ends of the fragments remain in contact or are only slightly displaced, and the main deformity is one of angulation, the forearm being bent with the concavity on the radial side. When separation has occurred the relative position of the fragments depends to a large extent on the direction of the causative violence, but in all cases there is a tendency for shortening with overlap to take place, and for the two bones to be drawn together, reducing the interosseous space. In fractures of both bones the classical signs will be observed, and the diagnosis is evident.

When there is no appreciable separation between the fragments, the general alignment of the forearm should be corrected and a plaster

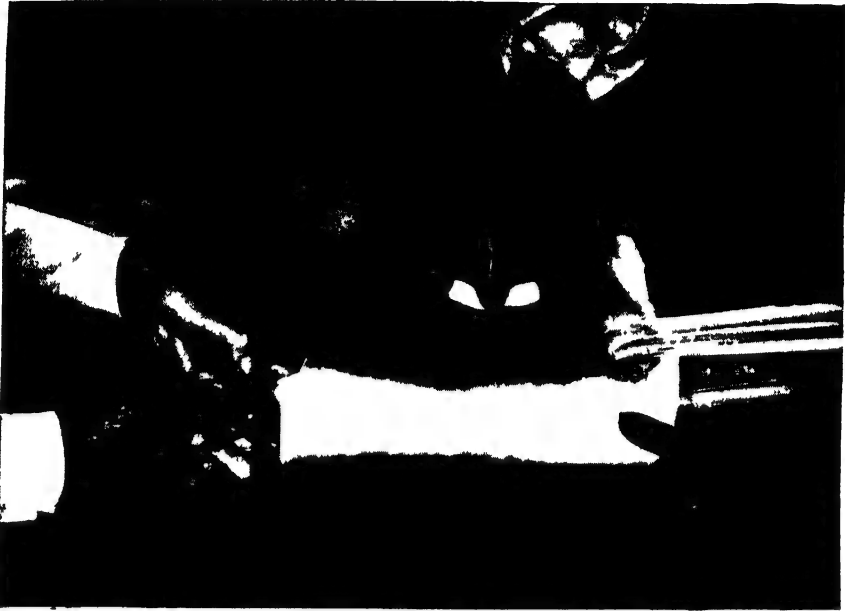


FIG. 475

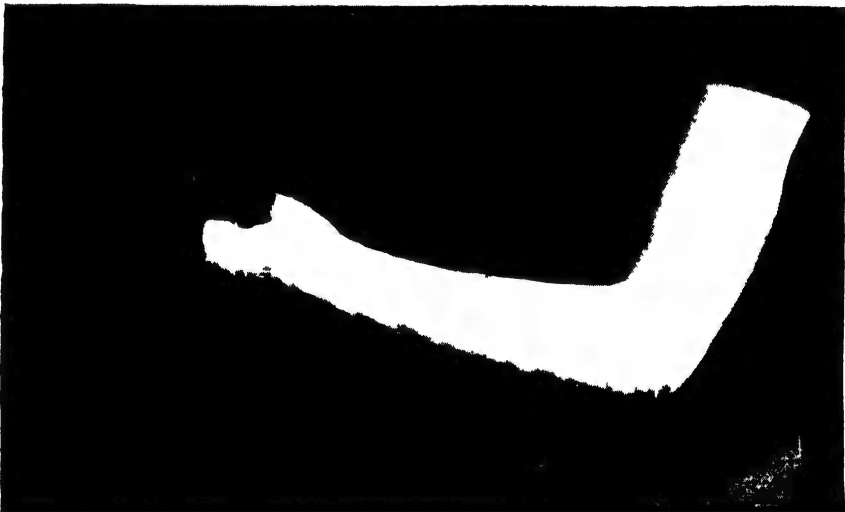


FIG. 476

#### **Fracture of both radius and ulna**

Strong traction is maintained against the counter-pull of a calico sling. After reduction a plaster slab and case is applied. Completed plaster for fractures of the forearm bones. The operator is testing for flexion contraction of the fingers, one of the signs of ischæmic contracture of the muscles due to a tight plaster.

(Watson-Jones.)

case applied, reaching from above the elbow to the palm of the hand. An anæsthetic is not always required. When the fragments are separated it is very important that accurate reposition should be

obtained, since considerable loss of rotary movements is otherwise very probable and cross union between radius and ulna, entirely abolishing such movements, will take place in a number of cases. Reduction should first be attempted under anæsthesia by traction in the long axis of the forearm, with strong counter-traction on the upper arm, the elbow being flexed to a right angle, and pressure on the distal fragments in such a direction as will bring them into alignment; for this purpose an X-ray, to show the exact displacement, is essential. Should this manœuvre succeed, a plaster case is applied (Figs. 475 and 476) and moulded so that the section is oval and not round, in this way maintaining separation of the bones from the interosseous space. The plaster must extend from the axilla down to the metacarpal heads, the elbow being at a right angle. Fractures above the middle of the forearm are splinted in elbow flexion and full supination, those below in the mid-position. Subsequent moulding of the fragments and replaster is often necessary especially after swelling has subsided. The plaster, which must not be replaced at any time by a shorter plaster, must remain on for eight to ten weeks and often longer. Immediately after the initial reduction a careful watch must be kept on the hand for signs of commencing ischæmia. Complaints of pain in the hand by the patient, signs of disturbance of the circulation and limitation of extension of the fingers are signs which call for immediate action, the plaster being at once split longitudinally and opened out. When a satisfactory reduction cannot be obtained by manipulation, reduction by open operation is essential, internal fixation being avoided if possible.

### FRACTURES IN THE NEIGHBOURHOOD OF THE WRIST JOINT

The region of the wrist is commonly injured by falls on the outstretched hand. In childhood the force of such an injury is usually transmitted to some point higher up the limb. In adolescence the lower epiphysis of the radius is most likely to separate. In adults the radius is strong, and dislocations or injuries of the carpal bones are relatively common. After middle life the lower end of the radius becomes progressively more brittle, and Colles's fracture is the commonest injury.

**Colles's Fracture** is commonly seen in the elderly, and is caused by a fall on the outstretched hand. The line of fracture passes through the expanded lower end of the radius, usually  $\frac{1}{2}$  to  $\frac{3}{4}$  in. proximal to the wrist joint. The classical fracture is oblique, passing upwards and backwards, but the line may be roughly transverse and varying degrees of comminution are the rule. Impaction is practically always present and usually firm. The styloid process of the ulna is frequently snapped across at its base, or the internal lateral ligament may be torn from its tip. The lower radial fragment remains attached to the ulna by the triangular ligament, and is displaced upwards, outwards and backwards to a varying extent and also rotated outwards and backwards (Fig. 477).

There are swelling and pain over the wrist, and voluntary movements are lost or much diminished. When viewed from the

back, the hand appears deviated to the radial side, the whole wrist is broadened and the head of the ulna abnormally prominent. From the side the lower radial fragment and the wrist are seen to project backwards from the line of the forearm, making, with the flexed fingers, the "dinner fork" deformity. Tenderness will be found over the lower end of the radius and tip of the ulnar styloid, and the radial styloid process lies at the same level or higher than the ulnar and more posteriorly. Crepitus is absent in most cases. While these signs are typical, displacement and deformity may be slight or absent, and the fracture can only be suspected by local swelling and tenderness and must be confirmed by an X-ray. It must be remembered that the lower articular surface of the radius normally looks forwards and slightly inwards as well as downwards.

*Treatment.*—Exact anatomical restoration is of the utmost importance in Colles's fracture, and is

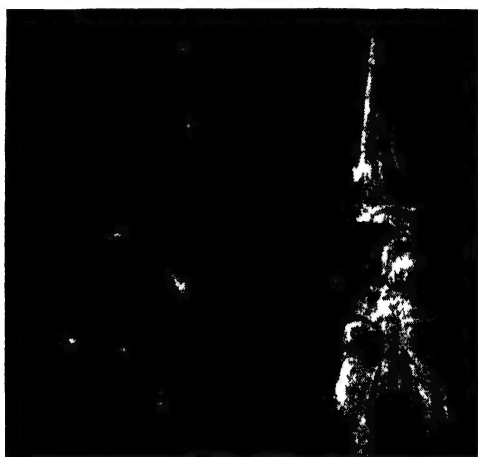


FIG. 477

Two views of a Colles's fracture illustrating typical displacement.

possible in the great majority of cases. Impaction should never be allowed to remain except in very old patients. In reduction the points to remember are that the lower fragment must be disimpacted and then pushed downwards and tilted forwards and pushed inwards firmly towards the ulna. The disimpaction is obtained by strong traction on the hand, this should be sufficient; but if it is not, while strong traction is maintained the wrist may be further but gently extended. The reduction is then carried out by direct pressure

on the lower radial fragment, which is pushed forwards and inwards and tilted forwards. With strong hands applying regulated pressure it should always be possible to get complete anatomical reduction, except in cases where much comminution is present. The person who reduces the fracture should hold it in position while the plaster is applied. The wrist is slightly flexed in full ulnar adduction. A posterior plaster slab is applied from just below the elbow to the metacarpal heads and this is fixed by plaster bandages so that the thumb and fingers are free, but the finger metacarpals are completely immobilised (Fig. 478). The patient starts immediately to exercise the fingers, elbow and shoulder many times a day. The limb is supported in a sling when not in use and the patient is encouraged to use the limb for dressing, eating and other daily duties, and gradually increase the activities of the limb. The sling should be discarded early. The plaster is kept on for four weeks. If there was much swelling at the time of reduction, the plaster will become loose when the swelling subsides and a new closely fitting plaster must be substituted after ten days. Unreduced or malunited Colles's fractures can still be reduced by manipulation up



to three or four weeks after injury, but later, and certainly after three months, open operation is essential for reduction.

**Separation of the Lower Radial Epiphysis** (juxta-epiphysial fracture) occurs in adolescents. The causes are those leading to Colles's fracture in adults, and the clinical features are similar, except that the displacement of the epiphysis is usually a purely dorsal one, without radial deviation or shortening. The displacement must be fully reduced by traction and strong pressure. There is a distinct tendency for redisplacement to occur. Hence these cases are best immobilised after reduction by a plaster case as in the Colles's fracture. The plaster must be kept on for three weeks.

**Reversed Colles's (Smith's) Fracture** is caused by a fall on the back of the hand, with the wrist flexed. The radius is broken at the same



FIG. 478

Completed plaster for Colles's fracture of radius. It extends over the thumb metacarpal and is closely moulded to the radius. There is no more than strapping in the palm. (Watson-Jones)

level as in Colles's fracture, but the line of fracture is usually transverse. The lower fragment carrying the wrist is displaced forwards. The styloid process of the ulna is often fractured in addition. Seen from the back the wrist is broadened, and the lower end of the shaft and the head of the ulna form a prominence sloping upwards towards the radial side. The hand and wrist lie on a plane anterior to that of the forearm.

Smith's fracture should be reduced under local anæsthesia by a manœuvre similar to that employed for the reduction of a Colles's, except that the lower radial fragment is pressed very strongly backwards after traction has disimpacted the lower radial fragment. When the lower fragment has been fully reduced, the wrist is immobilised in a plaster case in slight dorsi-extension. The plaster is kept on for four weeks.

**Chauffeur's Fracture** is caused by the backfire of a car which is being started by hand. The handle is forced violently against the palm of the operator's hand. In most cases a typical Colles's fracture

results. When, however, the hand is medially adducted at the time, a "chauffeur's fracture" may result. The line of fracture passes from the lower articular surface of the radius to a point on the outer side of the shaft 1 to  $1\frac{1}{2}$  in. above the joint. The triangular fragment thus detached is displaced upwards to a varying extent. The displacement is reduced by pulling the hand first downwards, then by manipulation, the radial fragment being pressed strongly back into position. Plaster is applied from just below the elbow to the metacarpal heads with the wrist in the neutral position.

If the handle slips from the grasp it will swing round and strike the back of the forearm above the wrist, producing a transverse fracture of the radial shaft  $1\frac{1}{2}$  to 2 in. above the lower end. The lower fragment is displaced forwards. This fracture, which has also received the name "chauffeur's fracture," may be caused by other forms of direct violence or, in children, by falls. Reduction is obtained by traction and backward pressure on the lower fragment. If, as is sometimes the case, redisplacement occurs readily, the fragment may require fixation by open operation.

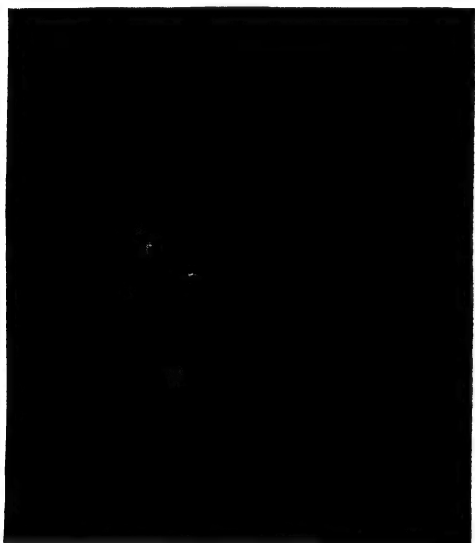


FIG. 479  
Fracture of the carpal scaphoid.

**FRACTURES OF THE CARPAL BONES** may be caused by direct or indirect violence. Those due to direct violence vary in extent and severity with the force causing them, and do not lend themselves to classification. Fractures due to indirect violence usually involve the scaphoid or semilunar.

**The Scaphoid** is broken in adults by falls on the hand. The common fracture is transverse across the middle of the bone and there is little displacement. The main symptoms are pain on dorsiflexion and radial adduction of the wrist, but this is so slight that the condition is often looked upon as a sprain. On examination, some swelling will be noticed in the anatomical snuff-box, and direct pressure on the bone is painful. Very careful X-ray examination is required. Radiographs in three different planes are necessary and may have to be repeated in cases where tenderness in the radial side of the carpus follows an injury (Fig. 479).

If the position is good, as it is in the great majority, the wrist should be fixed in plaster in 45 degrees dorsi-extension until the fracture is united. The plaster must ensure complete immobilisation of the fragments of the scaphoid and must grip the thumb metacarpal as well as the other metacarpals (Fig. 480). The fracture may heal in six or eight weeks, but some cases have to be immobilised for six months or more. Removal of the plaster and further careful X-ray examina-

tion is essential in deciding whether union is sound. Patients can usually carry out their full work in the plaster case, provided the plaster does not get wet or loose.

Excision of the scaphoid or a fragment of it is not followed by good results, the function of the hand usually being poorer after than before surgical intervention. In cases where union has not been obtained by prolonged fixation in plaster or in those where the fracture has been overlooked at the time of injury, the resultant painful wrist can often be materially benefited by exposure of the fracture, the removal of sclerosed bone and further plaster immobilisation. Subcutaneous drilling of the fractured surfaces or the insertion of small

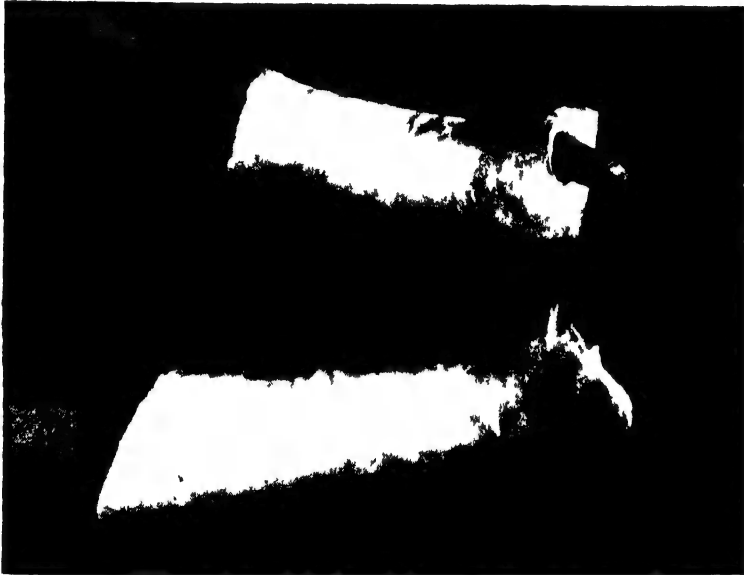


FIG. 480

Bilateral fracture of the scaphoids showing the type of plaster case used. It extends to the metacarpal heads and includes the whole of the first metacarpal. The hand is tightly gripped so that there cannot be any trace of wrist movement, but the plaster in the palm does not extend beyond the transverse skin creases. (*Watson-Jones*)

bone grafts have not given such satisfactory results as the simple roughening of the fracture surfaces.

**The Semilunar** is also broken by falls on the hand and in boxing. Symptoms are even less marked than in the case of fractured scaphoid, and use of the hand is continued. Under these circumstances the bone may become rarefied and later collapse. Cases seen early should be treated by eight weeks fixation in plaster. It is doubtful whether excision in late cases is of any benefit.

#### DISLOCATION OF THE WRIST AND CARPAL BONES

**Sprains of the Wrist** are caused by twisting strains and by forced extension or flexion. A pure strain is uncommon and should never be diagnosed until an X-ray has excluded fracture. The joint is

swollen and painful and all movements are limited. A sprained wrist should be strapped firmly with adhesive plaster, followed after a week by massage and graduated active movements.

**Dislocation of the Wrist (radio-carpal) Joint** is rare and, as a rule, the result of gross violence. The displacement of the hand may be posterior or anterior; the former is usual. The deformity in wrist dislocation closely resembles that in Colles's or Smith's fracture, and because of the severe pain and considerable swelling which appears within a few moments the clinical distinction may be difficult. The normal relation of the two styloid processes in a dislocation should permit its recognition.

Reduction is effected by traction under anæsthesia and is usually easy. A plaster splint well moulded round the wrist and extending to the metacarpo-phalangeal joints, should be applied with the wrist in a position of 45 degrees dorsiflexion. It is kept on for six weeks, after which massage and movements are given.

**Dislocation of the Semilunar** is much commoner than that of the wrist. It usually follows forced dorsiflexion. The semilunar is more commonly displaced and rotated forwards, so that its concave lower surface faces anteriorly. The patient complains of pain and limitation of wrist movements. A hollow may be noticed on the back of the wrist proximal to the os magnum, and an ill-defined swelling is felt in front under the flexor tendons. Grasping movements are weak, and pain referred to the distribution of the median nerve is often present. An X-ray is necessary to establish the diagnosis.

The dislocation is reduced by a strong steady continuous pull on the hand, without any levering movements of flexion or dorsiflexion. This should be continued for at least ten minutes and combined with strong pressure anteriorly over the dislocated bone. After reduction is complete, traction should be maintained during the application of a dorsal plaster splint which for the first ten days should hold the wrist in 45 degrees flexion. This manœuvre should be successful even in the worst cases if the injury is fairly recent. In cases of six months standing with a median nerve lesion, the bone should be removed. This, however, will not improve the function of the wrist and therefore is only indicated when the nerve is damaged. In cases of one to six months standing, reduction may be carried out by open operation combined with the screw traction method of Böhler.

**Dislocation of the other Carpal Bones** is usually due to severe direct violence, and the line of separation may involve any of the intercarpal or carpo-metacarpal joints. In mid-carpal dislocation the displacement of the distal portion is backwards and is often associated with a fracture of the scaphoid. Such injuries do not lend themselves to any useful classification.

## FRACTURES OF THE METACARPALS AND PHALANGES

**Fracture of the Base of the First Metacarpal** (Bennett's fracture) is due to indirect violence applied in the line of the bone, and is most frequently seen in boxers. The fracture passes obliquely downwards

from the middle of the articular surface. remains articulating with the trapezium, and the shaft is displaced upwards, outwards and backwards (Fig. 481). There are pain and swelling over the base of the thumb.

Reduction is easily affected without anæsthesia by traction and pressure on the base of the shaft. The traction must be maintained until the union is sound, otherwise the displacement of the shaft will recur. The traction is obtained either by skin traction or pin traction from a wire finger splint incorporated in a forearm and hand plaster. These are retained for four or five weeks (Fig. 483).

**Fractures of the other Metacarpals** may follow direct or indirect violence. In the first case the fracture is transverse or comminuted, and there is much bruising but little displacement. With indirect violence (blows on the knuckles), the line of fracture is often oblique and deformity is more common,



FIG. 481  
Bennett's fracture.



FIG. 482  
Fracture of neck of fifth metacarpal with typical displacement.

being one of shortening and angulation backwards. In either case there are pain and tenderness over the injured bone, and, if it is grasped by the knuckle, abnormal mobility and increased pain will be noticed. When there is displacement, the line of the knuckles will be irregular (Figs. 482 and 484).

**Fractures of the Phalanges** are usually due to direct violence, and the proximal is most commonly affected. The fracture may be transverse or comminuted; when displacement is present it takes the form of forward angulation.

Fractures without displacement, whether of the metacarpals or phalanges, require simple splinting only. In the case of the metacarpals a malleable iron splint is the best; in the phalanges, a slip of metal bent at 45 degrees opposite each interphalangeal joint. When there is displacement, traction is necessary. A length of fine rustless wire or a Brock's pin (Fig. 485) is passed through the pulp of the finger and attached to one end of a padded wire or metal splint whose other end is embedded in a plaster casing at the wrist. By bending splint and finger together, extension is exerted on the finger and its metacarpal. In the case of metacarpal

fractures, the metacarpo-phalangeal joint should be straight and only the finger joints bent, while a dorsal slab of plaster may be placed over the back of the hand if angulation is present.

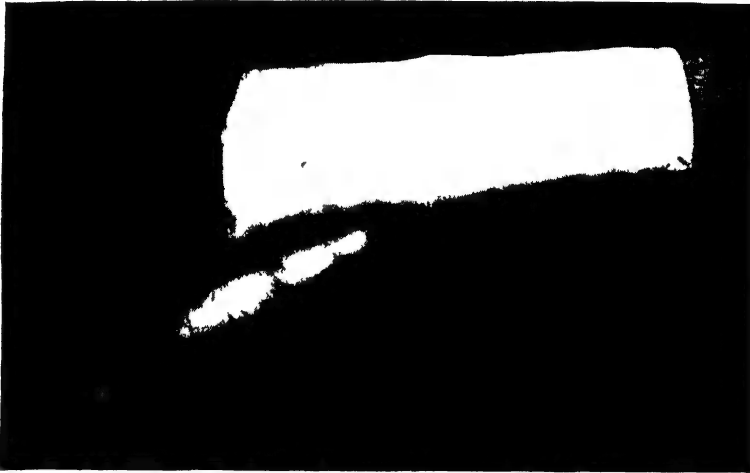


FIG. 483

Plaster well moulded over the base of the thumb and continuous skin traction for Bennett's fracture-dislocation of first metacarpal.

In cases with multiple fractures of metacarpals and phalanges, extension can be applied to all fingers from a special metal frame attached



FIG. 484

Plaster cast for fractured neck of fifth metacarpal. While the plaster is setting the phalanx is pressed backwards and the displacement is reduced. (Watson-Jones)

at both ends to a plaster case at the wrist and passing round the ends of the fingers and some 2 to 3 in. beyond them (Fig. 486). Extension is made from the fingers either by strapping or by fine

wire or gut through the pulp of the distal segment. Many of these metacarpal frames have adjustable screws to allow alteration in tension on each individual finger.

#### **Injuries of the Joints of the Hand.—**

Sprains of metacarpo-phalangeal and inter-phalangeal joints are very common. The whole joint and its peri-articular structures are swollen and movements are limited. The chief feature of these sprains is their chronicity, and three or four months often elapse before full painless movement is regained. The affected joint should be protected by strapping and voluntary movements encouraged.

#### **Dislocation of the Metacarpo-phalangeal Joints**

is caused by forced hyperextension. The base of the first phalanx is displaced on to the back of the metacarpal and forms an obvious projection. The digit is held semiflexed and movements are limited and painful.

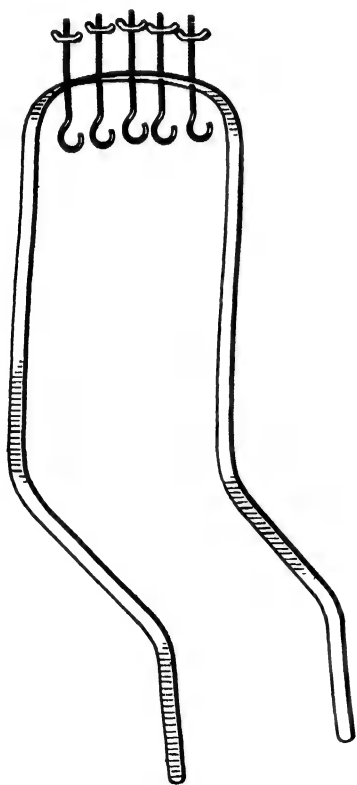


FIG. 486

Splint for the treatment of fractures of the phalanges which need extension.  
(Allen & Hanburys.)

a small chip of bone carrying the insertion of the tendon is detached (Fig. 487). The terminal phalanx remains flexed and voluntary extension is impossible.



FIG. 485

A Brock's Pin.  
(Allen & Hanburys.)

Reduction should be attempted by pulling on the finger, hyperextending at the same time, and then pushing the phalanx forwards while extension is continued. Reduction is by no means easy. The glenoid ligament remains attached to the base of the phalanx and catches behind the neck of the metacarpal. In the case of the thumb the heads of the flexor brevis and the tendon of the flexor longus pollicis, which grip the head of the metacarpal, further hinder reposition. When manipulation fails, reduction by operation, preferably through a postero-lateral incision, is necessary.

**Dislocation of the Interphalangeal Joints** is relatively infrequent, and the more distal phalanx is always displaced backwards. Reduction is easy, but the joint will be painful for months and may be permanently arthritic.

**Mallet Finger** is due to blows on the tip of the finger and is therefore very common in wicket-keepers. The extensor tendon is torn from the base of the distal phalanx, or, more commonly,

To obtain a full functional recovery the finger must be immobilised for not less than six weeks with the proximal interphalangeal joint



FIG. 487

Mallet finger showing fragment of bone from postero-articular aspect of terminal phalanx.

flexed 90 degrees and the distal joint hyperextended to its limit. This is best done by a carefully applied anterior plaster case extending



FIG. 488

The finished result after the plaster has dried. (*Farguharson.*)

from the web to the tip of the finger. Even when the injury is of many weeks' duration this method, continued for eight weeks or more, will give a satisfactory result (Fig. 488).

A. E. PORRITT.  
R. Y. PATON.



## CHAPTER XLVI

### INJURIES OF THE LOWER LIMB AND OF THE SPINE

#### INJURIES OF THE LOWER LIMB

##### FRACTURES OF THE PELVIS

**F**RACTURES of the pelvis fall into two groups: those which involve integrity of the pelvic girdle and those which affect outlying portions. The first are grave injuries, the second in most cases serious only when they are accompanied by lacerations of important soft structures.

**Fractures of the Pelvic Girdle.**—The pelvis, formed by the two innominate bones and the sacrum, is a strong ring of bone and ligament which transmits the weight of the trunk to the lower limbs and encloses the lower part of the abdominal cavity. Fracture is due to considerable violence, such as motor accidents, falls from a height, or crushing forces. A break at one part of the ring is almost necessarily accompanied by one at the opposite part, and fractures usually occur at the two weakest places, the thyroid foramen in front and the region of the sacro-iliac joint behind. In front, the line of fracture passes through the horizontal ramus of the pubis at its outer end, and the descending ramus at its lowest part. Behind, the sacro-iliac joint itself is only occasionally separated, but the typical fracture passes from the iliac crest 1 or 2 in. external, to enter the joint below; less commonly it involves the ala and the first two foramina of the sacrum, again entering the lower part of the joint. There is usually little displacement, but sometimes the side which is fractured behind is displaced upwards.

Fracture of the pelvis may be accompanied by damage to any of the pelvic viscera, nerves or blood vessels, but of these structures only the urethra and bladder are commonly injured. The urethra is torn in its membranous part by rupture of the triangular ligament. The base of the bladder, which is comparatively fixed, may be injured by a similar mechanism or lacerated by fragments of bone. The fundus of the bladder, if it is full at the time, may be ruptured by the violence of the injury. The rectum, vagina and pelvic blood vessels being less firmly attached are rarely involved, and nerves also usually escape injury, unless the line of fracture passes through the sacral foramina.

**Diastasis of the Symphysis Pubis** may occur during childbirth or follow falls on the perineum. In the latter case, injury to the urethra or base of the bladder is common. The pubes usually spring together after the violence has ceased, so that no displacement remains to

indicate the injury, which can therefore only be recognised by damage to the urethra, and by extreme pain localised to the symphysis when the iliac crests are compressed or movement is attempted.

The *diagnosis* of fractured pelvis is difficult. Owing to the severity of the accident the patient is usually shocked, often even unconscious, while severe visceral injuries may distract attention from the fracture. The cardinal symptom is a sense of insecurity, with inability to move the legs or trunk. Pain is felt in the pelvis when movement is attempted, and is increased by passive movements of the legs or compression of the iliac crests. Tenderness may be found over the fracture of the pubic ramus or near the sacro-iliac joint, and irregularity of contour may be noticed on palpation of the ischial and pubic rami from the perineum or through the vagina or rectum. There is, however, no gross deformity unless one side of the pelvis is displaced upwards. In this case the measurement from the umbilicus to the anterior superior spine is decreased on the affected side, but other measurements, such as Nélaton's line and Bryant's triangle, are unaltered. In every case, injury to the urethra and bladder should be suspected until it has been excluded and the patient should be warned not to micturate. When the urethra is torn, blood trickles from the meatus and a swelling appears in the perineum; if the patient attempts to pass urine, none appears at the meatus, but extravasates into the perineum. When the bladder is torn, urine cannot be passed, but no blood appears; if the rupture is intraperitoneal, the abdomen soon becomes distended. A catheter should always be passed. When the urethra is torn, the catheter is arrested in the perineum, and a few drops of blood may be withdrawn. When the bladder is ruptured the catheter passes easily, but no urine, or at most a few drachms of blood-stained fluid, is obtained.

*Treatment of Fractured Pelvis.*—The treatment of accompanying visceral lesions (pp. 760 and 785) is a matter of prime importance and should be dealt with as soon as possible after the causal accident, due allowance being made for allaying shock, which is often severe in these cases. In most fractures the displacement is slight and prognosis is little altered by preliminary investigation and treatment of injuries to neighbouring soft parts. Reduction is as a rule unnecessary, but occasionally gentle manipulation or a short period of leg traction may effect a definite improvement in position. The majority of cases simply require careful nursing in bed—preferably on a divided mattress—the pelvis being pulled together either by a firm binder, adhesive strapping or, if necessary, a light plaster.

Immobilisation is maintained for a minimum of six weeks, although before the end of this time leg movements should be encouraged in the lying position. In all but the worst cases the patient can sit out of bed within two months from the date of injury and should start walking with the aid of crutches. As a rule, reasonable walking or return to work is not possible under three months.

**The Acetabulum** is fractured by indirect violence.

The upper and back part of the rim is often broken in dislocation of the hip. Such an injury may be suspected when the dislocation is

reduced easily, is accompanied by crepitus and recurs after reduction; the diagnosis is confirmed by X-rays. The limb should be fixed in a Thomas' splint in a position of full abduction, and weight traction applied for eight weeks. A walking caliper is worn for a further ten weeks.

The floor of the acetabulum can only be fractured by force transmitted through the femur, either along its neck from falls on the trochanter, or along the shaft. Depending on the degree of violence, the socket may be splintered without displacement, or the head of the femur driven into the pelvis—so-called *central dislocation of the hip joint*.

Walking is impossible and all voluntary movements of the hip limited. Pain is felt in the groin and is often referred along the distribution of the obturator nerve. Passive movements, especially of internal rotation, are restricted. The leg is shortened, the trochanter raised and nearer the midline than its fellow. On rectal examination a boggy swelling and occasionally crepitus will be felt on the side wall of the pelvis. An X-ray will confirm the diagnosis.

Skeletal traction, with the limb abducted, is the most satisfactory method of obtaining reduction. The extension, through a tibial tubercle pin, must be maintained for at least two months, after which a weight-bearing caliper must be worn for three months. The fractured portion of the acetabulum may not be restored to position as the head is pulled out and then open operation will be required.

**The Crest of the Ilium** can be broken by direct violence. There is usually little displacement, the attached muscles keeping the fragments in position, and in any case only minor disability will follow deformity in this situation. It is usually sufficient to support the injured site with bands of elastoplast for three weeks.

**The Anterior Superior and Anterior Inferior Spines of the Ilium**, especially the latter, may be broken by muscular violence. The accident usually occurs in boys about the age of puberty, and leads to localised pain at the site of fracture, and limited extension of the hip joint. A good X-ray will demonstrate the injury.

There is usually little separation, and it is sufficient to keep the patient in bed for four weeks, with the thigh flexed over a pillow. When the spine is completely detached it may be fixed with a screw or bone peg.

**The Ischium** is broken by falls in the sitting position and blows on the tuberosity; there is usually little displacement. The chief symptom is pain in the buttock, increased by sitting, movement of the limb, and defaecation. Rest in bed for a period of four to six weeks is sufficient treatment, but a prolonged period of pain and disability may ensue.

**The Sacrum** may be broken in its upper part in conjunction with fractures of the whole pelvis, the line of fracture passing through the upper foramina. In this case involvement of the sacral plexus is common.

Fractures of the sacrum alone usually involve the lower half, and are due to blows, or falls in the sitting position. The line of fracture

is transverse, and the lower fragment is displaced forwards. The rectum may be torn or the lower sacral nerves injured. The displaced fragment can be pushed back by a finger in the rectum and shows little tendency to redisplacement. The treatment consists in rest in bed for six weeks and attention to the bowels.

**The Coccyx** is fractured or displaced by falls in the sitting position. There is normally little displacement, but the lower fragment or the whole coccyx may be displaced forwards or to one side. Pain is experienced in sitting, walking and defæcation. Any displacement can be reduced by manipulating the fragments between the index finger in the rectum and the thumb on the surface, but reduction can seldom be maintained. In the majority of cases, nevertheless, no residual disability exists even when the fragments remain displaced, especially if the patient can be kept in bed for a week or ten days. Strapping the buttocks across the top of the natal cleft gives considerable relief from pain in the early stages. A few cases, however, will complain of persistent pain (*coccydynia*) after this injury. For them an extensive course of physiotherapy should first be prescribed and, if this fails, the injection of some analgesic solution (*e.g.*, "A.B.A.") should be tried before excision of the bone is recommended. This latter operation is not difficult but relieves the pain in only about half the cases, as it is not essentially the bone which is at fault. The true cause of *coccydynia* is involvement of the coccygeal sensory nerves in fibrous tissue formed as a result of the primary injury.

### DISLOCATION OF THE HIP JOINT

The hip joint possesses great natural strength, and dislocation is therefore rare. Violence applied to the hip in childhood usually leads to a juxta-epiphyseal fracture, and in the aged to fracture of the femoral neck. In adults a pure dislocation can occur only if violence is applied when the hips are fully abducted, so that the head of the femur is thrust against the lowest and back part of the joint, where the capsule is thin and the bony margin of the acetabulum deficient. The head of the femur may be forced out of the socket in other directions, but only if the acetabular rim is fractured at the same time.

Dislocation is due to forced hyperabduction, common instances being the sudden separation of the thighs on an insecure or moving foothold, and the fall of a weight on the sacrum of a labourer stooping with legs wide apart. The head of the femur is forced through the capsule at its lowest point, and thereafter passes up behind or in front of the acetabulum, the direction depending chiefly on that of the force causing the dislocation. The Y-shaped ligament nearly always remains intact, and governs the position of the limb. Four typical dislocations are described, two posterior and two anterior.

**Posterior Dislocations.**—(a) Gluteal, (b) sciatic. If the head of the femur is forced backwards after leaving the joint it may pass above or rupture the tendon of the obturator internus, and come to lie over and behind the acetabulum, when the dislocation is called gluteal; or it may rest below the intact tendon, immediately behind the

acetabulum, when it is called sciatic. In posterior dislocations the leg is flexed, adducted and internally rotated, the sole of the affected side resting on the dorsum of the sound foot, the knee above the sound knee (Fig. 489). Real shortening of 1 to  $1\frac{1}{2}$  in. is present, and apparent shortening in excess of this. The great trochanter is raised, Scarpa's triangle feels empty and the head can be made out under the gluteal muscles. All movements are very restricted. There is always considerable pain and shock, and there may be evidence of pressure on the great sciatic nerve. There is less shortening and inversion in a sciatic than in a gluteal dislocation.

**Anterior Dislocations.**—(a) Obturator, (b) pubic. The head of the femur, passing forwards, may remain at the thyroid foramen (obturator dislocation) or come out to rest against the horizontal pubic ramus (pubic dislocation). In anterior displacements the leg is flexed abducted and rotated outwards. There is apparent lengthening. The head can be felt in Scarpa's triangle, and the femoral vessels are displaced inwards. Pain may be referred along the distribution of the obturator or anterior crural nerves.

*Reduction* should be carried out immediately under general anæsthesia, which must be pushed to the point of complete muscular relaxation. The patient is placed on his back on a low couch or the floor, and an assistant steadies the pelvis. The head of the femur is in each case made to retrace its course to the lowest part of the joint. In posterior dislocations the thigh is first fully flexed, adducted and internally rotated, relaxing the Y-shaped ligament. The surgeon then presses firmly downwards on the flexed knee, bringing the head below the acetabulum. He then rotates the thigh outwards to bring the head towards the midline, circumducts the thigh outwards and finally extends it.

In anterior dislocations the thigh is first abducted and rotated outwards, then pressed downwards in this position, rotated inwards while pressure is maintained and finally circumducted inwards and extended. In either case the reduction will occur with an audible snap during the movements of circumduction and extension. If



FIG. 489

Traumatic dislocation of the hip joint with typical adduction, internal rotation deformity and shortening. (Watson-Jones)

manipulation fails after several attempts, the dislocation can be reduced by strong traction on a Hawley table, but such a method inevitably damages the articular surfaces.

After reduction the leg should be immobilised in a plaster spica for six weeks, in a neutral position apart from slight abduction, before weight-bearing is allowed. When there is also a marginal fracture of the acetabulum an extension should be used for four weeks before the plaster spica is applied.

**Irregular Dislocations** are those in which the Y-shaped ligament is torn. The head of the femur may pass into any position, and the characteristic deformities of the regular dislocations are not seen. Reduction and redislocation are both easier than in the regular varieties, and a longer period of recumbency is advisable.



FIG. 490

Adolescent coxa vara.

## FRACTURES OF THE FEMUR

### THE UPPER END

**Fractures of the Head of the Femur** are rare, and are usually seen as complications of dislocation of the hip joint. The head may be indented or fissured, but the displacement is usually so slight that the injury can only be recognised with certainty by X-rays. Weight traction in a Thomas' splint is applied for four to six weeks, but daily movements of the joint must also be practised. At the end of six weeks a walking caliper is fitted. Osteo-arthritis is an almost unavoidable sequel.

**Separation of the Upper Femoral Epiphysis (Adolescent Coxa Vara)** is a common injury between the ages of 10 and 15 years. The epiphysis is occasionally separated at the time of a severe accident. More frequently the causative accident is a minor one and the injury, which is probably a cancellous fracture without separation in the juxta-epiphyseal region, passes unnoticed. This injury is most commonly seen in "fat boys." There is no immediate disability and the child continues to get about. The weight of the body gradually displaces the head downwards on the neck, which comes to lie against the roof of the acetabulum, and is also rotated forwards (Fig. 490). Increasing pain and limping call attention to the disability. On examination the thigh is found to be externally rotated, and slightly flexed and adducted. There is about  $\frac{1}{2}$  in. of shortening, and the trochanter is raised to a similar extent. The neck may be felt as a prominence in Scarpa's triangle. Movements of abduction and

internal rotation at the hip joint are considerably limited. An X-ray will demonstrate the displacement of the head of the femur. The other hip may be similarly affected, so it must also be kept under careful observation throughout.

In the majority of cases partial repair has already taken place and gradual correction only is possible. In any case, forcible correction is undesirable as it leads to osteo-arthritis. Both legs, therefore, are placed in Thomas' splints slung from an overhead frame in a position of slight abduction (about 20 degrees). Extension weights, in an average case 15 lbs. to the affected limb and 8 lbs. to the sound one, are applied. The progress of correction is checked by films taken with a portable X-ray apparatus. When correction is complete it is most important to remember that the essential feature of the treatment is to get the epiphysial line fused either by healing or by operation. Not until the fusion is sound must weight-bearing be permitted.

If repair has taken place in a position of deformity, intertrochanteric osteotomy may be necessary to correct adduction, and to delay the development of arthritis.

**Fractures of the Neck of the Femur** (Intracapsular Fractures) are typically seen in the aged, and may follow apparently trivial accidents, such as tripping over a carpet. The line of fracture is irregularly transverse through the narrowest part of the neck

(Fig. 491). The amount of separation varies. The deeper reflected fibres of the capsule pass inwards on the front of the neck from the intertrochanteric line towards the head, and form strong fibrous bands which may retain some connection between the fragments and prevent displacement. Occasionally the distal fragment is impacted into the head. More commonly there is complete separation, and in this case the distal part of the neck is displaced upwards by the force of the accident and the pull of the muscles, and rotated outwards by the weight of the limb as the patient lies in bed.

In cases without separation, or in those with impaction, the clinical signs of fracture are few, and diagnosis may be almost impossible without X-rays, which should always be demanded a good lateral view being absolutely essential. Pain is minimal and the patient is sometimes able to walk. On examination there is little or no shortening or other deformity and the condition may be thought to be one of osteo-arthritis aggravated by injury. In osteo-arthritis, however,



Fig. 491

Intracapsular fracture of the neck of the femur.

there is usually a long history and obvious wasting of the thigh muscles.

Typically the patient is helpless, unable to walk or move the limb, and may be considerably shocked. Pain is felt over Scarpa's triangle and bruising appears over the outer part of the space, but this is late in appearance and of moderate degree. The limb lies fully everted. Shortening is usually obvious, and on measurement may be from  $\frac{1}{2}$  to 3 in. The trochanter is raised to the same extent, and the iliotibial

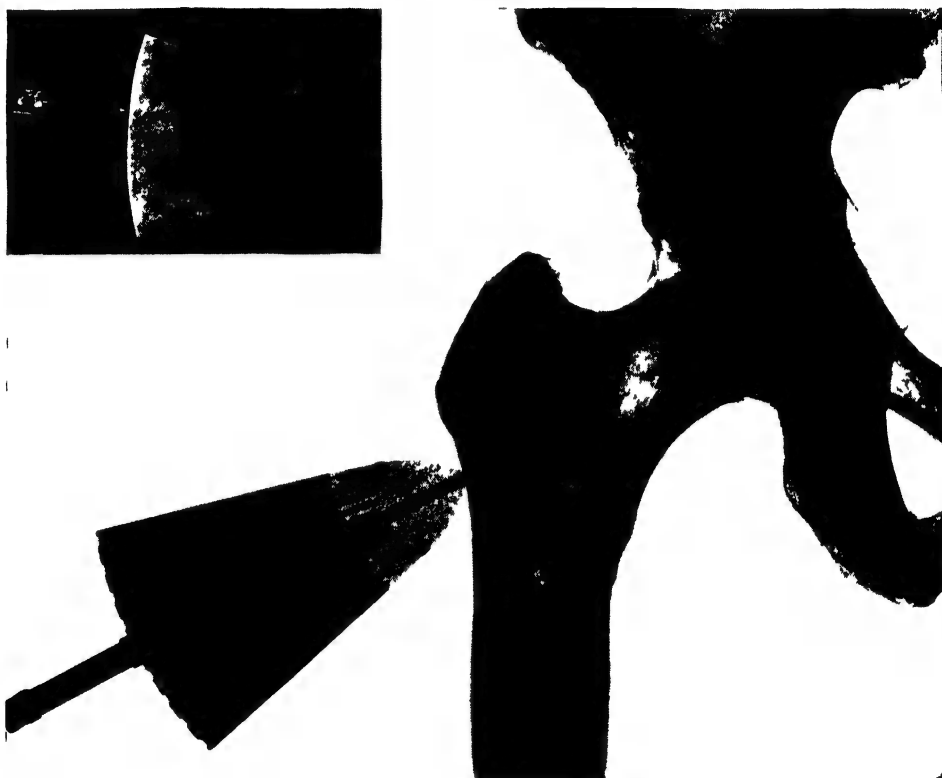


FIG. 492

Engel-May directing apparatus to facilitate accurate insertion of guide wire. The radiating cannulae are marked with lead strips. The correct line is chosen first in anteroposterior view, and then in lateral view after rotating the apparatus through 90 degrees. (Watson-Jones.)

band is slack. On moving the limb, crepitus is noticed and pain in the groin increased.

Union after fracture of the neck of the femur is in all cases greatly delayed, and non-union is common. The reasons for such unsatisfactory repair are the age and poor general health of the average patient, atrophy of the neck which has predisposed to fracture, and the lack of an independent blood supply to the head of the femur; in addition, apposition between the fragments is often imperfect, and immobilisation incomplete and insufficiently prolonged.

In healthy vigorous patients the best prospect of bony union in perfect position and ultimate recovery of complete use of the limb is offered by immediate fixation of the fragments. After reduction of



the deformity by manipulation and, if necessary, traction, antero-posterior and lateral X-ray examination is carried out and, if the position is satisfactory, a guide wire is inserted from a point on the outer aspect of the shaft of the femur  $\frac{1}{2}$  in. below the lower margin of the great trochanter up along the axis of the neck into the upper fragment. The satisfactory placing of this guide wire having been checked by X-ray examination in the two planes, a Smith-Petersen pin is driven along the wire (Fig. 492). The length of the pin must be carefully calculated. Early movements of the hip are encouraged from the day after operation, but weight-bearing is not permitted for at least two months and not until there is X-ray evidence of union. The pin is about  $\frac{1}{2}$  in. in diameter and is three-flanged, being Y-shaped in transverse section (Fig. 493), the narrow blades causing very little damage to the cancellous bone of the neck while fixing the fragments firmly.

An alternative method is the *Whitman plaster*. Under general or spinal anæsthesia the fractured limb is extended on a Hawley table till shortening is overcome; it is then rotated inwards to bring the distal fragment into apposition with the head, and finally abducted to the fullest extent, when the broken surfaces are forced together by the leverage of the shaft over the fulcrum of the trochanter resting above on the rim of the acetabulum. The limb is maintained in this position by a double plaster spica from the nipple line to the foot, which is well moulded round the iliac crests. The plaster must extend down to and include both feet. After two months the plaster is removed from the foot to above the knee; in another month it is removed completely and if X-ray examination shows satisfactory union a month to six weeks is spent moving the limb in bed without retentive apparatus. A weight-bearing caliper may then be applied and worn for at least six months.

When the condition of the patient does not warrant the risk of anæsthesia and immobilisation in plaster, satisfactory correction can usually be obtained by traction. The limb is slung in a Thomas' or Hodgen's splint, and a pull of about 20 lbs. applied by skeletal traction. When shortening is overcome, which should occur within twenty-four hours, the leg is inverted and slightly abducted. If an X-ray then shows satisfactory position, the extending weight may be reduced to 10 lbs. After eight weeks a light plaster spica is applied, extending to just above the knee, and worn for a further three months. A walking caliper must then be used as described above.

Of these possible methods that of traction is very useful in the elderly type of patient that usually suffers from this injury, and it is often followed by satisfactory function, although non-union is fairly common. The Whitman plaster is not, on the whole, to be advocated, non-union being almost as common as with traction and the plaster



FIG. 493

Smith - Petersen pin showing also cross-section of the blades. (Allen & Hanbury's.)

being difficult to apply and cumbersome to wear; the advantages over traction are few and the disadvantages many. The Smith-Petersen pin is consequently becoming more and more popular for most cases and many advances in the technique of introduction both by open operation and by closed methods have recently been suggested. The success of this method, however, depends on good X-ray photographs, accurate reduction of the fracture and careful and skilled technique in the insertion of the pin. In cases of ununited fracture of the neck of the femur, an osteotomy should be done just above the level of the lesser trochanter and the shaft displaced medially so that it comes to lie under the head.

In the very old, where prolonged recumbency is considered hazardous, it may be necessary to abandon any attempt at obtaining union. The patient is fitted with a "bucket-top" caliper, and allowed to walk after a few days. The caliper must be worn permanently, since without union the neck of the femur slips up on to the dorsum ilii and gives a flail joint, on which walking is almost impossible.



FIG. 494

Drawing of the femur showing the usual lines of fracture of the upper and lower ends.

**Fractures of the Neck of the Femur in Childhood** are commoner than was realised before the general use of X-rays. The fracture is usually of the greenstick type, and the child may continue to walk. Coxa vara develops later. Such fractures should be immobilised in plaster for eight weeks in full abduction, and a caliper worn for three months after union.

**Trochanteric Fractures** (Extracapsular Fractures of the Neck) differ in every respect from those of the neck (Fig. 494). They are caused by major violence, are seen in healthy adults and unite readily. The accident is in most cases a fall on the trochanter on a hard surface, as in skating. The fracture is usually extracapsular behind and partly intracapsular

in front. It is irregular and almost invariably comminuted, the neck, whose under surface bears the very dense *calcar femorale*, being driven into the trochanteric region, breaking it into several fragments. Some degree of impaction is usually present.

Clinically the patient is shocked, in great pain and unable to use the limb. The trochanteric region is bruised and swollen. The leg is everted and shortened from  $1\frac{1}{2}$  to 2 in. The trochanter is raised, broadened and nearer the midline than the opposite one, but these measurements can rarely be made accurately owing to the swelling. The iliotibial band is relaxed. Because of the frequency of impaction some amount of voluntary movement may persist, and crepitus is usually absent.

Owing to the comminution which is always present trochanteric fractures must be treated by continuous traction. Both limbs are slung in Thomas' splints from an overhead frame at an angle of about 25 degrees abduction. The injured limb is in addition flexed 30 degrees at the hip, and a knee flexion piece attached to the splint.

A weight of 10 lbs. is attached to the sound limb and one of 30 lbs., which may be reduced to 15 lbs. as soon as the position is satisfactory and there is some evidence of union, to the injured limb. After eight weeks the splints may be removed and hip movements allowed in bed. One week later the patient is fitted with a walking caliper, which must be worn for at least three months.

**Fractures of the Great Trochanter** or its epiphysis are rare, and are usually due to direct violence. In most cases there is negligible separation, and fixation of the limb in abduction for four weeks is sufficient treatment. If the process is entirely separated, it should be fixed with a screw or bone peg.

**Fractures of the Lesser Trochanter** are due to muscular violence, and are often seen as a complication of extracapsular and intertrochanteric fractures.

#### THE SHAFT

**Fractures of the Shaft** of the femur may occur at any level, and may be caused by direct or indirect violence. Fractures due to direct violence are usually transverse, but may be comminuted, often to an extreme degree; those due to indirect violence are oblique or spiral, and usually single. The displacement depends to some extent upon the direction of the force causing the fracture. In all cases there is a tendency for the lower fragment to be drawn up by muscle spasm, causing shortening, and to be rotated outwards by the weight of the leg. At the upper end the proximal fragment is flexed, abducted and rotated outwards by the muscles inserted into the trochanter. At the lower end the distal fragment is tilted backwards by the origins of the gastrocnemius.

The patient is helpless and often shocked. Shortening, abnormal mobility and crepitus are usually apparent, and make the diagnosis of fracture easy, but the deep situation of the bone and the swelling, which is usually extensive and appears early, render an exact recognition of the site of fracture and of the displacement difficult. An X-ray is required to establish these facts.

*Treatment.*—All fractures of the femoral shaft are accompanied by varying degrees of shock and this condition should always receive suitable treatment before more radical measures are attempted. During this stage and as a first-aid method the injured limb should be fixed by simple extension (*e.g.*, a clove hitch around the boot) in a large Thomas' knee splint. This is preferable to a long Liston splint, which does not allow extension.

All fractures of the femoral shaft are reduced by extension. This may be applied manually and held by suitable position as in fractures of the upper and lower thirds; it may require strapping or skeletal traction, as in most fractures of the middle third; or it may necessitate in the worst or difficult cases operative intervention and mechanical methods.

**Upper Third.**—The situation here is governed by the fact that it is impossible to control the small upper fragment, which is flexed by the iliopsoas and externally rotated and abducted by the gluteus

medius and obturator internus. Hence reduction requires the bringing of the large lower fragment—the rest of the limb—into line with this displaced upper fragment. Immobilisation alone is not sufficient as the upper fragment is very liable to become redisplaced unless continuous traction is also applied. This can be achieved by using Robert Jones' abduction splint, by a plaster spica with adhesive strapping extension fastened to the raised foot of the bed, the body-weight maintaining the traction, or by the well-leg traction method.

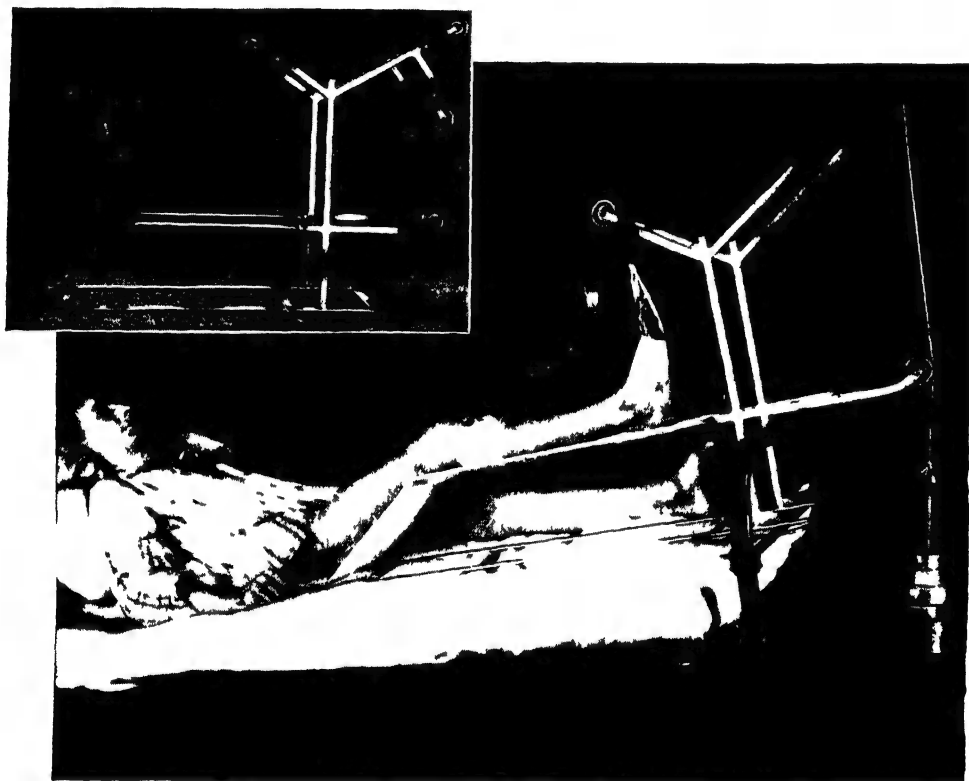


FIG. 495

Bohler-Braun splint with tibial skeletal traction for supracondylar fracture of femur. The angle of the splint must lie behind the fracture, not behind the knee. An alternative, lower line of traction is sometimes necessary in supracondylar fractures and can be secured by the four-pulley splint (inset). (Watson-Jones)

**Lower Third.**—In these fractures there is a large upper fragment and a small relatively uncontrollable lower fragment, usually flexed by the heads of gastrocnemius—sometimes to the extent of producing pressure on or actual damage to the popliteal vessels. Reduction, therefore, consists in aligning this large fragment to the lower small one by manipulation and fixation on a Thomas' knee splint or a Braun's splint (Fig. 495) traction being maintained either by strapping or by a pin or wire through the tibial tubercle. In either splint the knee is held flexed 45 degrees and the foot is supported at a right angle, the foot of the bed being raised.

**Middle Third.**—Here treatment depends upon the type of fracture

and the displacement. Direct violence usually produces transverse fractures with or without displacement. In the former case overlap and shortening occur, in the latter only angulation deformity is possible. Indirect violence is responsible for the very common oblique or spiral fractures in which both overlap and angulation may occur, and in which the tendency for the sharp ends of the fragments to become entangled in surrounding muscle fibres is very marked.

Transverse fractures without displacement require no reduction, the limb being fixed by strapping extension in a straight Thomas' knee splint and suitable pads or slings applied to the side bars of this to correct any angulation deformity.

Transverse fractures with overlap may in a few cases be reduced manually if seen early or if they occur in weakly or poorly muscled patients. In this case they are treated as above. The majority of such cases, however, require prolonged extension. This may be applied either by strapping extension or by skeletal traction. In the former case a weight of some 15 to 20 lbs. is required, in the latter up to 30 lbs. is applied at first and this can be halved when reduction is effected and only fixation traction is necessary. Skeletal traction is certainly the more effective. It is obtained by a Kirschner wire through either the lower part of the femoral shaft just above the condyles or the tibial tuberosity. In view of the possibility of sepsis and also of the liability to stiffness in the knee joint following supracondylar extension, skeletal traction through the tibial tuberosity is preferable. The latter, despite the heavy traction employed, does not cause laxity of the ligaments, provided the weight is carefully regulated and reduced when the fracture begins to unite.

In either type of extension due attention must be paid to adequate counter-extension by raising the end of the bed on blocks, and to the prevention of foot drop by the addition of a suitable foot-piece to the splint or by fixing the sole of the foot via a small strip of strapping and a cord to a small weight of about 1 lb. from a pulley on the overhead Balkan frame.

If such extension methods have not proved successful in reducing the overlap within a week or ten days, operative reduction must be considered. Operative correction, however, tends to increase the fixation of the quadriceps to the femur and so ultimately limit the range of flexion in the knee joint. Exposure is obtained by an incision on the outer aspect of the thigh and the fragments are levered into place. The fracture being transverse in type no internal fixation should be necessary and the leg is put up with fixed extension on a straight Thomas' splint.

In the oblique and spiral types of fracture little can be achieved by manipulation, so that extension by one of the above methods is usually essential. Even so, the liability of the fragments to become impacted in surrounding muscle makes the possibility of ultimate operative fixation more likely, and in these cases some form of internal splintage may be necessary. The less internal mechanical fixation the better, and it should be remembered that the presence of these retentive fittings in no way lessens the necessity for the usual external splinting and fixation.

In all types of fracture, once reduction and immobilisation are obtained, every effort must be made to maintain function in the rest of the limb. The knee joint must be held in a slightly flexed position and must never be allowed to become hyperextended. Foot, ankle and, where possible, knee movements should be encouraged, and faradic stimulation to the quadriceps *in situ* in the splint will do much to prevent the marked muscle-wasting which accompanies these fractures. Traction or immobilisation should be maintained for ten to twelve weeks, after which, if clinical and radiological union are satisfactory, the leg should be allowed to move freely in a 'Thomas' splint, the patient still being kept in bed for about four to six more weeks. At the end of this time a well-fitting walking caliper is applied and the patient allowed to get about. At this stage effusion into the knee joint is common, but this should rapidly disappear as the tone of the quadriceps is recovered. Weight-bearing without the caliper is seldom possible before nine months from the time of the fracture.

**Fractures of the Femoral Shaft in Children** are comparatively common. They are due to the same causes as those of adults, and present similar clinical features, but partial fractures without displacement are more often seen.

In new-born infants fractures of the shaft of the femur produced during delivery are best treated by being left alone. The recommended method of fixing the thigh by flexing it fully on to the baby's abdomen is not necessary. In this type of fracture it is astonishing how the most marked displacement corrects automatically within a week or two and how rapidly union occurs.

In children under five the "gallows" method of Bryant gives excellent results and has obvious advantages for nursing purposes over any kind of splint. A strapping extension is applied to both legs from groin to ankle and the cords from these extensions are tied to a bar fixed transversely above the frame of the cot at such a height that the buttocks are just lifted clear of the mattress. After union has occurred (four or six weeks), another month in bed in a light plaster should be followed by the use of a walking caliper for three months.

In older children the methods advocated for adults are preferable. Skeletal traction, preferably by Kirschner wire, is well tolerated. Where the fracture is a partial one, a fissured fracture without displacement, or one of the greenstick variety, a plaster spica is recommended.

### THE LOWER END

Fractures of the lower end of the femur are due to direct violence. The line of fracture is either transverse just above the condyles or T-shaped, a vertical fracture running from the transverse one into the intercondylar notch (Fig. 494). The upper fragment is usually displaced forwards, and engaged in the fibres of the quadriceps. The lower fragment is tilted backwards by the gastrocnemius. In T-shaped fractures the shaft may be driven between the condyles, separating them widely.

The region of the fracture is swollen, and the knee joint distended

with blood or synovial fluid. Abnormal mobility and crepitus become apparent when the limb is handled. Shortening of from  $\frac{1}{2}$  to  $1\frac{1}{2}$  in. can be made out, and in T-shaped fractures the condylar width is usually increased. The lower end of the shaft can be felt above the patella, and the tilted lower fragment may be palpable in the popliteal space, but swelling usually prevents their recognition. The popliteal artery may be compressed or torn by the lower fragment, so that the leg and foot are cold and the tibial pulses absent. If the artery is torn, a swelling appears rapidly in the popliteal space, and gangrene of the leg usually follows in a few hours.

*Reduction* of transverse or T-shaped supracondylar fractures should be carried out at the earliest moment in order to relieve possible pressure on the popliteal vessels. Direct control of the lower fragment is obtained by skeletal traction. This is applied from a wire through the tibial tubercle. The knee is flexed to 45 degrees and a Braun's splint is used. Twenty to 30 lbs. extension is used initially, but must be reduced to 10 or 15 lbs. after reduction has been effected. If there is still separation of the condyles after general alignment is restored, they are forced together by manual pressure or a screw clamp, the jaws of which are padded with felt. These fractures usually unite in from eight to twelve weeks, and after union is radiographically complete active movements of the knee are started. A caliper is worn for a further three months.

T-shaped fractures which cannot be brought into satisfactory position by manipulation must be replaced by open operation through a lateral incision.

Fractures of one condyle may be caused by direct violence, or by strains of abduction or adduction applied to the leg. When there is no separation the limb is fixed in a plaster case, with the leg forced towards the opposite side from the injured condyle; the case is retained for eight weeks, but can be bi-valved to allow gentle knee movements within three to four weeks, and thereafter a walking caliper worn for three months. If there is separation, the condyle should be replaced by operation if the displacement cannot be reduced by extension and manipulation.

**Separation of the Lower Femoral Epiphysis** is usually due to forced hyperextension of the knee in patients up to the age of 20 years. The epiphysis is displaced forwards, the patella forming a prominence in front, while the end of the shaft projects into the popliteal space. Displacement may, however, occur in any direction, depending on the nature of the violence, *e.g.*, in forcible correction of knock-knee, when the femur has been insufficiently divided with the osteotome, the epiphysis may be displaced outwards. If completely separated, it is rotated backwards by the gastrocnemius. Replacement should be attempted by manipulation under anæsthesia or by the method recommended for supracondylar fracture; operative reposition is seldom necessary.

### THE PATELLA

Fractures of the patella are of two distinct types; those caused by a sudden muscular contraction and those due to direct violence.



**Fractures due to Muscular Contraction** are usually the result of a stumble, the full power of the quadriceps being suddenly exerted on the patella while the knee is flexed. The quadriceps tendon or patellar tendon may in some instances be torn, but in the great majority the patella itself suffers, being snapped across the convexity of the lower end of the femur. The line of fracture is single and transverse, generally about the middle of the bone, but sometimes nearer the upper or, more commonly, the lower end (Fig. 496). The quadriceps tendon is ruptured laterally on either side of the patella. The separation may be negligible, only a linear crack marking the fracture, but in a typical case the muscular pull continuing after the actual fracture pulls the fragments apart, a gap of from  $\frac{1}{2}$  to 2 or 3 in. separates the two parts, the torn fringes of the quadriceps aponeurosis hang over the broken edges and the lateral parts of the capsule are torn. The joint cavity becomes distended with blood and synovial fluid.



FIG. 496

Fracture of the patella.

The patient is unable to walk or extend the leg. The knee is swollen, and the gap between the fragments may be felt through the skin.

In those cases where there is no separation of fragments, or where the patient is too old or too ill to stand operation even under local anæsthesia, an attempt should be made to approximate the fragments with strapping, the band above the patella being of horseshoe shape. The knee is immobilised in full extension in plaster or on a back splint, and movements are started after four

weeks, including walking, but the splint or plaster should not be removed for six weeks.

In the average case, operative treatment offers the best prospect of reasonable function, since only by this means can separated fragments be approximated, aponeurotic fibres cleared from between them, the capsule repaired and blood removed from the joint. The accurate reconstitution of the quadriceps tendon, of which the patella is only a part, is the aim of the operation. The front of the joint is exposed by a horseshoe incision convexity downwards, the fragments separated, and blood and clot removed from the joint. The broken surfaces of the patella are cleared, and aponeurotic fibres clipped away from the edges. The two parts are then fixed together in accurate apposition, so that no ridge is left on the articular aspect, by strong catgut sutures through the aponeurosis. The quadriceps tendon on either side is carefully sutured with mattress stitches. A light plaster



case is applied and after ten days walking is allowed. The quadriceps is exercised from the beginning. The plaster is retained for two months.

**Fractures due to Direct Violence** are the result of blows or falls on the patella. The bone is fissured in an irregular manner, but unless the patient has attempted to walk after the injury there is usually little separation and the capsule is not torn. The knee joint is distended with fluid, but contains little blood.

Operation in such cases is often unnecessary. The treatment is that for fractures due to muscular violence without separation. If the fragments are separated they must be carefully dissected out. The quadriceps tendon is then reconstituted with strong catgut mattress sutures. By overlapping portions of the tendon the final result is such that the absence of the patella is not noticed externally. A firm wool pressure bandage is applied and no splint is necessary. The patient can start to walk and exercise the knee after ten days. This procedure has given surprisingly good functional results, and it can also be used in simple fractures of the patella. In cases where one of the two fragments is small, excision of this fragment followed by reconstitution of the quadriceps tendon is all that is necessary. Compound fractures call for excision of the patella.

### INJURIES OF THE KNEE JOINT

**Contusions.**—Owing to the size of the knee joint, bruises involving articular cartilage or synovial membrane, which may lead to considerable effusion of fluid, are not necessarily accompanied by damage to the capsule. In the absence of such damage, rest is unwise. The joint should be strapped or firmly bound with a crêpe bandage to promote absorption of fluid, and active use of the limb encouraged from the start. Massage and quadriceps exercises will hasten recovery.

**Sprains** are usually the result of indirect violence, movements of rotation or of abduction or adduction beyond the normal range of the joint. Fibres of the capsule are torn to varying degrees, or separated with a flake of bone at their point of attachment, and the synovial membrane is inevitably injured at the same time. Sprains are characterised by effusion into the joint, pain on movements which stretch the torn ligament, and tenderness at the point of injury.

Forcible abduction of the leg is the form of indirect violence to which the knee is most often subjected, and **injury to the internal lateral ligament**, therefore, the most common form of sprain. Abduction may be due to falls or slips, or to blows on the outer side of the knee while it is bearing weight. There is a sudden severe pain on the inner side of the joint, and the patient, while able to put his foot to the ground, can only walk with pain and difficulty. Swelling appears within a few minutes and increases slowly. The knee cannot be fully extended, but this limitation only involves the last 20 degrees or so of extension, and the movement is arrested not by a sudden block but by pain and muscle spasm. On examination, swelling of the joint is apparent and bruising may be seen on its inner side. A point of tenderness will be found over the internal lateral ligament, most

commonly at its femoral attachment; this accurate localisation of the tenderness is an important point in the distinction of a sprain from a torn internal cartilage, where the tender point is half-way between the patella and lateral ligament, and from a nipped synovial fringe, where it is close to the patellar tendon. It must, however, be borne in mind that the internal cartilage is often damaged at the same time as the ligament. A gap may be felt in the course of the ligament, on abducting the knee pain is increased and laxity of the joint may be demonstrated.

The *treatment* of a torn internal lateral ligament is necessarily a compromise between the need for securing sound repair in the ligament and the desire to assist absorption of fluid and maintain the tone of the muscles. The measures to be adopted will depend upon the severity of the ligamentous damage, since unsound repair will leave a permanently weak knee. If the ligament appears to be torn across, the limb is fixed in a plaster case from the groin to the ankle, with the knee flexed 30 degrees and adducted as much as possible. Quadriceps exercises are ordered from the start, and walking is allowed after a week. After eight weeks the plaster is removed, but a shoe, raised  $\frac{1}{4}$  in. on the inner side of the heel, is worn for three months, and quadriceps exercises and massage are continued till full functional control of the joint has been regained. The provision of a knee cage should be confined to those cases where a marked lateral rock still exists after prolonged quadriceps rehabilitation. In some cases where the periosteal attachment to the femoral condyle has been separated, ossification occurs and this has been called Pellegrini-Stieda's disease—traumatic ossification following an internal lateral ligament injury. In less severe injuries the joint may be strapped over a small felt pad at the site of injury, and walking on a wedged shoe commenced after a week.

**Dislocation of the Knee.**—This injury can only occur when both lateral and both cruciate ligaments have been torn, and it is therefore due to gross violence. The displacement of the tibia on the femur may be lateral, anterior or posterior, the direction depending on that of the violence. Lateral dislocations are more common, while anterior and posterior ones are more serious, owing to pressure on the popliteal vessels. There are great pain, considerable swelling and inability to walk. The diagnosis is usually obvious, but it is difficult to exclude a concomitant fracture.

The reduction of a dislocation of the knee is extremely easy, since the ligaments are torn, and it should be performed at once. The limb should be fixed in a fully extended position in a close-fitting plaster case extending up to the groin and including the foot. Quadriceps exercises may be started after a fortnight, and walking may be allowed after the fourth week, but the case should be retained for six months. Considerable limitation of movement, only part of which may be regained, is to be expected after such prolonged fixation, but any shorter period is insufficient to allow strong repair of the torn ligaments. A flail knee cannot be efficiently controlled by a cage or satisfactorily repaired by any known operation, and is a far greater disability than a stiff and stable one.

**Dislocation of the Patella** is a rare injury and seldom occurs in a normal joint, there usually being some knock-knee deformity or laxity of ligaments due to previous injury or disease. Dislocation may be caused by direct violence, or, in the case of faulty alignment, by the sudden pull of the quadriceps while the knee is flexed. The only common displacement is outwards. Inward displacement and rotary displacement, the patella being turned through a right angle with one edge engaged in the trochlear surface, are described, but seldom encountered. Dislocation is accompanied by considerable pain and the knee is fixed in the flexed position. The patella can be seen and felt in its abnormal situation, and, after straightening the knee, can usually be pushed into place without difficulty.

**HABITUAL DISLOCATION** of the patella is seen in women. The first dislocation occurs in knock-kneed girls at about the age of puberty, and is due to injury or some sudden strain. Thereafter displacement recurs with lessening provocation and increasing frequency, and may happen almost daily. Dislocation may be prevented by a knee cage which limits flexion and carries a pad supporting the patella on its outer side, but in most cases operation is preferable. Any pronounced degree of knock-knee should be corrected as a preliminary measure. The most successful method of curing the recurrent dislocation is by transplanting the tubercle of the tibia and the patellar tendon to the inner side of the tibial crest; laxity of the inner side of the capsule may be taken up at the same time. The patella may also be excised and the quadriceps strongly reconstituted.

### INTERNAL DERANGEMENTS OF THE KNEE

The term "internal derangements" embraces a group of injuries to the knee, usually due to twisting or abduction strains, in which damage to internal structures predominates. Some lesion of the capsule, or more especially of the internal lateral ligament, almost necessarily accompanies the intra-articular one.

**Nipping of a Synovial Fringe.**—In sudden unguarded movements of the knee joint, folds of synovial membrane may be drawn between the articular surfaces and nipped when these latter come into apposition. Synovial thickening or laxity of ligaments from previous injury or disease predispose to the accident. A typical example of such an injury is nipping of the ligamentum alarium internum, the free fold which passes from the infrapatellar pad of fat to the interior of the joint. Sudden abduction of the leg separates the joint surfaces on the inner side, and a sudden return to the normal position crushes the fold between the internal femoral condyle and the internal meniscus. There is momentary pain on the inner side of the joint, often severe enough to inhibit the muscles maintaining extension and "let the patient down." Walking is, however, possible with the knee slightly bent.

Upon examination after a recent injury the joint is found distended with fluid, and some swelling on the inner side of the patellar tendon will also be noted. Full extension is impossible, but the limitation is

indefinite within about 10 or 15 degrees of the full range, this being in sharp contrast to the complete block to extension at 30 or 40 degrees flexion which is found when the internal semilunar cartilage is engaged between the joint surfaces. A point of tenderness is usually found over the pad of fat just to the inner side of the patellar tendon; in injury to the internal lateral ligament the tender point is on the inner side of the joint, midway between front and back, and in damage to the cartilage it is half-way between these places.

Nipping of the fringe is followed by effusion into the loose fat underlying it, which in turn makes fresh nipping likely; with repeated injury the fringe becomes thickened and fibrous and liable to be caught in any sudden movement. Full extension is impossible and the attempt painful.

After the first attack the knee should be strapped to reduce swelling, and the heel of the shoe raised  $\frac{1}{2}$  in. to prevent full extension of the joint. If the pain and disability persist, manipulation under an anæsthetic—acute flexion and pulling the tibia forward on the femur—will often effect a cure. Should the injury become habitual, excision of the thickened fringe is necessary.

#### INJURIES OF THE SEMILUNAR CARTILAGES

The semilunar cartilages (menisci) of the knee joint are crescentic structures lying on the upper surface of the tibial tuberosities. The internal cartilage is C-shaped, broader behind than in front. It is firmly attached to the capsule of the joint posteriorly and medially, especially to the internal lateral ligament, but the narrow anterior horn is only anchored by its tip to the transverse ligament, and behind this possesses considerable movement. The external cartilage forms three-quarters of a circle, and is broad throughout. Its two horns are attached close to each other in front and behind the tibial spine, and its periphery is only loosely connected to the capsule, being separated from the external lateral ligament by the tendon of the popliteus. Both cartilages are wedge-shaped in section, and serve as washers which convert the flat upper surfaces of the tibial tuberosities, to which they are attached by the coronary ligaments, into saucer-shaped hollows in which the femoral condyles move. Thus the cartilages form part of the tibial articular surface, but they can glide on the tibia to some extent with movements of the femur. These are chiefly hinge movements, but some rotation takes place during which the internal condyle moves round an axis formed by the external one. The external cartilage is thus exposed to hinge and rotary movements only, while the internal is also called upon to follow antero-posterior gliding movements. This, together with the inward inclination of the femur and the greater liability for force to be applied to the outer side of the knee, has been held to account for the greater frequency of injuries to the inner meniscus, which exceed those of the outer in the proportion of 9 to 1.

**The Internal Semilunar Cartilage** is damaged by being crushed between the internal condyle of the femur and the underlying tibia, having immediately prior to this been drawn between them. The cartilage may be brought into this vulnerable position, either in movements of abduction, when the inner side of the joint is opened up

and the cartilage moves into the space thus formed, or in movements of forced internal rotation of the femur, when the mid-point of the cartilage is pulled backwards by its attachment to the internal lateral ligament and the anterior half is straightened across the front of the joint. It is doubtful if either displacement can reach the stage at which there is risk of injury if the attachments of the cartilage are normal; some of these attachments are either torn at the time or have been stretched by previous injury, disease, knock-knee deformity or habitual posture. In most cases the causative injury is a combined movement of abduction and internal rotation with the knee slightly flexed, as in a drive at golf or a sudden turning movement in tennis or cricket; the knee is straightened in the position of strain, and the cartilage is sheared between the articular surfaces. The cartilage injury may be a longitudinal split, in which the central portion is displaced towards the intercondylar space (bucket-handle type), there may be separation of the peripheral attachment, or there may be tears of the central free margin or of the anterior or posterior horn. The more the knee is flexed at the time of the injury, the more posterior is the lesion in the cartilage.

The patient is usually adult. During a sudden turning movement he feels a sharp pain on the inner side of the knee, which lets him down. On attempting to move he finds he cannot straighten the leg the last 30 degrees ("locking") and is usually unable to walk. The joint becomes rapidly distended with synovial fluid. After a variable period, and as the result either of manipulation or some sudden movement, the leg can again be extended fully; the freeing of the joint is as sudden as the locking, and is accompanied by the sensation of something "clicking" or "slipping into place" on its inner side. The effusion subsides and the knee recovers.

If a case is first seen with the knee locked the cartilage must be replaced by manipulation. The inner side of the joint is first opened up to free the cartilage by flexing the knee and abducting the tibia on the femur; in this position the tibia is internally rotated to its fullest extent, and the knee is then straightened. It is more usual for the case to be seen after spontaneous reduction of the locking, and the diagnosis depends in the main upon the history. Locking, however, is not an essential clinical feature, as several types of cartilage injury do not give rise to locking but do occasion attacks of giving way and insecurity of the joint. The points to which importance must be attached are that the movement causing the accident was one of abduction or internal rotation and of some violence, that locking was immediate and complete in a position of at least 30 degrees flexion, that subsequent freeing of the joint occurred suddenly, that synovial effusion followed the accident and that a tender point is found about midway between the patellar tendon and the internal lateral ligament, together with a slight localised fullness at this point.

The menisci are repaired after injury by connective tissue. If the attack is the first one, and locking has been reduced early and completely, it may be assumed that the torn portion has returned to its normal position. Tears of the avascular cartilage may not heal at

all or heal only very imperfectly, while peripheral attachment tears unite if the joint is immobilised for four to six weeks. Treatment consists in firm bandaging and quadriceps exercises to remove the synovial fluid and restore the tone of the muscles; a shoe, raised on the inner side, to facilitate repair of the coincident injury to the internal lateral ligament and prevent abduction strains; and the avoidance of all games involving sudden turning movements for three months. If these precautions are not carried out, or if the injury is repeated before healing is complete, repair does not take place, or does so with some deformity of the cartilage. Repeated attacks are to be expected. These are brought on by strains similar to those causing the original injury, but occur more easily, sometimes appearing with any unguarded movement. In each there is typical locking, but the pain and synovial reaction become progressively less and reduction easier. While the disability is therefore less severe, it must be remembered that recurring derangements of this nature lead to traumatic arthritis in the joint. After a second attack the damaged cartilage should be excised.

**Injuries of the External Semilunar Cartilage** are less common. They are caused by strains of adduction, combined in most cases with rotation, and present a similar history and physical signs to those outlined above. The pain is usually localised less accurately, and may be referred to the back of the joint. The treatment is similar to that for lesions of the internal cartilage.

**Rupture of Cruciate Ligaments.**—The cruciate ligaments, either both or the anterior alone, may be torn (usually incompletely) by violent twisting movements, in which the internal lateral ligament is almost necessarily damaged at the same time. The immediate symptoms are those of a severe sprain, pain in the joint, effusion and inability to walk; later, when the pain becomes less and the fluid subsides, instability of the joint will be noticed. On examination the characteristic feature is an increase in the anteroposterior movements of the femur on the tibia. The knee should be flexed to a right angle to relax the lateral ligaments, and in this position the tibia is pulled forwards and pushed backwards; an increase of forward movement indicates injury to the anterior, and of backward movement to the posterior ligament. Some anteroposterior movement is possible in the normal knee, and the sound side should be examined for comparison. An X-ray may show that a flake of bone in front of the tibial spine has been torn from the tibia with the attachment of the anterior ligament.

When rupture of the anterior cruciate ligament is recognised at an early stage, the limb should be immobilised in a plaster case for three months with the knee flexed 30 degrees. Injury of the posterior cruciate ligament is immobilised in full extension with the head of the tibia pulled well forwards. Injury of both cruciate ligaments demands similar immobilisation, but in a position of semi-flexion. In an old case with excessive movement, useful function can usually be obtained by re-educating the muscles and providing a knee cage extending to a socket in the heel, which effectively prevents anteroposterior movements, while allowing those of flexion and extension. Operations for



reconstruction of the cruciate ligaments have been devised, but these seldom give more than temporary improvement. In some cases of rupture of the crucial ligaments the disability is surprisingly slight.

**Fracture of the Tibial Spine** *per se* does not occur but it may be avulsed together with tibial attachment of the anterior cruciate ligament. It may be possible to replace the separated fragment by manipulation, but if this is unsuccessful operative reduction is advisable. After satisfactory reduction the knee is immobilised in extension until union is sound, quadriceps exercise being practised assiduously.

**Loose Bodies in the Knee Joint.**—The discussion of loose bodies in the knee is included in this section because some of these are traumatic and others partly traumatic in origin, and because the symptoms they cause closely resemble those of a torn cartilage. One type of loose body is due to osteochondritis dessicans, usually involving the intercondylar aspect of the internal condyle of the femur. As a result of aseptic necrosis a portion of cartilage and underlying bone is separated and becomes loose in the joint. Other loose bodies are thickened synovial fringes or osteophytes, the result of osteo-arthritis, which have become detached by violence. Others again are due to a metaplasia of the synovial membrane into cartilage, plaques of flattened, oval or rounded shape being formed in the lining surface of the joint, which at first sessile, later are attached by a thin membrane, and eventually become free. Bodies of the last type may be very numerous. The characteristic symptoms of a loose body are repeated attacks of locking. These attacks differ from those found with a torn cartilage in that they recur more often, the position of locking and of pain varies, the reaction is less, and unlocking usually follows within a few moments. The patient is generally aware of the body and can locate its position. An X-ray will confirm the diagnosis and demonstrate the number of loose bodies present. The treatment is removal by operation. When the body is single and movable, it should be manipulated to the inner side of the suprapatellar pouch and removed through a small incision. If multiple bodies are present, or one is in an inaccessible position, wide exposure of the joint is necessary; this may be obtained by a long curved incision to the inner side of the patella.

### FRACTURES OF THE TIBIA

**Fractures of the Head of the Tibia** may be due to direct violence, to falls on the feet or to lateral strains. Fractures due to direct violence are comminuted and often compound, but there is no gross separation. Those due to falls on the feet are usually T-shaped, the shaft being driven between the tuberosities. Forced abduction or adduction tends to produce a fracture of the whole or part of one tuberosity, which is displaced downwards by the corresponding femoral condyle. The external tuberosity is much more frequently fractured, and it is essential to understand that this is only one item in the injury, an acute valgus strain with tearing of the internal lateral, and possibly the cruciate, ligaments. All the signs of fracture are usually present, and the knee is distended with blood and synovial fluid. The diagnosis is

clear, but accurate recognition of the degree of comminution and of the position of the fragments is impossible without X-ray.

An exact restoration of the articular surface, both as regards the position of the fragments and the relation of their plane to that of the shaft, is essential to subsequent good function. When there is no displacement, a plaster case, padded only round the knee and ankle, should be applied from the groin to the toes. The plaster must be worn for twelve weeks, quadriceps exercises being started from the first and walking being permitted after four weeks.

Displacements must be corrected by powerful traction and manipulation. A Kirschner wire is inserted through the os calcis and the limb extended on a Hawley table until the length and general alignment have been restored. Displaced fragments must then be manipulated into position by pressure of the hands or a compression clamp. Open operation is necessary only when there is much comminution with displacement. When exact replacement, checked by a second X-ray, has been attained, a plaster case should be applied and the wire withdrawn from the os calcis. In cases with considerable comminution or accompanied by fractures of the femur or the tibial shaft, continuous traction through a wire in the os calcis is sometimes preferable to the plaster case.

**Fractures of the Tubercle** are due to muscular violence, and are only seen before the epiphysis has joined. In boys around the age of puberty, partial separation of the tongue-like prolongation of the epiphysis which carries the tubercle is comparatively common, and has received the name of Osgood-Schlatter's disease. This partial separation is due to repeated jerks upon the patellar tendon, such as occur in school games, rather than one single strain. Swelling and tenderness are found over the tubercle, and pain is produced by contraction of the quadriceps and by kneeling. In mild cases it is sufficient to prohibit games until the symptoms have disappeared; in severe ones the quadriceps should be thrown out of action for six to eight weeks by plaster case.

In adolescents the tubercle may be completely detached by a single muscular jerk of the type that would produce fracture of the patella in an adult. The fragment should be fixed in place by a strong catgut suture.

**Detachment of the Upper Tibial Epiphysis** bearing the tubercle is a rare accident, usually due to indirect violence, and seen between the ages of 6 and 12 years. The epiphysis may be displaced forwards, laterally or backwards on the shaft; in the last case the process for the tubercle is necessarily fractured. Replacement can be carried out under anæsthesia without great difficulty. A long plaster case must be applied and retained for eight weeks, but it may be bisected after the third week for the practice of knee movements.

**FRACTURES OF THE SHAFT** of the tibia alone are usually caused by direct violence. The fracture is transverse or comminuted, and may involve the shaft at any level. In children such fractures are more frequently oblique or spiral. The fibula serves to maintain alignment, so that there is commonly little displacement beyond some angulation.



The general line of the leg should be restored to that of its fellow, a plaster case applied and worn for eight weeks. If the fracture line is at or above the middle of the shaft, the plaster must extend up to the thigh, the knee being slightly flexed. A walking iron, described under fractures of the tibia and fibula, may be added after the third week, or earlier if the injury is to the lower half of the bone.

### FRACTURES OF THE FIBULA

The shaft and upper end of the fibula may be broken by blows on the outer side of the leg. The line of fracture is usually transverse, and the separation negligible. Twisting forces applied to the foot may also break the fibula in a spiral manner at its weakest point, the upper third of the shaft.

The disability caused by a fracture of the shaft of the fibula is not great, and walking, though painful, is possible. The diagnosis is usually made by the discovery of bruising and persistent pain at one point in the line of the bone, and by tenderness at this point when the fibula is "sprung" by pressure at some distant point. In fractures near the neck the external popliteal nerve may be injured, either at the time by the agent causing the fracture or later by pressure of callus. There are no forces tending to cause displacement, and with rest, strapping, and, later, massage, complete recovery of function can be expected.



FIG. 497

A, anteroposterior, and B, lateral views of fractures of the tibia and fibula due to indirect violence.

### FRACTURES OF THE TIBIA AND FIBULA

Fractures of the tibia and fibula together, due both to direct and indirect violence, are among the commonest encountered in practice.

In fractures due to *direct violence* both bones are broken at the same level. When the violence is moderate the line of fracture is transverse, or comminuted to some extent. The displacement is variable and depends on the nature of the injury, but is not usually great; with comminution, however, there is a tendency for small portions of tibia to be detached and rotated between the main fragments. Many of these fractures are compound. In those due to

severe violence, such as motor accidents, the soft tissues are often greatly lacerated and soiled and the bones grossly comminuted.

Fractures due to *indirect violence* are usually the result of falls on the feet, or twisting strains while the leg is bearing full weight. The level of fracture is almost constant, each bone breaking at its weakest point, the tibia at the junction of the middle and lower thirds, the fibula in its upper half. The line of fracture in each is oblique or spiral, reaching its lowest level in front and to the inner side (Fig. 497, A and B). Since the force continues to act after fracture, there is usually displacement, the leg being shortened and the upper fragment overriding the lower on its inner side. Indirect fractures are often compound, the sharp part of the upper tibial fragment making a punctured hole in the skin.

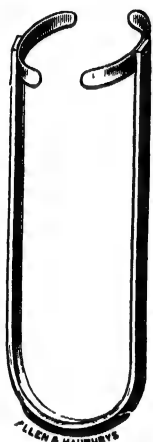


FIG. 498

A Böhrer's iron, which is incorporated in plaster of Paris to permit walking.  
(Allen & Hanburys.)

The *diagnosis* of fracture of the tibia and fibula is usually obvious, all the classical signs of fracture being present. Swelling, especially in simple fractures, may reach an extreme degree, even endangering the vascular supply of the limb.

*Treatment* will depend upon the nature of the fracture and the displacement. Fractures without displacement (usually transverse), which can be brought into good position by manipulation and are reasonably secure after reduction, should be immobilised in a plaster case applied directly to the skin. If there is much swelling, the limb may be kept on a back splint for a few days before the plaster is applied. Except in a few cases of fracture in the lower third where the danger of redisplacement is ruled out by the nature of the fracture, the plaster must extend to above the middle of the thigh with the knee in 5 degrees of flexion. The foot is held at a right angle and the plaster must support the plantar aspect of the toes.

Within a day or two a bar of iron, bent in the form of a U, is attached to the case in the line of the malleoli with additional plaster bandages, its lowest part projecting 1 in. or more below the sole of the case (Fig. 498). The patient is allowed to walk on this till the eighth to tenth week, when the plaster is removed. If union is then satisfactory, walking is allowed and exercises ordered to increase ankle movements; if there is weak union or none, a fresh walking plaster is applied.

In unstable fractures, such as oblique and spiral, traction is advisable for at least three weeks after reduction. The pin through the os calcis is incorporated in a below-the-knee plaster and the limb is supported on a Braun's splint. Ten to 15 lbs. weight are used, the foot of the bed being raised on blocks (Fig. 499). Later, when swelling has subsided and X-ray shows that the reduction is satisfactory, a skin-tight plaster from the toes to above mid-thigh is applied, the pin being left in until the plaster has firmly set.

Where there is considerable displacement, reposition can be obtained as a rule by screw traction on a Böhrer frame (Fig. 500) or Hawley

table. The knee is flexed, and in Böhler's method the traction is applied by a calcaneal clamp. When the position is good, two pins are inserted,

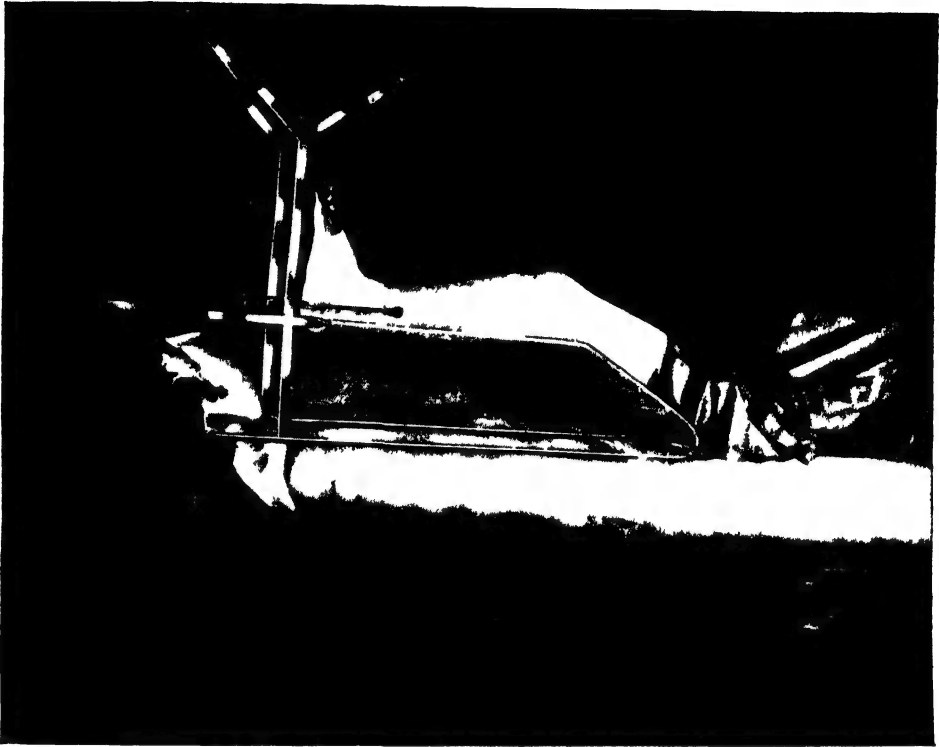


FIG. 499

#### Fracture of shaft of tibia

In unstable fractures and all fractures with severe swelling the transfixion pin is incorporated in the plaster and continuous traction is maintained for five or six weeks. (*Watson-Jones.*)

one through the upper and one through the lower end of the tibia, and these are incorporated in a plaster case, the calcaneal clamp being

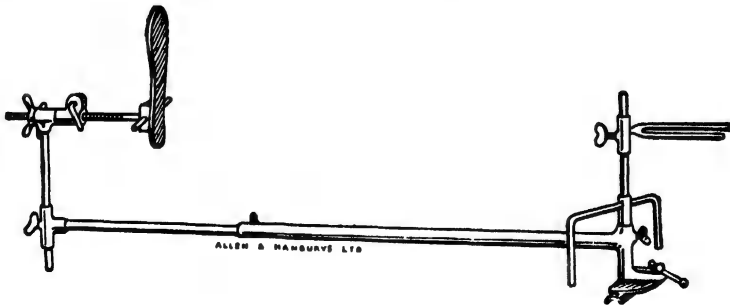


FIG. 500

Böhler's screw-traction frame. (*Allen & Hanbury's*)

removed when the plaster is dry. In four or five days Böhler applies a walking iron to the same case over the lower pin and the patient gets up. Alternatively, continuous traction may be used, a pin being

driven through the os calcis. This has the advantage of allowing cedema to subside before the application of a plaster which must be split longitudinally if put on in the presence of much swelling. The screw traction method is likely to give better reposition and Böhler advocates the inclusion of transfixion pins in any plaster that is used for walking in the first two or three weeks.

If satisfactory position cannot be obtained by these methods, usually because some muscle or small fragment of bone is interposed between the main ones, operation is in most cases advisable. The fragments are replaced and if they are then secure the limb is immobilised in plaster, which, after three weeks is replaced by a walking plaster case. If the fracture shows a tendency to redisplacement, the fragments should be fixed with a bone plate.

### INJURIES INVOLVING THE REGION OF THE ANKLE JOINT

Fractures of the ankle joint are, almost without exception, the result of indirect violence applied to the foot. Such violence may take the form of strains of abduction or adduction, or of external or internal rotation; since a much greater part of the foot projects in front of the ankle than behind it, abduction is usually accompanied by external rotation, and adduction by internal rotation. In such strains the astragalus remains with the foot and is rarely injured, but forms the instrument by which the malleoli are fractured. Fractures due to pure lateral or rotary strains are described, but in the great majority of instances the two are combined. Abduction and external rotation account for most of these fractures; violence in the opposite direction is usually less severe, and causes a sprain rather than a fracture.

**Abduction Fractures**—POTT'S FRACTURE.—These fractures are produced by indirect violence, the force being a mixture of abduction, eversion and external rotation of the astragalus and foot.

This force falls first on the inner side of the ankle joint and results in either a torn internal lateral ligament or a fracture of the tip of the internal malleolus. This allows the astragalus to be forced out against the fibular malleolus. In the majority of cases the strong inferior tibio-fibular ligaments remain intact and act as a fulcrum around which the fibula is bent. If the force driving the external malleolus outwards is strong enough the shaft of the fibula gives way at a distance above the tibio-fibular ligaments approximately equal to the distance from those ligaments to the tip of the malleolus. This fracture is typically oblique, running from behind downwards and forwards. If the abduction element predominates over the eversion, the fracture line tends to approximate to the level of the tibio-fibular ligaments and is more transverse. Either of these types constitutes a "Pott's fracture."

Secondary forces conspire to produce the more severe forms of ankle fractures. Anterior or posterior shift accompanying the main twisting force may lead to the so-called "marginal fractures" of the lower cup-shaped articular surface of the tibia, and these, together

with disruption of the malleoli, destroy the mortice joint of the ankle, allowing the astragalus and foot to be pulled backwards by the attachment of the tendo achillis to the os calcis. In the worst type a combined upward force leads ultimately to rupture of the inferior tibio-fibular ligaments and the displacement upwards between the tibia and fibula of the astragalus. This is a Dupuytren's fracture-dislocation (Fig. 501) of the ankle. Many of the more severe types of abduction fracture are comminuted.

The *clinical picture* is usually obvious. A typical history of injury, the usual signs of fracture, marked and rapid swelling and discoloration and a characteristic deformity—the foot being abducted and everted, plantar-flexed and drawn backwards—make diagnosis easy. But an X-ray in two planes should always be taken both before and after reduction.

By far the majority of cases can be *treated* by manipulation, reduction and fixation in plaster. If the case is seen so long after the fracture that swelling is excessive, temporary fixation on a back splint with right-angled foot-piece, together with elevation of the whole leg combined with frequent massage, should be carried out until such time as it is deemed possible to apply a skin plaster.

The essence of reduction is good general anæsthesia and a well-relaxed tendo achillis. This is best achieved by having the patient prone on the table with the thigh hanging vertically over the end of the table, and the leg, flexed at right angles at the knee, lying horizontally on a stool, the foot pointing downwards over the end of the stool. This position will result, in most cases, in the automatic reduction of the backward displacement, if not, only slight pressure on the heel completes it. The astragalus is easily pushed inwards so as to come into closest apposition with the outer aspect of the internal malleolus. With the patient lying in this position the fracture does not tend to redisplace and a posterior plaster slab is applied from just below the knee, round the heel to just beyond the toes. The plaster case is then completed from the tuberosities of the tibia to the toes (Fig. 502). This method ensures the easy reduction of both the backward and outward displacements and can be carried out single-handed. The foot should be at right angles, but can be dorsiflexed further if necessary. An immediate X-ray should show that the upper surface of the astragalus is horizontal, that the inner aspect of the astragalus is "well home" into the outer aspect of the internal malleolus and, in the lateral view,

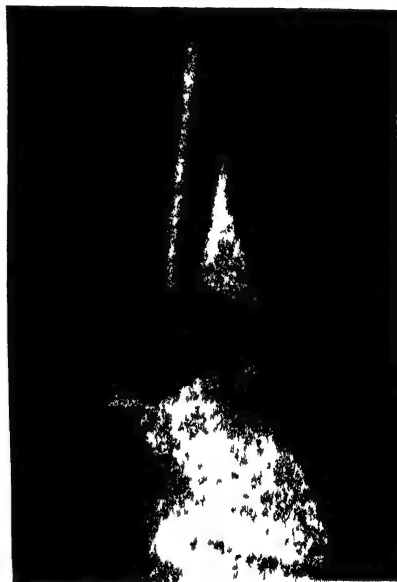


FIG. 501

A fracture-dislocation of the ankle joint, i.e., a Dupuytren's fracture.

that the astragalus is articulating accurately with the forward main portion of the tibia. A Böhler's walking iron is applied and the patient allowed to get about. If the swelling at the ankle joint has been at all marked, the patient should be seen in a day or two after the reduction, as the plaster may have become so loose as to require reapplication. This ambulant treatment is continued for from six to ten weeks according to the severity of the fracture and the success of reduction. When the plaster is removed, the foot, ankle and leg should be firmly strapped with elastoplast for a further two to three weeks, and a wedge on the inner side of the heel of the shoe may help considerably. A



FIG. 502

After manipulative reduction an assistant holds the foot in dorsiflexion while plaster is rapidly applied (Fig. 502, A). During this stage, or while the plaster is setting, backward dislocation may recur. The foot must be pulled strongly forwards and inwards by the surgeon's hands, dorsiflexed with his knee, and held while the plaster sets (Fig. 502, B). (*Watson-Jones.*)

well-treated case of abduction fracture should be walking normally within three months from the date of injury.

In those few cases where manipulative reduction is unsatisfactory, or calcis skeletal traction with the knee flexed, or open operation and mechanical fixation of the fragments, may be necessary. In cases seen late, when the deformity is uncorrected, or the astragalus remains tilted or has not been forced sufficiently inwards or forwards, considerable pain and disability are going to result. Further attempts at reduction can be made up to six to eight weeks after injury. If these are unsuccessful it will probably be necessary to arthrodese the ankle joint in 10 degrees of plantar flexion.

**Adduction Fracture of the Ankle (WAGSTAFFE'S FRACTURE)—**  
**First Degree.**—The external malleolus is fractured transversely at the

joint level, and the internal malleolus obliquely at its base, the line of fracture passing upwards and inwards. There is no displacement.

*Second Degree.*—The foot and astragalus are displaced inwards, the fibular fracture is the same as in that of the first degree, but the tibial fracture usually starts in the lower articular facet and passes upwards and inwards, a triangular piece, including the malleolus and part of the lower end, being detached and displaced upwards.

Adduction fractures should be reduced under general or local anaesthesia by methods similar to those employed for Pott's fracture. The plaster case should be applied with the foot dorsiflexed to 90 degrees and midway between inversion and eversion.

**Separation of the Lower Tibial Epiphysis.**—Separation of this epiphysis is caused in children and adolescents by violence similar to that which produces abduction or adduction fractures in adults. Similar displacements are found, but since the injury is at the epiphyseal line, the displaced fragment of the tibia includes the articular surface as well as the malleolus. The fibular epiphysis may be separated, or the bone fractured above the epiphyseal line. The treatment is that of ankle fractures in adults. Premature synostosis of the lower tibial epiphysis may result from this injury and give rise to increasing varus deformity at the ankle because of the continued growth at the lower fibular epiphysial line. This must be excised to stop its growth and correct the deformity.

**Fracture of the Astragalus.**—The astragalus is rarely injured, but it may be broken by falls from a height in the standing position. If the foot is at right angles at the time of impact, the body of the bone is crushed, the fracture being comminuted, and the bone flattened from above downwards and broadened. If the foot is dorsiflexed a transverse fracture of the neck is more common. The head is displaced upwards and the calcaneo-cuboid joint is usually dislocated at the same time.

Fractures of the astragalus are accompanied by great pain and disability. There is much swelling and bruising round the ankle, but the malleoli and os calcis can be felt in their normal relationship. The diagnosis is therefore made by exclusion and must be confirmed by X-rays.

Fractures without obvious displacement should be immobilised in an unpadded plaster case with the foot at right angles. A walking iron can be added in a day or two, but weight-bearing should not be allowed for at least two months.

Fractures of the body with displacement are restored to approximately correct shape by traction and manipulation. A Kirschner wire is passed through the os calcis, and screw traction applied on a Böhler's frame. When shortening has been overcome, the sides of the bone are compressed by a screw clamp padded with felt until its diameter is that of the uninjured astragalus. An unpadded plaster case is applied and the limb transferred to a Thomas' splint, when a weight of 10 lbs. is attached to the Kirschner wire. After four weeks the wire is withdrawn and a fresh walking plaster applied. Weight-bearing is allowed after twelve weeks.

Fractures of the neck, which are usually associated with dislocation of the subastragaloid joint, are replaced under anæsthesia by traction and forced plantar flexion of the foot. A plaster splint is applied in the plantar-flexed position. After eight weeks a fresh walking plaster is made with the foot at right angles. This plaster is discarded after a month, but an arch support should be worn for three months.

**Sprains of the Ankle Joint.**—Sprained ankle is a very common accident. It implies rupture of part of the internal or external ligaments of the ankle joint, by an outward or inward twist of the foot. The only common sprain is that caused by the foot being turned inwards, a force of adduction and internal rotation, which throws a maximum strain upon the anterior fasciculus of the external lateral ligament. Sudden pain is felt just in front of the external malleolus, with a sensation of something tearing. In severe sprains a snap may be heard. The ankle swells rapidly and bruising appears on the outer side. Upon examination it is found that the malleolar measurements are normal, there is no crepitus, the bruising is in front of, rather than over, the external malleolus and an area of tenderness is discovered in the hollow immediately in front of this point. X-ray examination is always necessary to exclude fracture of the malleoli or of the base of the fifth metatarsal.

The foot should be bandaged firmly and rested completely for forty-eight hours to prevent further effusion. A firm strapping of elastoplast is then applied and the patient allowed to walk in a shoe, the sole of which has been raised  $\frac{1}{4}$  in. on the outer side to prevent inversion. After ten days the strapping may be left off and massage ordered, but the raised shoe should be worn, in all except slight cases, for six weeks. If after three months the ankle is still painful and swells after exercise, and there is limitation of inversion and plantar flexion, it should be manipulated under an anæsthetic, special attention being directed to regaining the normal anteroposterior movement between the astragalus and tibia.

**Dislocation of the Ankle.**—This is an uncommon injury due to severe violence; only forward and backward dislocations are possible without fracture, and of these the backward is the less rare. The deformity is usually obvious, the foot appears short and the heel is more prominent than its fellow. A third degree abduction fracture is excluded by the absence of crepitus and the normal position of the two malleoli.

Reduction is easily accomplished, but an anæsthetic is usually advisable. A walking plaster should be applied with the foot at right angles and retained for eight weeks. If swelling persists after removal of the plaster the ankle should be strapped with elastoplast.

## FRACTURES OF THE TARSUS, METATARSUS AND PHALANGES

**Fractures of the Os Calcis.**—Two types of fracture are seen in the os calcis, compression fractures due to falls on the feet in the standing position and fractures of the tuberosity. The former are much the commoner.



**COMPRESSION FRACTURES** are caused by falls from a height on to the feet, or by jumping from a moving vehicle, the bone being crushed against the ground by the weight of the body transmitted through the astragalus. The resulting fracture is comminuted in an irregular manner. The os calcis as a whole is flattened and broadened, the front portion compressed from above downwards and expanded in the transverse plane, the back portion drawn up. The patient complains of great pain and is unable to walk. The heel is bruised and swollen, the hollows round the tendo achillis being obliterated. On examination crepitus

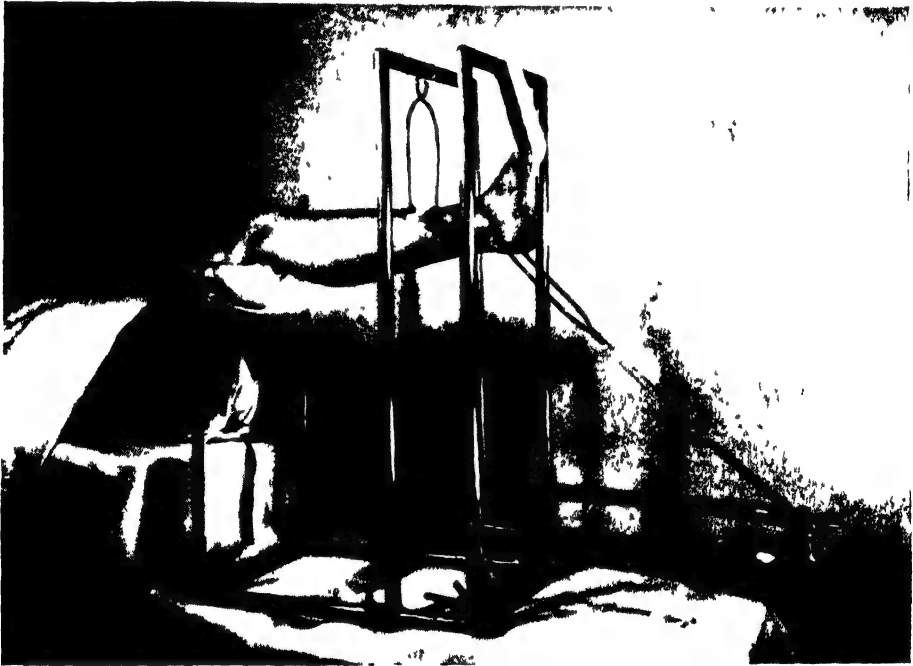


FIG. 503

**Fracture of the os calcis.**

A wire through the shaft of the tibia steadies the leg. A second wire through the os calcis enables traction to be made backwards and downwards in the long axis of the tuberosity of the os calcis. In this position a plaster is applied. (*Watson-Jones*)

may be found; lateral movements of the heel are resented, but gentle movements of the ankle joint can be performed.

Fractures of the os calcis with little or no displacement should be treated in a walking plaster for six weeks. Where there is deformity reduction should be attempted at the earliest opportunity by Böhler's method. A wire is inserted through the tibia in its lower fourth, and a second wire in front of the point of insertion of the tendo achillis into the os calcis. The leg is suspended by the first wire in a Böhler frame, and traction is made on the second at 45 degrees to the line of the leg, until flattening and upward displacement have been overcome (Fig. 503). The bone is then compressed laterally by a screw clamp of special design until its width is similar to that of the opposite side. An unpadded plaster case is now applied with the foot at right angles

and split along the dorsum. The limb is transferred to a Braun's splint and a weight of 10 lbs. attached to the wire through the os calcis pulling downwards and backwards. After three months the wire is removed and a walking plaster applied. Weight-bearing should not be allowed for a further six weeks. These fractures, even when efficiently treated, may leave a permanent disability owing to traumatic osteo-arthritis of the subastragaloid joint. Cases of very persistent pain can often be relieved by arthrodesis of this joint.

**FRACTURES OF THE TUBEROSITY** are due to direct violence rather than muscular pull. A portion of the tuberosity, varying from a flake to a large triangular fragment, is displaced upwards. In young patients the epiphysis may be separated.

When there is no great displacement, a plaster case may be applied in the position of slight plantar flexion and retained for six weeks. Walking may then be allowed in a shoe with a raised heel. Even if there is marked displacement it is not difficult to reduce the fragment by manipulation.

The remaining bones of the foot are usually injured by direct violence, such as crushing or the impact of heavy weights. The fracture may involve the cuboid, scaphoid, cuneiforms or metatarsals, and often implicates more than one bone. Pain, bruising and disability are always present, but deformity and crepitus are seen only in the more severe fractures. While the diagnosis is usually clear, the bones affected and the degree of displacement can be established only by X-rays.

The foot should be moulded back to correct shape under anaesthesia, care being taken to restore the longitudinal and transverse arches, and enclosed in an unpadded walking plaster. Weight-bearing may be allowed after eight weeks in a shoe fitted with an arch support.

**Fractures of Single Metatarsals.**—The shafts of the 2nd, 3rd and 4th metatarsals and the base of the 5th may be broken by minor degrees of violence, such as walking over rough ground in heavy boots (march fracture or *piéd forcé*). Several of the metatarsals may be involved. The fracture is transverse or oblique and the separation is negligible. Persistent pain and swelling over one metatarsal will suggest the diagnosis of fracture, which must be confirmed by X-rays. The fracture is sometimes such that it may be missed in X-ray examination, and only the subsequent development of a collar of callus reveals the site of the fracture.

If there is no displacement and the fracture involves the 2nd, 3rd or 4th metatarsal shaft, strapping the metatarsals together firmly with elastoplast is sufficient. If the 1st or 5th metatarsals are fractured or if there is any displacement in fractures of the other metatarsals, a moulded walking plaster to include the calf must be applied for four weeks, after which it can be replaced by strapping. Extension will be necessary in fractures of the necks of the metatarsals where marked displacement occurs.

**Fractures of the Phalanges.**—Any of the phalanges, but especially the terminal phalanx of the great toe, may be broken by the fall of weights or by stumbling against hard objects. In most cases there is no gross displacement. If there is deformity the alignment should

be corrected and the toe strapped for three weeks to a padded strip splint of aluminium; otherwise the foot should be rested until bruising has disappeared.

## INJURIES OF THE SPINE

Injuries of the spine may be caused by indirect or direct violence, the latter being responsible for a small proportion only and these the less serious.

**Fractures of the Vertebral Bodies.**—The vertebral bodies are fractured by forcible flexion of the spine beyond its normal limits,



FIG. 504

A compression fracture of a lumbar vertebra without dislocation



FIG. 505

Kummell's disease

so that the anterior parts of the bodies are compressed. The thin shell of compact bone is first broken in front, but when it has yielded the body offers little resistance to further compression and becomes split by radiating fissures, the direction of which is chiefly transverse. If the force continues the bone becomes compressed in the long axis (the deformation being most marked in front) and expanded in the lateral and to a less extent in the anteroposterior planes. The neural arches are first held together by interspinous and articular ligaments, but with further flexion these rupture and the laminae are torn apart. The portion of the spine above the fracture is then displaced forwards on the lower part. Fracture of the bodies usually involves one vertebra only, but two or three adjacent ones may be broken. The last two dorsal and first lumbar vertebrae are those most commonly injured; next in frequency come the upper dorsal vertebrae. In the cervical region the bodies are shallow and the intervertebral discs wide and

resilient, so that a force of flexion sufficient to injure the column produces separation of the neural arches before fracture of the bodies. Extension injuries of the spine are rare.

**Compression Fracture without Dislocation** is fairly common. The injury may be the result of a fall or some industrial accident, and is sometimes seen after violence of quite moderate severity, such as sudden stopping of a vehicle or missing a step. Often there is no deformity and the fracture may be unsuspected at the time. After the accident the patient complains of pain, sharply localised to one part of the spine, usually the lower dorsal region, and holds his back stiffly. Pain is increased by jolting and by movements of the trunk. On percussing the spinous processes one is found to be tender, and jarring the head produces pain at the same level. In other cases the spinous process of the affected vertebra may form a visible prominence.

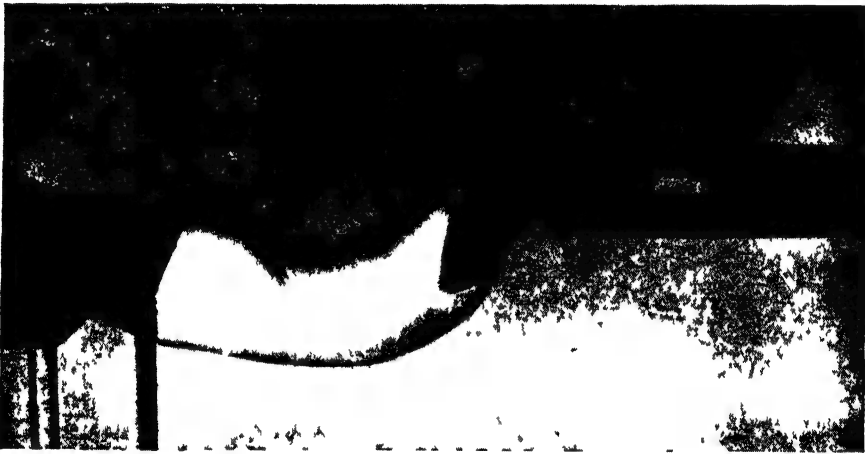


FIG. 506

Correct position for postural reduction. The lower table extends to the upper thighs; the upper table is clear of the chest. (Watson-Jones)

Girdle pains are sometimes produced by irritation of the spinal nerves and rarely there may be symptoms of pressure on the anterior columns of the cord, due to a bony fragment projecting into the vertebral canal. An X-ray should be taken in every case of injury to the spine. It is extremely important that even slight fractures should be recognised early, and this requires very good films. The anteroposterior view will give little help in the diagnosis of a fissured fracture without displacement. A good lateral film (Fig. 504) or a stereoscopic pair must always be obtained.

If such fractures are unrecognised and untreated, the vertebra, even when it is not deformed by the accident, will slowly yield to the force of gravity and become compressed in its long axis. The final result is a wedge-shaped vertebra, a condition which used to be called **Kummell's disease** (Fig. 505) before its traumatic origin was recognised. Weakness and inability to carry out strenuous work, persistent pain at the site of fracture and, later, osteo-arthritic changes and irritation of the spinal roots by osteophytes are the outcome.

Fracture of a vertebral body should be treated by correction and immobilisation at the earliest opportunity. The deformity has been produced by forced flexion; extension, if applied within a week of the accident, will restore the length of the vertebra and mould the fragments into approximately normal position. The patient is given  $\frac{1}{4}$  gr. of morphia and placed prone between two tables, with the thighs resting on one and the arms on another—which latter should be 12 in. higher (Fig. 506). A vest is slipped over the trunk, and the sternum, pubis, iliac crests and spinous processes lightly padded with adhesive felt. When the spine is fully hyperextended, a close-fitting plaster jacket is applied, well-moulded round the pelvis and extending in front from the episternal notch to the pubis (Fig. 507). When set, the plaster is trimmed to free the axillæ and thighs. After ten days' recumbency the patient is allowed to walk in the jacket, which must be retained for four months, after which a spinal support should be worn for another three months. From the very beginning the patient is made to carry out



FIG. 507

Correctly applied plaster extending from the groins and symphysis pubis to the clavicles. The lumbar spine cannot be flexed. (Watson-Jones)

regularly while in plaster and latterly he can leave off the support for this purpose. The exercises should be of the extension type, and flexion of the spine should be avoided until late.

**Fracture - dislocation of the Spine** is caused by violent flexion of the trunk, such as may occur in motor accidents, or when a weight falls on the shoulders of a stooping labourer. The body of the vertebra is first fractured (Fig. 508) and the neural arches are then torn apart by rupture of the ligaments and dislocation or fracture of the articular processes. The upper part of the fractured vertebra is displaced forwards on the lower, and the cord is compressed between the lamina above and the posterior edge of the lower vertebral body. In rare instances the cord escapes injury, but some interruption of its function is almost inevitable. The patient is usually



FIG. 508

A fracture dislocation of the cervical spine.

profoundly shocked and in great pain. Sensation and movement in the lower limbs may be lost. The distribution of the paralysis will vary with the level of the fracture; this subject is considered in detail under injuries of the spinal cord (Chap. XLII). Upon examination bruising will be noticed at the site of injury, and the spinous process of the fractured vertebra forms an obvious projection. The sternum may be fractured transversely in its upper part, the manubrium overriding the body. *Great care should be taken in examining any patient who may have sustained a fracture of the spine in order to avoid increasing the displacement, and all such cases should be lifted and transported in the prone position.*

Paralysis may be due to compression of the cord only or to destruction of its tissues; in the first case recovery may ensue, in the second none is possible. A destructive lesion is the commoner, but since it is impossible to recognise the nature of the neural damage on clinical grounds, it should be assumed in every case that the cord is compressed only, and the pressure removed by correcting the deformity. This should be done at once in order to avert further damage to the spinal cord, but the patient is often too shocked to allow of much manipulation. Perhaps the best method is for the surgeon and assistant to apply traction and counter-traction on the head and feet respectively while pillows are wedged in under the deformity to hyperextend the spine. This is done on a firm bed on which the patient is left until shock has passed off sufficiently to allow a plaster bed to be made. A turning case is constructed as well, so that the position of the patient can be altered and attention paid to the skin of the back. Constant care is necessary to avoid bed-sores, infection of the bladder and contractures of the lower limbs.

**Dislocation of the Spine.**—Pure dislocations are seen only in the cervical region.

**UNILATERAL DISLOCATION** is caused by forced movements of lateral flexion of the neck combined with rotation. The lower articular process of one vertebra, usually the 3rd or 4th, slips over the upper one of the vertebra below on one side and lies in front of it, the articulation of the opposite side remaining normal. The neck is flexed and rotated to the opposite side to that of the injury. Pain is felt at the site of dislocation and all movements of the neck are restricted. On examination an irregularity will be felt in the line of the transverse processes and tenderness found at the level of injury. The body of the upper vertebra may be palpated as a projection in the posterior pharyngeal wall. The cord is not injured, but pain may be referred along the dorsal root of the cervical nerve emerging below the displaced articulation.

**BILATERAL DISLOCATION** is due to forced flexion of the neck. The body of the vertebra is usually fractured in addition, but this is not invariable. Even with fracture of the body, the cord in the cervical region not infrequently escapes damage.

The neck is bent forwards and held stiffly. Very little movement is possible, and in sitting or walking the patient thrusts the chin forwards and turns the eyes up. A break in the line of the transverse

and spinous processes will be felt, and tenderness is found in both situations.

Dislocations of the cervical spine should be reduced immediately under general anæsthesia. Steady traction is applied to the head, which is then pulled backwards in bilateral, and rotated towards the injured side in unilateral, dislocations. Reduction occurs with an audible snap. After reduction a well-moulded plaster collar should be applied over a padding of felt; this should support the jaw in front and the occiput behind, and below should take its bearing on the sternum and clavicle. The plaster should be retained for four months.

**INJURIES OF THE ATLAS AND AXIS.**—The only common injury of the upper cervical vertebræ is forward displacement of the atlas on the axis, due to accidents in which the head is wrenched violently forwards. The transverse ligament may be ruptured or the odontoid process fractured. In the first case the medulla is impaled on the odontoid process and death is instantaneous. In the second the medulla is protected by the transverse ligament, and there may be no neurological symptoms. The head is held stiffly and all its movements are painful. The spinous process of the axis is unduly prominent. Many of these injuries, however, escape recognition for some time and an X-ray is necessary to establish the diagnosis. The displacement should be reduced by traction under anæsthesia and a plaster collar applied as for other cervical dislocations.

**FRACTURES OF THE NEURAL ARCHES.**—Fractures of the spinous processes and laminae are occasioned by direct violence. The former are more commonly injured in the dorsal and the latter in the cervical regions. Pain, loss of movement, signs of local injury and occasionally crepitus suggest the diagnosis of fracture, which must be confirmed by X-rays. The cord is occasionally injured by a depressed fracture of a lamina, especially in the cervical region. When there are signs of cord injury, operation for removal of the pressure is imperative. In other cases a plaster jacket or collar should be made.

The **TRANSVERSE PROCESSES** are injured in the lumbar region by direct violence; the injury can be demonstrated only by X-rays. Rest in bed for four weeks, with the leg flexed over a pillow to relax the psoas, is sufficient treatment.

### **FRACTURES OF THE STERNUM**

The sternum may be broken by indirect violence accompanying fractures of the spine. The fracture is a transverse one at the level of the manubrio-sternal junction, and the upper fragment overrides the lower. The deformity disappears when that of the spine is corrected.

Fractures of the sternum due to direct violence are the result of severe accidents, such as motor and aeroplane smashes, or falls under the wheels of a vehicle. Fractures of the ribs and injury to the intrathoracic viscera commonly accompany the sternal injury. Such fractures involve the upper part of the bone and take the form of irregular fissures without gross separation. The patient is usually

shocked and in great pain ; dyspnoea and disordered action of the heart are also characteristic. The pain is increased by movement, and local tenderness is found over the site of fracture.

The sternum should be immobilised by crossed bands of adhesive plaster passing from the axillary line on one side to the clavicle on the other. The patient is nursed in the Fowler position, which favours diaphragmatic respiration and minimises the risks of pulmonary complications. After three weeks he may be allowed out of bed.

### FRACTURES OF THE RIBS

*Fractures due to direct violence* are caused by accidents similar to those responsible for fractures of the sternum, and are often accompanied by such a fracture, or one of the scapula. Their site and severity naturally vary with the nature of the force causing them. In many cases they are compound and accompanied by injury to the lungs, heart, diaphragm, liver or spleen. Surgical emphysema and intrathoracic hæmorrhage are common.

Fractures which are not compound or accompanied by internal injury should be treated by strapping, on the lines laid down for fractures due to indirect violence. Immediate operation is necessary in cases of compound fracture, to prevent sepsis and avoid the danger of open pneumothorax ; injuries of the diaphragm, liver or spleen must be treated at the same time. Surgical emphysema, though occasionally alarming, is usually reabsorbed. Pneumothorax will also, in most cases, disappear spontaneously ; if it is causing embarrassment to the heart or increasing in amount, the positive pressure in the pleura may be reduced by inserting a needle through an intercostal space. A hæmothorax should be aspirated as soon as shock has subsided. Otherwise fibrosis and other complications are likely to follow (cf. Chap. XXIV, p. 467).

*Fractures of the ribs due to indirect violence* are more commonly encountered in practice. They are caused by deformation of the thorax following blows, falls or crushing accidents. The ribs are broken at a point just in front of their angles. The middle ribs, the 4th to the 8th, are most commonly involved, and two or three are usually fractured together. The line of fracture is oblique or transverse and there is rarely any displacement. On this account an X-ray may fail to demonstrate the injury.

The patient complains of sudden pain at the point of fracture, and of the sensation of something snapping. The pain remains localised at the same spot and is increased by coughing or deep respiratory movements. There may be shock and cyanosis, but these are unusual. On examination, tenderness will be found at the point of fracture, and pain is produced at the same site by compressing the whole thorax or by pressing on the injured ribs at some point distant from the site of injury. These signs serve to distinguish a fracture from a bruise of the chest, in which tenderness is noticed only on pressure at the site of injury.

The fractured ribs should be immobilised by fixing the injured



side of the chest with bands of adhesive strapping. These bands should pass from the nipple line on the sound side in front to the scapular line on the same side behind, and in the vertical direction should extend from two ribs above to two below the injured ones. They should be applied in the position of full expiration. In the absence of any injury to the lungs the patient may be allowed to walk. Union occurs in from four to five weeks.

The **first rib** is occasionally fractured by forced depression of the shoulders, the clavicle causing the damage; injury to the nerve of Bell, with paralysis of the serratus magnus, may complicate such a fracture. A sling should be worn for three weeks.

A. E. PORRITT.

R. Y. PATON.

## CHAPTER XLVII

### DISEASES OF BONE

**G**ENERAL CONSIDERATIONS.—The skeleton is composed of bones which have two main functions; firstly, to provide a rigid frame and thus to protect certain organs of the body from injury, and secondly, to afford attachments for the muscles and their tendons. The bones of the skeleton differ, therefore, in their shape and size according to the functions they have to perform. Every bone is composed of two types of osseous tissue, the outer or compact bone and the inner or cancellous bone. *Compact bone* is composed of concentric plates or lamellæ which are arranged in relationship to the Haversian canals, these latter being occupied by small arterioles derived from the periosteal and medullary arteries. Since hard bone is incapable of expansion, when inflammation occurs in these canals, the vessels within are liable to be obliterated by pressure of the inflammatory products, and the blood supply is thus cut off, with consequent death of the area of bone affected. *Cancellous bone* is composed of tissue arranged in the form of trabeculæ, the direction and strength of which vary according to the strains and stresses to which the particular bone is subjected. Two types of trabeculæ are seen, pressure and traction, depending upon whether they are required to strengthen the bone against either body-weight or the pull of muscles. The spaces between these trabeculæ are filled with bone marrow in which run blood vessels and lymphatics. Inflammation of the cancellous tissue and medullary tissue or marrow differs from that in compact bone in that the blood vessels are not so readily occluded and thus necrosis is less likely to result. The amount of each type of osseous tissue varies in each individual bone. In the flat bones the outer compact layers form two plates, between which lies a small amount of cancellous tissue, the best examples being the bones which compose the vault of the skull. In the short bones, such as are found in the hands and feet, a thin layer of compact bone is found covering or enclosing a disproportionately large amount of cancellous tissue. The long bones are made up of a shaft, which is composed of a tube of compact bone known as the *diaphysis*, joined on in the child to two *epiphyses* by a disc of cartilage called the *epiphyseal cartilage*, at which level increase in length of the bone takes place. The shaft at either end close to the epiphyseal cartilage contains cancellous bone, which diminishes as the middle of the shaft is approached. The cancellous spaces are occupied by a fatty marrow which is continuous with that in the medullary canal. From the medulla extend processes of connective tissue serving to provide a scaffolding by means of which the blood vessels are carried into the Haversian canals. Covering all bones is a vascular fibrous sheath known as the *periosteum*, which varies in thickness according to the particular bone and the age of the patient. It can be easily separated from the underlying bone in children, but in adults it is more firmly attached. From this membrane pass blood vessels and lymphatics to enter the canals opening upon the surface of the compact bone. The *periosteum* is generally regarded now as being entirely a limiting membrane,

which has no power of forming bone itself; but this is a question upon which there is not universal agreement, as some investigators still maintain that the periosteum is osteogenic. This membrane is very firmly attached at the junction of the epiphysis to the diaphysis, a matter of considerable importance when any acute inflammatory condition develops in the bone. The *vascular supply* of a bone comes from two main sources :—

1. **The Nutrient Artery** or main blood supply which, passing through the compact bone via the nutrient canal, enters the medullary cavity where it proceeds to break up into two main branches which pass to either end of the bone, terminating in a plexus at the *metaphysis* (that part of the bone immediately on the diaphyseal side of the epiphyseal cartilage). Besides supplying the medulla and the inner surface of the shaft of the bone, branches are given off which enter the Haversian canals where they anastomose with small branches from the periosteal vessels.

2. **The Periosteal Vessels** supply the outer layers of the compact bone, their branches anastomosing with those from the nutrient artery in the Haversian canals. Besides these two main sources, the junction of the epiphysis and the metaphysis is supplied with blood from the *circulus vasculosus articuli*, which is formed by the various articular arteries supplying the neighbouring joint.

*Development of Bone* takes place either in (1) membrane or (2) cartilage. The growth of a bone developed from cartilage occurs in three directions. The shaft increases in length by growth at the metaphysis, the amount varying at different situations. Thus, in the lower limb an increase in length occurs principally on either side of the knee joint at the lower end of the femur and the upper end of the tibia, whilst in the upper limb increase in length occurs mainly at the shoulder, *i.e.*, the upper end of the humerus and the wrist joint, *i.e.*, the lower ends of the radius and ulna. Increase in breadth or thickness occurs by the deposition of new bone beneath the periosteum, although this membrane itself probably does not lay down any new bone. The density of a bone is increased by the deposition of new bone in the Haversian systems and the surrounding lamellæ. In bones which have been developed in membrane the power of regeneration, should such a bone be destroyed, does not exist, but in those which are developed from cartilage the possibilities of repair are considerable.

Certain developmental abnormalities occur in which one bone or any part of it may fail to develop. Nothing is known as to the factors which are responsible for such a failure. Abnormal growth may occur, producing gigantism, which is the consequence of an imbalance of secretions from certain of the ductless glands.

## INFLAMMATION OF BONE

All inflammation of bone, of whatever nature or however produced, is properly known as an *osteitis*. When the inflammation begins in or involves chiefly the cancellous or medullary tissue it is called an *osteomyelitis*. Should the infection be limited principally to the periosteum and the underlying cortical bone it is known as a *periostitis*, whilst an *epiphysitis* is an inflammation starting in, and in the early stages being confined entirely to, the epiphysis itself. Whilst these terms are in themselves useful for many purposes it is impossible, owing to the intimate vascular supply, to imagine an infection of bone

being limited to any one part without the others being involved in some way.

The *phenomena of inflammation* are essentially the same as those which develop in any other tissue, except that in bone these are modified by its rigid structure. Acute inflammation of bone produces engorgement, exudation of fluid, emigration into the surrounding tissue of white cells and finally stasis, but owing to the resistance which the rigid structure of the bone offers to their escape, these products of inflammation accumulate, as they are unable to drain away. The pressure rises very rapidly in the Haversian canals and the



FIG. 509

Long-standing acute osteomyelitis of the femur showing a large sequestrum, which has penetrated the knee joint. A strong involucrum has been formed.



FIG. 510

A flake sequestrum from the surface of the femur as the result of a subacute periostitis. This can only occur if the endosteal blood supply is unaffected.

cancellous tissue, thus obliterating the vessels, so that the blood supply to the area of bone concerned is cut off. As a direct result, death of a portion of the bone occurs and this is called *necrosis*. Its extent depends upon the degree of congestion in the bone; at first it is impossible to define the area which has died, but after some days the portion of necrosed bone becomes white in appearance and is gradually separated by granulation tissue from the bone which has survived the acute inflammatory process. This separation is achieved at the expense of the dead bone, which, when separated from the living, is known as a *sequestrum* (Figs. 509 and 510). Its presence may be suspected from the presence of a persistent sinus leading from the skin down to the bone. If such a sequestrum is allowed to remain for many months after separation from the healthy

bone, its surface is gradually eroded by granulation tissue and becomes rough instead of smooth. When an acute osteitis occurs the periosteum is separated from the bone, first by œdema and then by the formation of an abscess (Fig. 511). After this latter has been incised the space left is filled with granulation tissue, and a layer of new bone is laid down beneath the periosteum. This new bone, which thus forms an ensheathing layer, is called the *involucrum*, having been formed by osteoblasts stripped off with the periosteum (Fig. 512). It is irregular

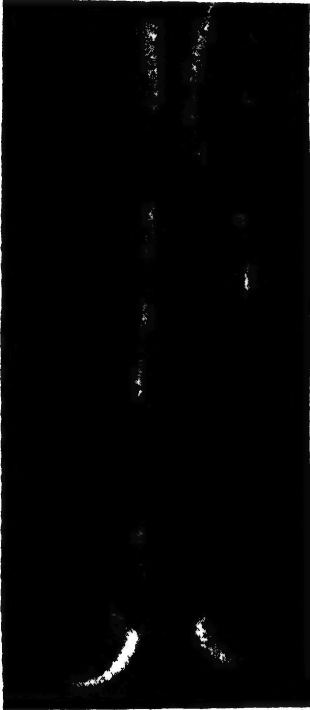


FIG. 511

The femur of a child showing the early stages of osteomyelitis and the wide stripping of the periosteum. The trephine hole was made for drainage, which proved inadequate and the child died.

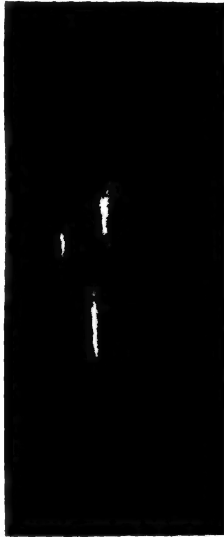


FIG. 512

An X-ray photograph showing a sequestrum of the tibia lying within the newly formed involucrum.



FIG. 513

An enormous exaggeration of the involucrum in an old, long-standing case of osteomyelitis of the tibia. At the upper end is a cloaca, through which a sequestrum can be seen.

in formation and may be very thick, depending upon the duration of the inflammatory process. Within it lies the sequestrum surrounded by granulation tissue, and perforating it are several openings or *cloacæ* (Fig. 513) which permit any discharge that may collect within the involucrum to escape to the exterior. When the sequestrum has become completely separated it can be extracted by removing part of the involucrum. These changes are well illustrated in Fig. 512.

When the destruction and absorption of bone keep pace with the inflammatory reaction, necrosis does not occur. Absorption of the hard bone permits the exuded serum to escape, so that the pressure around the vessels does not rise sufficiently to stop the circulation completely and thus bring about necrosis. The bone is destroyed

by the products of the inflammation and absorbed by osteoclasts at the same time, so that there is a microscopic destruction in contrast to a massive destruction or necrosis, this process being known as *caries* or rarefaction. It is seen best in a tuberculous infection, when the bone becomes fragmented and absorbed without the formation of a sequestrum. When caries is accompanied by actual pus formation it is known as *caries suppurativa*, whilst when it occurs without an abscess it is known as *caries sicca*, a condition which is typically and most commonly seen in a tuberculous osteitis of the upper end of the humerus in old people; when caries takes place in association with the formation of a well-defined sequestrum it is known as *caries necrotica*. This is sometimes seen in a pure tuberculous osteitis, but most often occurs if an acute pyogenic infection is superimposed upon a tuberculous lesion.

When the inflammation in a bone subsides the processes of repair begin, and new bone is laid down either to replace that which has been destroyed or to strengthen what has been left undamaged. In an acute pyogenic infection it has already been said that the involucrum, when first formed, is large in amount and soft in texture. If the inflammatory process becomes chronic, this new bone hardens and *sclerosis* is now said to have taken place. Sclerosis is best illustrated in an old syphilitic bone which may become as hard as ivory. In chronic tuberculous conditions of bone, sclerosis does not take place, and any new bone which is formed is small in amount.

### ACUTE OSTEOMYELITIS

*Etiology.*—In nearly every instance acute pyogenic infection of bone is the result of invasion by the *S. aureus*, although occasionally the *S. albus*, streptococcus or pneumococcus may be responsible. It is essentially a disease of children in the first decade of life, though it may occur later. In all such acute disease the infection is not confined to one particular portion of the bone, compact and cancellous tissue, medulla and periosteum alike being involved, but one part may bear the brunt of the infection. It is seen most commonly in children of the poorer members of the population, but as the result of the improved social conditions under which they live at the present day its incidence has dropped considerably, so that it is now not only an uncommon disease but also a less virulent one. The child who develops acute osteomyelitis is usually not in very good general health or else is recovering from one of the exanthemata. Trauma, whilst seldom the direct cause of the disease, is quite often a contributory factor by lowering the resistance of the bone to infection, producing thereby a disturbance of the circulation and thus enabling organisms to take hold and develop.

*Pathology.*—The infection, which is blood borne, enters the bone by the nutrient artery in nearly every instance and passes to the metaphysis, the source of the infection being a septic lesion of the skin or throat. In the metaphysis the organism produces an acute osteitis with abscess formation and destruction of cancellous tissue. The pus which is

formed at this site may spread in several directions (Fig. 514). The line of least resistance is into the medullary canal, along which the infection travels for some distance, though only exceptionally does the whole canal become affected. As the result of this acute inflammation, a certain amount of cortical bone may be destroyed and absorbed, creating thereby a channel by which the pus can escape from the medullary canal to form an abscess beneath the periosteum, which is thus lifted up from the underlying bone. In this way pus spreads some distance along the outer surface of the shaft. The epiphyseal cartilage itself is very resistant to infection and prevents direct spread of disease from the metaphysis into the cancellous tissue of the epiphysis, except under very exceptional circumstances. When the epiphyseal cartilage lies within the cavity of a joint (as in the hip) an acute infection of the metaphysis is likely to burst through and produce thereby an acute septic arthritis. If the disease starts in the epiphysis itself, which is rare, an acute epiphysitis results and the pathological process is exactly similar to that in any other situation, except that necrosis of the whole epiphysis, owing to its limited blood supply, is more likely and the joint is certain to become involved. Whilst any bone in the body may be the seat of an acute osteomyelitis, certain bones appear more liable than others. The lower end of the femur, upper end of the tibia and of the humerus are situations in which it is most often seen. When it occurs in such bones as the vertebrae or the ilium, the prognosis is more serious, as the disease is likely to spread widely in the cancellous tissue before its presence can be recognised, and treatment by surgical methods is more difficult.

*Clinical Signs.*—The disease in nearly every instance starts suddenly with an attack of severe pain in the limb, which is accompanied by a rise of temperature. The child very soon is obviously ill, having a flushed face, no desire for food and resenting any attempts to move or

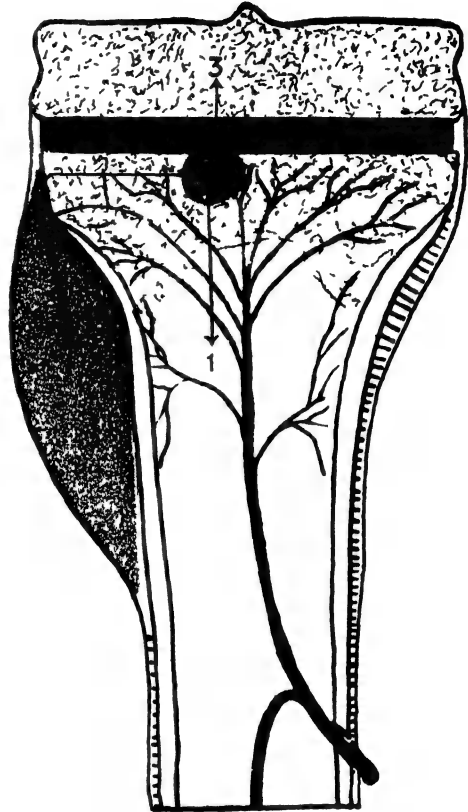


FIG. 514

**Acute osteomyelitis**

The blood supply of a bone is shown in red, viz., the nutrient artery and the many small periosteal vessels. A small abscess forms (green) beneath the epiphyseal cartilage (blue). 1, 2 and 3 indicate possible methods of spread. 2 shows a subperiosteal abscess which has destroyed the periosteal blood supply.

touch the limb. There may be rigors, and the temperature remains in the region of 102° to 104° F., accompanied by a rapid pulse. The tongue is dirty, and if the infection is very virulent other signs of toxæmia soon show themselves, the severity of which varies enormously. Beyond pain and tenderness in the limb there is often very little else in the way of physical signs and a diagnosis of acute rheumatism at this stage is liable to be made. When the infective process spreads into the medullary canal, the toxæmia is much more marked than it is when the disease remains more or less localised to the region of the metaphysis. A blood count at this stage will show a leucocytosis up to 25,000 or more and in about 50 per cent. of patients a blood culture will reveal the presence of the causative organism. If surgical measures are not adopted early, the signs of toxæmia become more marked and the temperature will tend to rise to higher levels. It may be possible to palpate an abscess over the end of the bone, and the soft tissues around it will be swollen and œdematous, whilst the neighbouring joint may develop a sympathetic effusion. Should the pus, which has collected under the periosteum, burst through this membrane, the tension for the time being will be relieved and the pain become less acute. The temperature will also fall, only to rise again within a few hours as the tension returns. At this stage of the disease an X-ray is of no value. Acute osteomyelitis in more than one bone at the same time is seldom seen, although it is quite common for the patient subsequently to develop subacute lesions in other bones during the period of convalescence. In the fulminating type of disease the whole shaft may be involved very rapidly, so that the diaphysis is filled with and surrounded by pus, which strips the periosteum completely off the bone.

The *diagnosis* of acute infective osteomyelitis may present many difficulties. It is often mistaken in the early stages for acute rheumatism, but in this disease it is usual for more than one joint to be involved and in an acute osteitis the disturbance of the patient's general health is more marked. An acute septic arthritis may give rise to difficulty, for the signs of the joint lesion may mask those in the underlying bone. This is especially so in acute arthritis of the hip joint combined with osteitis of the neck of the femur. Various acute illnesses, such as pneumonia, meningitis or any of the infectious fevers, may suggest some local disease of a bone during their early stages.

*Treatment.*—Surgical interference is called for as soon as the diagnosis has been made, for the sooner the tension within the bone is relieved the quicker will the signs of toxæmia subside and the less necrosis of bone will there be. The affected bone is exposed and the periosteum incised; if pus flows away freely it means that drainage is already established from the medullary cavity through the cortical bone, and for the moment nothing further need be done. Should no pus be present, but merely an œdematous condition of the periosteum, the metaphysis requires drilling with several holes to provide free drainage from the interior of the bone. Free opening by guttering it and scraping out the contents of the medullary canal is not desirable;



this removes both diseased and healthy bone, some of which may be quite capable of recovering from the effects of the inflammation, together with medullary tissue which is often normal. There is also the risk, if not the certainty, that when the bone is laid widely open healthy tissue will then become infected. The only surgical aim in acute osteomyelitis is to provide free drainage for the abscess inside the bone in the same way that an incision through the skin provides drainage for any other abscess. If, therefore, pus drains away freely when the periosteum is opened it shows that nature has already produced a channel from the infected area and that opening of the bone is not necessary. Removal of the whole diaphysis should be reserved for those patients in whom the condition does not show any signs of subsiding after the less drastic operation. When the bone has been drilled the wound is packed with gauze to prevent hæmorrhage and the limb then splinted. It does not matter what particular form of splint is employed, but in most instances plaster of Paris is the most convenient, provided it is applied over a sufficient quantity of wool to absorb the discharge which takes place. If the condition of the patient is satisfactory and the pulse and temperature are maintaining a lower level, this dressing should not be touched until the smell is no longer tolerable. With the help of deodorant bags this may be as long as four weeks. Sulphathiazole should be given in full doses for the first seven days. The plaster must be removed immediately if any of the complications explained on page 128 should occur. Should the neighbouring joint become infected it will require drainage and fixation in a Thomas's or some other suitable splint. Very occasionally it will be found necessary to perform an amputation to cut short the disease and save life, but in such grave cases the child is probably suffering from a septicæmia, when even amputation is a last resort and unlikely to save life. When all signs of acute infection have subsided, the limb will be left with one or more discharging sinuses which lead down through the involucrum to the portion of bone which has necrosed and is in the process of separating off as a sequestrum. This separation may take two or three months before it is complete, but as soon as it has occurred the sequestrum should be removed by cutting an opening through the involucrum large enough for the purpose. At the same time sufficient of the latter needs to be excised to enable the soft tissues to fall in and fill up the cavity; for unless this happens the cavity will continue to discharge in spite of the fact that the sequestrum is no longer there. Such a limb requires complete rest in the recumbent position after operation if it is to have any chance of healing.

The *prognosis* of acute osteomyelitis is always serious, but it is greatly improved if early surgical measures to release the tension within the bone are adopted, for thereby any toxæmia is likely to be lessened and the chances of a severe septicæmia developing are diminished. In the bones of the skull, vertebræ and ilium the prognosis both as regards life and ultimate healing is bad, for the infection is liable to spread, in spite of surgical measures, in the loose cancellous tissue which is present. Further, none of these bones possess any great power of response to inflammation by the formation of an involucrum,

and if a portion of one of them is destroyed seldom is any effort made to replace it. The prognosis has improved with sulphathiazole.

### ACUTE EPIPHYSITIS

Acute epiphysitis is a disease of infancy and is most commonly seen in the head of the femur, being always complicated by an associated acute arthritis of the hip joint. Whilst the prognosis is severe it is not hopeless, for by early and efficient drainage combined with fixation and extension the chances of recovery as regards life are good. The hip joint is permanently damaged and the epiphysis is either partially or completely destroyed. The acetabulum is also damaged, but to a less extent than the head of the femur. The stump of the neck becomes dislocated upwards and posteriorly upon the ilium, unless adequate measures are taken to prevent this, and these are not easy in an infant. The abscess which forms shows itself on the posterior aspect of the joint in most cases. The clinical signs are those of an arthritis, viz., pain, loss of function and general illness, the joint being held rigidly fixed by muscular spasm in flexion and adduction. The child in after life has a limp which varies according to the amount of destruction of bone, but has quite good movement in the false joint which has formed. There will be about an inch or an inch and a half of shortening.

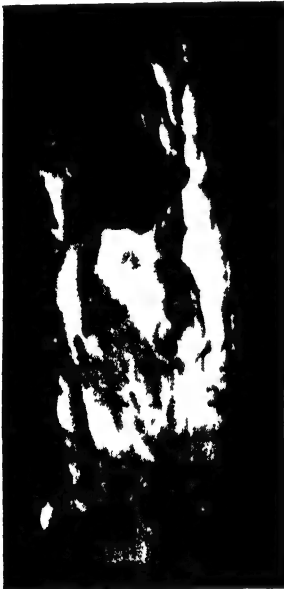


FIG. 515

A "ring sequestrum" removed from an amputation stump.

### ACUTE TRAUMATIC OSTEOMYELITIS

This condition which occurs as the result of a compound fracture or after an amputation (Fig. 515) is characterised by a septic fever and discharging wounds. In fractures the bone-ends must be got into proper alignment and fixed by traction, whilst at the same time adequate drainage provided, which will permit pus to drain away freely. Unless some such method of fixation is employed the sepsis cannot be controlled and the patient is likely to be worn out by constant pain.

In all types of acute infection of bone the patient is liable to develop a septicaemia or pyaemia, with the risk of other bones being involved.

### CHRONIC INFLAMMATION IN BONE

#### CHRONIC PYOGENIC INFECTION OF BONES

This may follow an acute attack of inflammation and continue for many years (Fig. 516). It also results from a bone infection which was never virulent enough to produce an acute abscess. In any chronic

osteomyelitis the amount of discharge will depend upon the size and number of the sequestra present and upon the extent of the cavity which contains them. The longer a chronic inflammation continues the thicker and more sclerosed does the involucrum become. The commonest form of chronic osteomyelitis which develops without any previous acute signs is a **Brodie's abscess** (Fig. 517). Described originally by Brodie as a tuberculous abscess of bone, it has since been proved in most instances not to be so, though quite often it is impossible to make a definite diagnosis except by bacteriological methods. It is generally caused by a *S. aureus* of low virulence which can be cultured in a pure strain from the pus, except in old-standing cases, when the cavity will be found to contain a clear yellowish coloured fluid which on culture is sterile. Absorption of bone has taken place without any active signs of inflammation, and around the cavity there is some sclerosis. Lining its wall there may be a thick pyogenic membrane, while in a very chronic lesion this lining resembles a serous membrane. Occasionally the cavity will contain a few small sequestra. No history of any

accident is usually obtained. In the course of time such a bone abscess, if the

infection is still active, gradually enlarges; when the contents become sterile the cavity ceases to increase in size but does not fill up with new bone formation. A Brodie's abscess develops in children, but is quite often not recognised until adolescent life. The site of the abscess, like all pyogenic infections of bone, is found in the metaphysis of the long bones, especially at the lower end of the tibia. A Brodie's abscess, by the time it is discovered, may be situated some distance from the metaphysis where it originally began as the result of growth having taken place at the epiphyseal line.

When a chronic osteomyelitis continues for any length of time the presence of this inflammatory process close to the epiphyseal cartilage induces a constant increase of blood supply to the affected bone and thereby produces

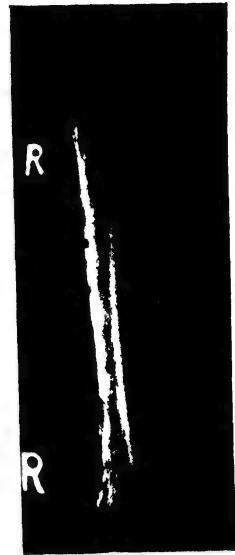


Fig. 516

Chronic osteomyelitis of the tibia involving the whole shaft. The raising of the periosteum and the formation of new bone are clearly shown.



Fig. 517

The lower end of the femur containing a Brodie's abscess. The sclerosis is well shown.

an increased growth in the bone, which becomes longer than the corresponding bone on the other side.

*Clinical History.*—Pain of a dull aching nature, intermittent in character, may be the patient's only complaint. On some days it may be entirely absent and on others, especially after use of the limb, it may become worse, but is never serious. Little may be thought of it at first, and its recurrence be the only reason for further investigation. Examination may reveal some tenderness over the affected bone with perhaps a little thickening, but unless the two limbs are very carefully compared it is quite easy to overlook the few signs present. If the condition becomes more acute the pain will increase and some œdema develop which will subside with rest, leaving a slight increase in thickening. X-ray examination reveals the true nature of the condition (Fig. 518).

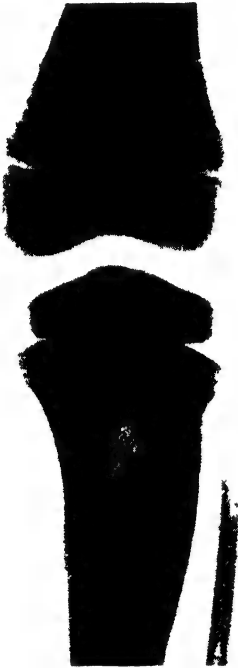


FIG. 518

An X-ray photograph of a Brodie's abscess in the upper end of the tibia.

*Diagnosis.*—In a case of chronic bone abscess it may be very difficult to arrive at a definite diagnosis by clinical examination. The X-ray picture will in most instances enable a correct diagnosis to be made. If the lesion is tuberculous no sclerosis will have taken place around the cavity, perhaps some minute carious sequestra may be seen, and a diffuse decalcification of the bone-end is generally present. If the infection is caused by a pyogenic organism the cavity is surrounded with a thin ring of denser bone, whilst if syphilitic the cavity will be encircled by dense bone and the whole circumference of the bone increased in size. A Wassermann test will assist in the diagnosis, but in a certain number the differentiation between a pyogenic and a tuberculous infection will not be possible until after the bone abscess has been opened.

*Treatment* of chronic osteomyelitis consists in a wide exploration of the affected bone with removal of any sequestra present, combined with a shelving of the sides of the cavity so as to render it shallow and thus enable it to heal up. Provided this is carried out thoroughly there are very few chronic cases which cannot be made to heal, although it may need several months care before this occurs. The only trouble is that if at a subsequent date the patient's general health is impaired or the limb injured, even years afterwards there is always a risk of a recrudescence of the inflammation. Should this happen drainage of the abscess with splintage until the incision has healed is all that is necessary. Treatment of a Brodie's abscess consists in opening the bone to allow the pus to escape. Its contents are cleared out, the sides of the bony cavity shelved off and the incision closed, the limb subsequently being fixed in plaster to maintain complete rest until it is soundly healed.

Many Brodie's abscesses heal by first intention with this treatment, and even if this desirable end is not achieved and a small sinus results, it will heal up provided the limb is rested.

### TYPHOID OSTEITIS

Infection of bone by the typhoid bacillus is a rare condition, which may occur during the period convalescence or not until many years after the acute attack of fever. The portion of bone affected lies just beneath the periosteum, a small abscess forms and sometimes slight destruction of the superficial layers of the compact bone may take place. The bone which is most often the seat of this form of osteitis is the tibia, the disease starting on its subcutaneous surface about the middle of its shaft. The ribs also may be the site of a typhoid osteitis. The onset of symptoms is insidious, beginning with pain and tenderness in the bone, with intervals in which there is a remission of these symptoms. After a time the pain becomes more persistent and œdema with redness of the overlying skin develops. X-ray examination may reveal a small cavity upon the surface of the bone just beneath the periosteum.

*Treatment.*—In some cases rest alone will suffice for the condition to settle down, but when an abscess has actually formed it requires incision to let the pus out, although a sinus is liable to form and may take many months to heal. In typhoid osteitis of a rib it is wiser to resect the diseased rib so that more rapid healing may be obtained.

### TUBERCULOUS DISEASE OF BONE

Infection of bone by the tubercle bacillus, which may take several forms, is always secondary to disease elsewhere, the primary focus being in most instances either in the mesenteric or bronchial glands in children and in the lungs in adults. The condition develops very slowly and by the time symptoms are complained of it has already been present some time. Tuberculous osteitis is, more often than not, associated with an arthritis of some nearby joint.

### TUBERCULOUS PERIOSTITIS

The disease starts in the bone just beneath the periosteum and not actually in this membrane itself. The process consists in the development of typical tubercles with formation of granulation tissue and caseous material. As a rule there is little destruction of bone, though an abscess with characteristic caseation is likely to form between the bone and the periosteum. This may burst through the periosteum and form a cold abscess which will in time involve the skin and lead to a sinus. The ribs, tibia, ulna (Fig. 519) and lower end of humerus are most commonly involved. Whilst such a condition may take several months to develop, sometimes in young children it may be a matter only of weeks, especially if there are multiple lesions. X-rays may reveal in the ribs a carious condition

of the bone, but in the tibia, ulna and humerus a good deal of new subperiosteal bone is often formed. The chief clinical sign is the appearance of a swelling which is tender on pressure. When in association with a rib the swelling in most cases rapidly increases in size and a cold abscess is formed.

The *diagnosis* may be very difficult in children, especially in the tibia, for an X-ray shows a periostitis very similar in many respects to that seen in syphilis. Clinically, the swelling may in the early stages feel firm and not unlike a sarcoma, but the X-ray will differentiate between a growth and tuberculous periostitis. If an abscess is present aspiration of the contents will confirm the diagnosis, as the tubercle

bacillus can always be found provided sufficient time and trouble are taken in looking for it; if further confirmation is needed, the pus may be injected into a guinea pig.

*Treatment.*—Beyond aiming at improving the general resistance of the patient and dealing with any abscess which may develop, there is little to be done except in disease of a rib. Here the cancellous bone very rapidly becomes infected, and treatment should aim at removing all the diseased bone by excising the affected portion of the rib. A sinus will persist for a little time, but the prognosis as regards recovery is good.



Fig. 519

Tuberculous periostitis of the ulna forming a small subperiosteal abscess.

### TUBERCULOUS OSTEITIS

This is the most common form of disease in bone produced by the tubercle bacillus and it develops in the cancellous tissue in either the metaphysis or the epiphysis itself.

*Pathology.*—The bacillus is deposited in most cases in the same soft new bone, at the metaphysis close to the epiphyseal cartilage, in which an acute infective osteitis commences. The pathology is in all respects similar to the development of a tuberculous lesion in any other tissue, and caries of the bone is produced by this process. The formation of sequestra is rare, but when they do occur they are small, soft and friable and lie in a mass of tuberculous caseous granulation tissue. Sclerosis of the surrounding bone is limited in degree, for the tuberculous process seems to inhibit the formation of bone rather than stimulate it, as do most other chronic infections. Occasionally a large sequestrum is formed lying in a mass of granulations and caseating material. This condition is known as *caries necrotica* and such a sequestrum must be removed before the lesion itself will heal. Whilst

a tuberculous osteitis may remain more or less encysted for a considerable length of time, it tends slowly to progress and by destroying the surrounding bone reaches the periosteum and forms an abscess, which strips this up from the bone. Owing to its close proximity to a joint there is always the liability of the epiphysis becoming involved, thus entailing the danger of infection spreading directly into the joint and producing a tuberculous arthritis. An acute tuberculous osteitis spreading rapidly along the shaft of a bone is occasionally seen. In young infants the short bones of the hands and feet are very liable to become the seat of this form of disease. The outstanding clinical sign of a tuberculous osteitis is similar to that of a Brodie's abscess, namely, a dull aching pain which is intermittent in character. Unless the diseased bone is superficial no local signs will be detected, for there is neither œdema nor redness of the skin and the condition may not become manifest until the adjacent joint shows signs of involvement.

When the phalanges or metacarpals are affected the condition is known as **tuberculous dactylitis**. A portion of the finger or toe becomes enlarged, fusiform in outline and painful. Within a short period the skin over the affected bone appears smooth and shiny and the pain more constant, whilst before long it becomes red and tender and an abscess forms. The disease commences in the cancellous tissue of the phalanx, producing a typical erosion or caries, and the compact bone then becomes involved. As this is destroyed, new bone is laid down beneath the periosteum making it appear that the bone has been expanded. In many patients this form of tuberculous osteitis involves more than one bone at the same time, and although it may settle down quietly, an abscess forms in the majority of cases. Interference with growth commonly occurs, leaving the affected finger shorter than the others.

The *prognosis* as regards this particular lesion is good. Occasionally it may become quiescent without any abscess developing, but even if this latter does form and burst it always heals up, when the underlying disease in the bone has resolved. Sometimes a fair-sized sequestrum is formed and may require removal. Whilst fixation upon a splint or in plaster is in theory desirable, in practice it is difficult to carry out on account of the size of the child's finger, and the condition does not appear to suffer from failure to immobilise the digit.

After the phalanges, the bones of the **tarsus** are most often the seat of a tuberculous osteitis, but here, owing to the proximity of the tarsal joints, such a focus of disease is more liable to produce serious consequences, in that these joints are almost certain to become infected. The scaphoid, or calcis or cuboid are the favourite sites of infection. Disease in these bones is always accompanied by an abscess, which may discharge through the skin to the exterior or into the joints of the tarsus. A sinus will develop and may continue to discharge for many months, but with infants treated under good conditions in the country such a track always heals eventually. The condition of *caries necrotica* is seen most often in the tarsal bones, and removal of the sequestrum which is formed becomes necessary before the lesion will heal. Under modern conditions such a tuberculous osteitis usually

does well, and in the child amputation should hardly ever be necessary, though in the adult this must, as in disease of the ankle joint, remain the method of choice.

### SYPHILITIC DISEASE OF BONE

Syphilitic disease of bone in all its forms is comparatively rare at the present day in consequence of the much improved methods of treatment, which have been employed in dealing with early syphilis during the last twenty-five years. These lesions vary somewhat in congenital and acquired syphilis.

#### ACQUIRED SYPHILIS

The disease may manifest itself in several ways : (1) osteoscopic pains, (2) periostitis, (3) diffuse osteitis and (4) gummata.

1. **Osteoscopic Pains** occur in secondary syphilis, especially in the bones of the lower limbs. They are of little importance and rapidly disappear as the patient comes under the influence of treatment. They are due to a transient subacute periostitis.

2. **Periostitis** occurs in the late secondary and early tertiary stages, in which a localised inflammation of the periosteum and bone beneath develops. There is an area of tenderness over the affected portion of the bone which becomes painful at night when the limb gets warm in bed. It is seen most commonly on the subcutaneous surface of the tibia and appears to develop in many instances after some minor injury. Such a periostitis usually clears up, leaving little permanent change, but sometimes in the process of repair a certain amount of new bone is laid down which becomes sclerosed, thus forming a *periosteal node* (Fig. 520).

In later tertiary syphilis periostitis, instead of being localised in this way to one small portion of the bone, may involve the whole shaft, the middle of which is affected more extensively than either end (Fig. 521).

3. **Diffuse Osteitis.**—This is the most common type of lesion in the tertiary stage. It may be associated with the formation of gummata and a considerable periosteal inflammation or may be a pure diffuse osteitis. The condition starts in the middle of the shaft and spreads to either end. The bone gradually becomes very much thickened and sclerosed and the medullary cavity obliterated. The tibia is the most frequent site, the bone becoming bowed anteriorly. In consequence of the sclerosis the vessels in the Haversian canals are constricted and this, **together** with the obliterative endarteritis which is present in all **tertiary** lesions, interferes with the blood supply to such an extent that necrosis of bone is liable to occur. Should a pyogenic infection subsequently be superadded, sequestra will form and may take a long time to separate. Such a diffuse osteitis occurs most commonly in children affected with congenital syphilis, when the typical "sabre tibia" is produced.

This diffuse osteitis, when it involves the nasal bones in infants, is



nearly always complicated by a secondary pyogenic infection, and the purulent discharge from the nose and the marked damage to bone is due to this as much as to the syphilitic osteitis. In the cranium a diffuse osteitis is often associated with a superficial gumma, which is likely to burst on the surface and become secondarily infected, after which necrosis occurs in the sclerosed bone.

**Gummata.**—Gummatous changes may show themselves in several ways :—

1. In association with a diffuse osteitis.
2. *Subperiosteal.*—These may be either single or multiple and they



FIG. 520

Syphilitic periosteal node at the upper end of the tibia, the rest of the bone being normal.

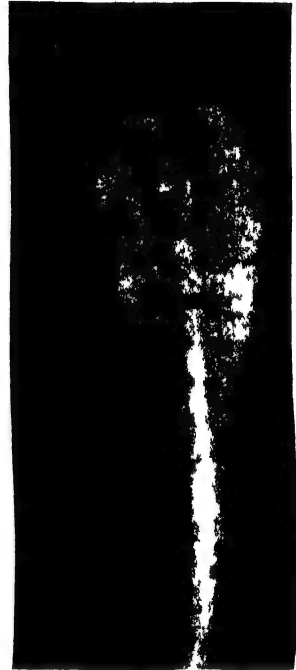


FIG. 521

Generalised periostitis of the ulna due to congenital syphilis in a young child

are seen most often in the sternum, the skull and the hard palate. When they burst and discharge their contents they are liable to become secondarily infected and extensive necrosis of bone will occur. Around this necrosed bone the sclerosis which takes place may prevent the sequestrum from separating and the ulcers may continue discharging for several years. In the skull the pericranium surrounding the necrotic area becomes gummatous and at the same time the subjacent bone undergoes sclerosis.

Periosteal gummata are also seen in the hard palate, where they reveal themselves as painless soft swellings, which after rupture leave an area of necrosing bone. After the sequestrum has separated there remains a perforation of the palate, which results in a direct communication between the nasal and buccal cavities.

3. *Central or Localised Gummata* may be seen in acquired syphilis, but are most common in children with congenital disease. This central gumma develops quite insidiously in children of about 10 to 12 years of age in the cancellous tissue of the metaphysis at the ends of long bones, especially the upper end of the tibia. A carious condition of the bone is produced resembling in many ways a tuberculous osteitis or a Brodie's abscess and, should it be accompanied by new bone formation beneath the periosteum, it may closely simulate an osteogenic sarcoma. The site at which such a gumma develops is close to the epiphyseal cartilage and the neighbouring joint is sometimes distended by a painless effusion.

*Clinically*, the swelling of the joint may be the only obvious sign, and the sole evidence of any osseous disease is that the patient complains of a dull ache in the end of the bone. Often no other sign of congenital syphilis is present. The absence of severe pain and of any characteristic findings in an X-ray should raise the suspicion of syphilis. The diagnosis from tuberculous osteitis and Brodie's abscess should be easy, as in the latter there is always a certain amount of surrounding sclerosis. An early sarcoma, before any periosteal bone has been formed, may give rise to difficulty in diagnosis. If the Wassermann reaction is negative the diagnosis can be decided only by watching the effect of antisypilitic treatment. Mercury, by mouth or in the form of inunction, combined with potassium iodide, will produce a most rapid alteration in the bone picture. In congenital syphilis other manifestations of the disease may be apparent, though this is not always so, the central gumma perhaps being the only syphilitic lesion.

#### CONGENITAL SYPHILIS

Various other bone lesions due to infection by the treponema pallidum which are peculiar to congenital syphilis are seen, though they are very rare at the present day.

1. **Periosteal.**—The formation of bone beneath the periosteum of the frontal and parietal bones occurs, producing thereby the so-called "Parrot's nodes," and these by their situation around the anterior fontanelle give rise to what is known as the "hot cross-bun" appearance of the skull. In the early stages this new bone formation is soft and, if treatment is given, will almost entirely disappear, but if left it becomes dense and sclerosed and remains as a persistent deformity.

2. **Craniotabes** consists in absorption of the bony tissue of the cranium, so that the bones are thinner. It occurs during the first six months of life.

3. A gummatus osteitis may develop in the phalanges, producing a **dactylitis** very similar in appearance to that seen in tuberculous osteitis of the fingers. The progress of the disease is often quite free from pain but, as often as not, it ends with a discharging sinus which may take some time to heal.

4. **Symmetrical Overgrowth** of the tibiæ occurs together with periosteal nodes. The length of the tibia is out of proportion to the

length of the femur. This condition was described by Clutton, who observed it in association with the painless synovitis of the knee joint which bears his name.

5. **Epiphysitis** or syphilitic osteochondritis was at one time a fairly common lesion in infants with congenital syphilis, but it is very rare at the present day. It consists in a gummatous infiltration of the epiphyseal cartilage with the formation of osteoid tissue, so that as a result of an obliterative endarteritis the vascular tissue in this situation becomes replaced with a yellowish material and later by granulation tissue. In consequence, a separation of the epiphysis, though it rarely happens, may take place. When the epiphysis does separate, the limb hangs useless in a condition known as syphilitic pseudo-paralysis. Under these circumstances secondary suppuration has been known to occur. This particular epiphysitis develops about the third month and it is characterised by an enlargement of the epiphysis which, in contrast to rickets, involves to some extent the diaphysis. It is nearly always symmetrical and, as in rickets, the wrists, knees and ankles are the sites most commonly involved.

In addition to the enlargements at the ends of bones there is tenderness and pain on attempted movement. An X-ray of the affected portion will show irregularity and widening of the epiphyseal line, but no characteristic feature. The diagnosis should usually be easy, as the condition develops in an infant at an earlier age than rickets and the Wassermann reaction is always positive. Scurvy is likely to occur in a child of about the same age, but the absence of hæmorrhage from the gums and in other situations should simplify the diagnosis.

*Treatment.*—All syphilitic lesions of bone require a thorough course of treatment such as would be given for any other manifestation of the disease. In a diffuse osteitis with periostitis these measures may fail to relieve the pain, and guttering the bone to lay open what remains of the medullary canal, thereby relieving the tension within the bone, will be necessary.

## DEFICIENCY DISEASES OF BONE

### RICKETS

**Rickets** is a nutritional disease met with in children, in whom it seldom develops before the age of 6 months or after the age of 18 months. It is seen in its worst forms in the slums of great cities, though it may develop in children brought up under the best of housing conditions. It is more likely to occur during a severe winter than in the spring or summer. It is produced by a variety of causes, the principal of which are the absence from the diet of an adequate amount of fats containing the fat soluble vitamin D, a deficiency of calcium salts and a lack of sunshine and fresh air. Breast feeding does not render the child completely free from the risk of rickets, but greatly reduces the incidence.

*Clinically.* the symptoms may be divided into two groups, the general or early, and the late or those affecting the osseous system.

**General.**—The onset is gradual. The child with rickets is fat and flabby in most instances, though if there has been much interference with its diet it may be thin and emaciated. It becomes irritable, cries more than is normal and is liable to attacks of gastro-intestinal disturbance evinced by vomiting and diarrhoea, the stools being green and slimy. Bronchitis is a frequent manifestation. Sweating about the head and throwing off the bed-clothes at night are characteristic. The abdomen is protuberant, the spleen sometimes enlarged and palpable. Any attempts to sit or walk are very much retarded, eruption of the teeth is delayed and, when they do appear, they are liable to early decay unless adequate treatment is given.

**Osseous Changes.**—The principal changes take place in the region of the epiphyseal cartilage and newly-formed bone in the metaphysis (Fig. 522). The epiphyseal cartilage becomes enlarged and irregular, whilst columns of cells are laid down in an irregular form and ossification around them takes place in a disorderly manner, so that masses of cartilage cells may be seen extending into the newly-formed bone, which is itself poorly calcified and mixed with areas of fibrous tissue. The number of blood vessels running through this area is increased. It is in consequence of these changes that the epiphyseal cartilage on section appears to be wide and irregular instead of a thin line with



FIG. 522

Femur and tibia from a case of rickets showing the characteristic changes described in the text.

a clean-cut edge in contact with the metaphysis, such as occurs in the normal bone. But in addition to these alterations in the formation of bone at the epiphyseal cartilage, the diaphysis becomes softened from the absence of a sufficient amount of calcium salts, and as the result of muscular action and weight-bearing the bones are liable to bend and develop deformities of various kinds. When the underlying cause is treated, ossification becomes normal and the bones harden up again. In certain bones attempts are made to strengthen the weakened areas by the formation of new subperiosteal buttresses and these, when the active disease has been cured, may remain as a permanent condition. When rickets has persisted for a prolonged period without proper treatment the bones appear to lose completely the power of proper development, and the child remains permanently dwarfed. Many mild deformities correct themselves and seldom at

the present day are any serious examples seen. The most characteristic clinical sign of rickets is an enlargement of the ends of the bones in the region of the epiphysis, and this can be seen most easily at the lower ends of the radius and ulna, in which situations there may be some tenderness. Changes in the thorax are visible in the enlargement of the costo-chondral junctions of the ribs, which produces what is called the *rickety rosary*. The softened ribs may be drawn in by the diaphragm, so that a groove on each side of the thorax is produced, known as *Harrison's sulcus*. At the same time the sternum appears more prominent, giving rise to the condition of *pigeon chest*. In the skull the vault may be enlarged owing to thickening



FIG. 523

Anteroposterior view of the bones of the lower extremity illustrating the X-ray appearances in rickets.

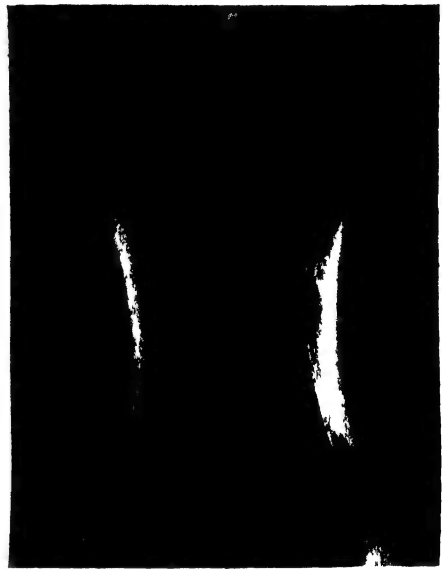


FIG. 524

An older child than that in Fig. 523 with more advanced changes.

of the frontal and parietal bones upon which bosses develop, the anterior fontanelle remaining open many months after the eighteenth, when it should in the normal way be closed. Deformities of the spine, kyphosis and scoliosis, seldom develop as the result of rickets, but in the pelvis a diminution of the anteroposterior diameter may in after life in women complicate pregnancy.

The tibia is the bone which exhibits deformities in rickets more commonly than any other (Figs. 523 and 524). There is an increase of the normal inward curve at the junction of the lower and middle thirds, and this may give rise to an appearance of genu varum or bow-legs. In the child, in whom active rickets has been allowed to persist for a long time without treatment, the tibia may also develop a marked anterior bowing in the same position, with the formation of a large posterior buttress devised to strengthen it. In the femur the normal anteroposterior curve may be increased and a knock-knee develop,

whilst at the upper end the neck may bend and a coxa vara result. Deformities of the upper limb are seldom seen except in children who have been neglected, when the bones will be bent partly by muscular action and partly by the weight of the body in crawling. When a fracture occurs in rickets the bone is liable to break across only a part of its circumference while the remainder bends. Such a condition is called a *greenstick fracture*.

*Treatment.*—The first essential is to cure the active disease by attention to the diet and general hygiene of the child. It requires plenty of fresh air and sunshine, whilst its diet needs the addition of cod-liver oil or vitoleum cream, which contain the vitamins necessary to cure the condition. The only certain evidence that active rickets has been cured is that afforded by an X-ray of the epiphyseal line. If any operative treatment for the correction of a deformity is contemplated, such an X-ray examination must always be carried out, for no forcible correction is permissible in the presence of active rickets on account of the risk of non-union occurring subsequently.

The mild deformities of the bones require no treatment for, when the active disease is cured, these harden up and gradually resume their normal shape. This is well illustrated by the fact that children with marked deformity of the tibia will, in the course of nine to twelve months, grow perfectly normal. There is, therefore, no need to restrict the child's activities by the application of splints to keep it off its feet. If it is necessary to correct a deformed tibia by operation, this can be carried out by osteoclasis, which consists in the production of a subperiosteal fracture of the bone by forcible manipulation, the limb being subsequently fixed in plaster in the corrected position until union has occurred.

Knock-knee or genu valgum, if of minor severity, is corrected automatically as the child grows when the active signs of rickets have ceased. In older children it is necessary to provide knock-knee irons which gradually correct the deformity, but if this is not accomplished by the age of 5 or 6 years it is then necessary to perform an osteotomy of the femur, the limb being subsequently splinted in the corrected position until union has taken place.

### ADOLESCENT RICKETS

This is a disease of which the etiology is at present unknown. The changes which occur in the bones are similar in many respects to those which are seen in rickets in an infant. It develops about the age of 12 to 15 years, and the patient is usually brought for advice on account of the development of some deformity, such as a genu valgum, which has appeared within a few months. The children who develop rickets at this age are mostly pale-faced, lethargic individuals with a history of repeated intestinal or pulmonary upsets in earlier life. The diagnosis between adolescent and renal rickets is often very difficult and must depend upon an estimation of the blood urea.

*Treatment* is very unsatisfactory. Fresh air and a good mixed diet with the addition of irradiated ergosterol appear to clear up the

condition, but it takes a long time before there is any noticeable improvement and a skiagram will often show irregularity of the epiphyseal line for a year or more. Cod-liver oil is very uncertain in its action and cannot be relied upon to cure the disease in the same way as it does in the infant. Any deformity, such as genu valgum, requires correction by an osteotomy, but the limb needs the support of a suitable splint for many months afterwards as the deformity has a tendency to recur.

### RENAL RICKETS

This disease is one in which changes occur in the skeleton with the production of deformities similar to those of ordinary rickets in a child who is suffering from chronic interstitial nephritis. It may develop at any age up to about 15 years. These children are pasty in colour, lethargic and have a very dry skin. A history of polyuria with nocturnal frequency is invariably obtained, and an examination of the urine reveals a low urea concentration, whilst in the blood there is a retention of urea up to as much as 150 to 200 mg. per 100 c.c. of blood. Seldom is any history of an acute attack of nephritis obtained and nothing is known as to the etiology of the renal disease. Enlargement of the epiphyses is the first sign and this is followed by the development of various deformities, of which the most common is genu valgum. As the disease progresses, separation of the epiphyses gradually occurs so that the child is unable to walk. These separations are seen most commonly at the lower ends of the radius, femur and tibia.

*Treatment* is of little avail, for the kidney disease slowly progresses until the patient dies of uræmia, although it may be many years before this takes place. The lower limbs can be straightened by gradual splintage, and then with light calipers the patient may be enabled to get about for a time. Open operations to cure any deformity are fraught with the risk of death from uræmia, brought on by the administration of an anæsthetic.

### OSTEOMALACIA

**Osteomalacia** is a very rare disease met with in pregnant women. There is a slow absorption of calcium from the bones, which become rarefied to such an extent that bending or spontaneous fractures may take place. The medulla is very vascular and hæmorrhages are liable to occur. As these are absorbed, clear cystic spaces are left scattered about in the medulla. The bones of the pelvis and the vertebræ are first involved, though in the later stages of the disease any part of the skeleton may show similar changes. In process of time the cortical bone becomes absorbed until it is represented only by a thin shell filled with hæmorrhagic areas alternating with clear spaces scattered amongst fibrous tissue. The condition progresses very slowly and may have intermissions, becoming progressively worse after each pregnancy. As the result of the softening the bones of the pelvis may sink inwards, producing thereby the triradiate pelvis which will prevent

any natural delivery. In time the limbs become bent and deformed so that the patient is unable to get about. No treatment has any influence upon the progress of the disease. Removal of the ovaries and abortion both fail to have any influence in checking the gradual softening and bending of the bones.

### SCURVY

**Scurvy** is a nutritional disease resulting from the absence from the diet of vitamin C, which is contained in fresh fruit and green

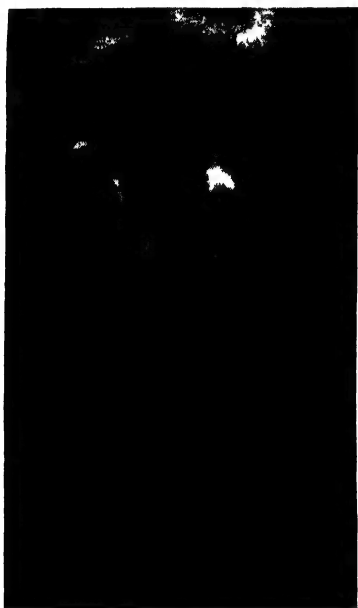


FIG. 525

Humerus with a spindle-shaped swelling, the result of a subperiosteal hæmorrhage in scurvy. This has caused a separation of the epiphysis from the head.



FIG. 526

The X-ray appearance of a subperiosteal hæmorrhage in scurvy. Note the fine line of ossification beneath the raised periosteum, and a slight anterior shift of the epiphysis.

vegetables. Its essential features are a general failure of health and hæmorrhages into various tissues, especially the gums. It is seen in the infants of well-to-do parents, who have been brought up on artificial foods without the addition to their diets of suitable fruit or vegetable juices. Owing to the vast improvement, which of recent years has taken place in the knowledge of the public as to how infants should be fed, it is rarely seen. In mild cases there may be a tendency for the child, which probably shows signs of rickets, to have spongy gums which bleed easily. When the disease has fully developed the child is irritable, the gums bleed and hæmorrhages from the bowel may occur. Complaint may be made of vague pains in the limbs. At the situations where pain is complained of, tender swellings develop close to the



epiphysis. Should hæmorrhage occur into the medullary canal, pain may be very severe. The swellings are subperiosteal hæmorrhages, which may be large in amount, stripping the periosteum from off the shaft for a considerable distance and even leading to a separation of the epiphysis from the diaphysis (Fig. 525). The diagnosis of infantile scurvy is generally very obvious, though it may be mistaken for hæmophilia. If the subperiosteal hæmatoma has become organised, the tumour which results is liable to lead to a diagnosis of osteogenic sarcoma, though this can always be disproved by an X-ray examination, which in scurvy will show a thin layer of bone beneath the periosteum which has been separated from the main bone (Fig. 526). In the days of sailing-ships and among troops in time of war scurvy was quite commonly seen, and hæmorrhages both intramedullary and subperiosteal were frequent.

*Treatment.*—This disease responds to treatment as soon as fresh fruit and vegetables are added to the diet, and it can always be prevented by the routine addition of orange juice in feeding an infant. Any subperiosteal hæmorrhages which occur are absorbed without permanent ill effects, though the limb requires rest by fixation on a splint during the painful stage.

## DISEASES OF UNKNOWN CAUSATION

### ACHONDROPLASIA

**Achondroplasia** is a not uncommon congenital disease of bone which, whilst not always hereditary, may occur in more than one member of a family and also in succeeding generations. Nothing is known as to the factors which are responsible for its occurrence. The essential change is one of abnormally premature ossification of cartilage bones, whilst membrane bones behave in a normal manner. In consequence of these alterations the achondroplasiac is very much dwarfed.

*Clinically*, the appearance is characteristic. The vault of the skull develops normally, but owing to the abnormal ossification of the base it is very much out of proportion, whilst premature synostosis of the sphenoid results in a depression of the bridge of the nose not unlike that seen in congenital syphilis. The limbs are stunted in comparison with the trunk, and the epiphyses at the ends of the long bones are enlarged, giving an appearance very similar to that of rickets, while the diaphyses are shortened. The fingers are spread widely apart owing to divergence of the metacarpal bones, producing the so-called "trident-hand." The child stands with a marked lumbar lordosis owing to the presence of a coxa vara. These children are perfectly healthy both bodily and mentally and, apart from their diminutive stature, develop in a normal fashion. No treatment has any influence upon the development of the bones. Many of these children later in life earn their living in a circus or on the stage as comedians.

### ACROMEGALY

**Acromegaly** is an uncommon condition which affects principally the bones of the skeleton in young adults. It results from an hyperplasia or adenoma of the anterior lobe of the pituitary body, the increased secretion of which produces a symmetrical overgrowth of the skeleton. The hands and feet are enlarged and thickened and the bones hypertrophied. Should the disease commence before the epiphyses have united, there may be a great increase in length of the shafts of the bones and many giants are excellent examples of this condition. The forehead and orbital ridges are prominent and the nose is enlarged and broadened. Both the upper and lower jaws become very prominent and the lower lip thickened and overhanging, the whole facial expression being most unpleasing.

X-rays will reveal an enlargement of the sella turcica and, in consequence of the proximity of the optic chiasma, an optic neuritis with partial loss of the visual field is a common complication.

A patient with this disease generally suffers from headaches, lassitude and a tendency to sleep excessively, the appetite both for solids and fluids also being above the normal. Loss or diminution of sexual power is usual in men and amenorrhœa develops in women. The progress of the disease is very gradual, but by the time it has fully developed the whole aspect of the patient is characteristic.

*Treatment* is of little value and entirely symptomatic. The removal of the tumour of the anterior lobe of the pituitary will at times produce an improvement at any rate in the field of vision, should this be seriously affected, but the mortality of the operation is a deterrent factor.

### OSTEITIS DEFORMANS OR PAGET'S DISEASE

This condition, described originally by Sir James Paget, is a disease in which there is enlargement, thickening and bending of certain portions of the skeleton with which is associated a considerable amount of bone pain. Although it may start in young adults, it is most often seen in men of about 50 years of age, women being only rarely affected. Its etiology is quite unknown, but it is generally considered to be a chronic inflammatory condition, which at first leads to a decalcification and softening of the bones concerned. This softening is followed by a hypertrophy and thickening of the bones which again become calcified, being then very much enlarged and deformed (Figs. 527 and 528). Whilst at first this osteitis may be localised to only one portion of the bone it gradually spreads and the medullary cavity eventually becomes entirely obliterated. Any of the bones of the skeleton may be affected but there appears to be a predilection for the tibia, femur, vault of the skull and bones of the pelvis. It may begin in one tibia many years before any other bone shows a sign of the disease.

The first change is an alteration of the bony trabeculæ, which lose their normal arrangement. The compact bone becomes thicker, though

its density is much diminished, and the periosteum is hypertrophied. As the disease progresses these softened bones bend, but later regain their rigidity as they harden up. The tibia is much thickened, more especially in the upper part, and then bends. The skull bones are also thickened. If a chronic osteitis, the nature of which is obscure, commences in one bone the diagnosis may often be settled by X-raying the skull or pelvis, which will reveal the mottled appearance so characteristic of this disease. The enlargement of the skull will result in the patient's need to buy hats of constantly increasing size. When the disease has become fairly general he stands with a rigid kyphosis, the head carried well forwards and the lower limbs bowed

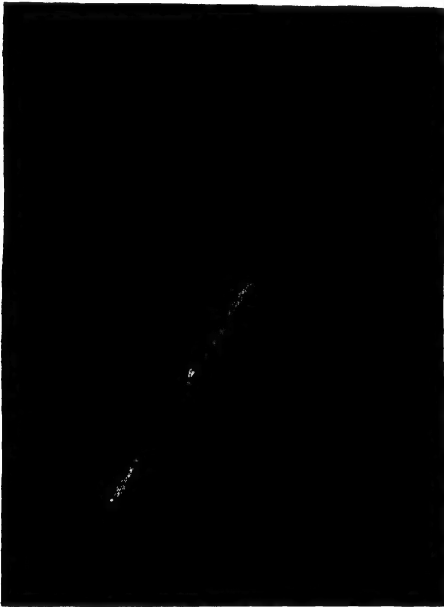


FIG. 527

Early radiographic changes in osteitis deformans.



FIG. 528

Late changes in osteitis deformans.

anteriorly. As the condition progresses he loses height and becomes so bent up that walking is difficult. Although the vault of the skull is so commonly affected in this disease the facial bones seldom show much change. The patient complains as a rule only of pain in a bone or bones. This may get gradually worse and at times be almost intolerable. Fractures seldom occur but, should they do so, union takes place satisfactorily. An osteogenic sarcoma is peculiarly liable to develop in a patient who has suffered from this disease for many years, and such growths, which are of the spindle-celled variety, have the usual prognosis associated therewith. Death, apart from the development of a sarcoma, takes place from some other disease, and these patients, owing to the rigidity of their chests, are very liable to develop pulmonary complications.

*Treatment.*—Nothing will stay the progress of osteitis deformans, but it may take many years to develop to the stage when the patient

presents the classical appearances. The pain may be relieved by certain measures, such as small doses of thyroid and X-rays which occasionally give relief. If the pain becomes so bad that the patient is prevented from sleeping at night and from walking about, guttering of the shaft to relieve the intramedullary tension will sometimes give considerable relief, at any rate for a time, whilst if the tibia is much bowed an osteotomy will correct the alignment. Fractures, if they occur, require treatment in the usual manner until union is firm. When a sarcoma develops amputation or treatment by radium may be called for to relieve pain. Death takes place, as in any other growth of this nature, from metastases in the lungs.

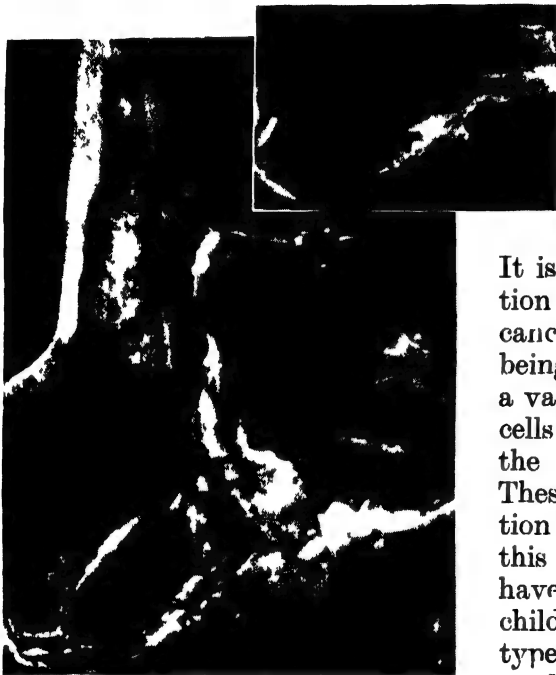


FIG. 529

Osteitis fibrosa in the lower end of the femur.

FIG. 530 (inset).

Osteitis fibrosa in the inner end of the clavicle.

### OSTEITIS FIBROSA

**Osteitis fibrosa** or fibro-cystic disease of bone was first described by von Recklinghausen in 1891.

It is characterised by the formation of cavities in the bone, the cancellous tissue and medulla being absorbed and replaced by a vascular tissue containing giant cells, which lines the walls of the cysts (Figs. 529 and 530). These are formed by the absorption of blood from the sites in this tissue where hæmorrhages have taken place. It occurs in children or young adults and two types are seen.

1. **The Solitary Bone Cyst** met with in long bones, especially at the upper end of the femur or the upper half of the shaft of the humerus. Slight pain may call attention to its presence, and this is revealed by an X-ray which

shows a clear area in the centre of the affected bone with thinning of the cortex. Again, a spontaneous fracture may occur with little or no violence in a bone, which has been unsuspected of being in any way abnormal (Fig. 531).

**Treatment.**—When a fracture occurs through such a cyst it is usually found that union takes place without any trouble and that the cyst is thereby cured. If, however, the cyst is discovered by X ray the bone must be exposed and its contents curetted out. Bone grafting to fill up the cavity may hasten a cure, but is not essential.

These cysts may contain blood and be lined by a vascular

membrane containing typical giant cells of the osteoclast type, or they may be filled with a clear yellow fluid.

## 2. Generalised Osteitis Fibrosa.

—This type of disease is rather rare. It may affect any of the bones but usually starts in one month or years before any other shows evidence of being affected. It differs from the solitary bone cyst in that it is associated with changes in the parathyroids. In some patients the adenoma of the parathyroid can be palpated, but it is not usually discovered until an exploration is carried out. As the result of its excessive secretion calcium is absorbed from the bones into the blood stream, the content of which rises to as much as 16 mg. per 100 c.c.

Pain in a bone calls attention to the condition which is revealed by X-ray. Spontaneous fracture may occur as in the solitary cyst, and unites in the same way (Fig. 532). Exploration of the cyst shows it to contain a clear yellowish fluid, but curettage alone does not cure the lesion, which may progress in the surrounding bone. Treatment consists in exploration of the parathyroids and removal of the adenoma, after which the disease of the bones will automatically improve. Medical treatment otherwise has no influence upon the disease.



FIG. 532

A radius showing the changes due to osteitis fibrosa in the greater part of its extent, and union after fracture.



FIG. 531

X-ray appearance of a single cyst in the upper end of the humerus, which has successfully united after a pathological fracture.

## FRAGILITAS OSSIUM OR OSTEOGENESIS IMPERFECTA

**Fragilitas ossium** is a congenital disease of bone in which there is a liability to the occurrence of fractures on the slightest provocation. It appears to develop in early childhood (Fig. 533), but is occasionally present in a new-born infant. There is a defective development of the compact tissue, so that the bones are brittle and shell-like. Nothing is known as to its causation. The fractures which occur are painful but similar to others except that they unite very slowly and then in a bad position if they have not been splinted continuously. When first seen these children have usually had many

fractures, and apart from the fact that their sclerotics are sky-blue in colour little else is to be found. The diagnosis of fragilitas ossium is not usually made until several fractures have been sustained (Fig. 534). Many of these infants die when quite young, but others appear to outgrow their tendency, although they are liable to be left with distorted and atrophic bones, which are quite incapable of fulfilling their normal function.

No treatment beyond that of the fractures is of any value. Many extracts of the ductless glands have been tried but they have no influence upon the condition of the bones.



FIG. 533

A small child with multiple deformities following fractures in fragilitas ossium.



FIG. 534

X-ray picture of the child in Fig. 533.

### OSTEOCHONDRITIS

**Osteochondritis** is a condition affecting certain bones during the period of growth, of which little or nothing is known either as to etiology or pathology. It is supposed by some to be a mild chronic infective process, whilst others consider it to be a response to some slight trauma, which produces an interference with the blood supply of the affected portion of bone. In none of the variety of conditions which can be grouped under the title of osteochondritis does suppuration ever take place, and therefore it is probable that the suggestion of trauma as the primary cause is the more likely one.

### PSEUDOCOXALGIA

Perthes' disease is an affection of the hip joint seen in children between the ages of 5 to 10 years. In its signs and symptoms it much resembles a tuberculous arthritis, and up to thirty years ago was always diagnosed and treated as such. Nothing is known as to its etiology or pathology, but it is certainly not a tuberculous condition.

The child, who is fit and well nourished, is found to have developed a limp and to complain of a pain in one hip joint. The onset is insidious and seldom is there any history of an accident even of the most trivial nature such as is common in a child of this age. The patient is a boy more often than a girl.

*Clinical Signs.*—Examination shows a perfectly healthy child who walks with a limp. The movements of the hip joint, unlike those of a tuberculous arthritis, are not limited in every direction. Flexion is nearly full in range, whilst abduction in flexion is very much limited, if not entirely abolished (Fig. 534). All the other movements are restricted

in range as the result of involuntary muscular spasm. The great trochanter is more prominent on the affected than on the sound side, and a fullness can usually be felt in Scarpa's triangle over the head of the femur. A positive Trendelenburg's sign is present. Muscular wasting does not occur, nor is the gluteal fold absent, as happens in a tuberculous arthritis. The X-ray appearance varies according to the stage at which the condition is first seen. It differs markedly from that of a tuberculous arthritis, in which there is an early decalcification of all the



FIG. 535

A boy illustrating the limitation of abduction during flexion of the hip which is so characteristic a sign of pseudocoxalgia.



FIG. 536

X-ray appearance in an early stage of pseudocoxalgia. Note the increased density in the right femoral head and the commencing flattening.

FIG. 537

The late results of poor treatment in pseudocoxalgia in the left hip. Note the great broadening of the neck of the femur and the bad re-formation of the head.

bones of the hip joint. This does not occur in pseudocoxalgia. In the early stages the epiphysis of the affected femur looks denser than that of the other side, and is also flattened, making the joint line between the head of the femur and the acetabulum appear wider than normal (Fig. 536). At a later stage the epiphysis is seen to be fragmented whilst the neck of the femur has become thickened. Little difficulty should be experienced in making a correct diagnosis, for the whole clinical picture is quite unlike that seen in a tuberculous arthritis.

*Treatment.*—If adequate treatment is instituted at an early date, completely normal function should be obtained. Rest in recumbency with a fixed extension is essential. No weight-bearing of any kind must be permitted until the skiagram shows that the bones concerned have recovered their normal density. Such treatment needs to be maintained for a period of twelve months or perhaps longer.

Treated in this way a hip joint with a full range of movement in

every direction is obtained, but if neglected an impairment of function results, which in later years may be followed by an osteo-arthritis in consequence of a misshapen femoral head articulating with an acetabulum, into which it does not fit (Fig. 537). Treatment in recumbency, whilst changes are taking place in the upper end of the femur, does not prevent the femoral neck from becoming thickened or the epiphysis from being flattened out to some extent. It does, however, maintain a smooth articular surface fitting the acetabulum, the upper lip of which grows out a little to accommodate the slightly enlarged head.

### KOHLER'S DISEASE

This is an osteochondritis affecting the tarsal scaphoid. It commences most commonly in boys about the age of 4 or 5 years. The onset is sudden, the child limping and complaining of pain in the foot. The dorsum of the foot over the scaphoid becomes swollen, hot and tender, symptoms which in many ways resemble those of a tuberculous osteitis of this bone. An X-ray shows the scaphoid to have a bony outline smaller than that of the other foot. The nucleus is denser, flattened and sometimes fragmented. It can be differentiated from a tuberculous osteitis by the fact that neither is there rarefaction of the bone, nor any loss of density in the other bones of the tarsus (Fig. 538).



FIG. 538

X-ray appearance in Kohler's disease.

until symptoms subside and an X-ray shows that the bone has recovered its normal outline.

*Treatment.*—If the symptoms are very marked, the foot requires fixing in plaster for a month; otherwise all that is necessary is to keep the tarsus strapped

### SCHLATTER'S DISEASE

This condition is seen most often in boys at school, and everything points to it being traumatic in origin. The upper portion of the tibial tubercle is lifted up from the underlying shaft of the tibia by the pull of the ligamentum patellæ. Pain, swelling and tenderness are the clinical signs, whilst the tubercle itself appears to be enlarged. An X-ray will show it lifted up from its base and at times also fragmented (Fig. 539).

*Treatment.*—If the tender area is strapped, full activity, except participation in strenuous games, may be permitted. In most patients



the symptoms persist for about six weeks. It is seldom necessary to fix the limb in plaster of Paris.

#### SCHEUERMANN'S DISEASE

This condition is an osteochondritis affecting the epiphyses of the vertebræ in young children. It is likely to produce later a kyphosis as the result of disturbed development of the vertebræ, unless the child is treated in recumbency during the active stage. Unfortunately many children have already developed a kyphos before advice is sought.

#### KEINBOCH'S DISEASE

This is a condition somewhat similar to Kohler's disease, which develops in the semilunar bone of the wrist. It differs, however, in

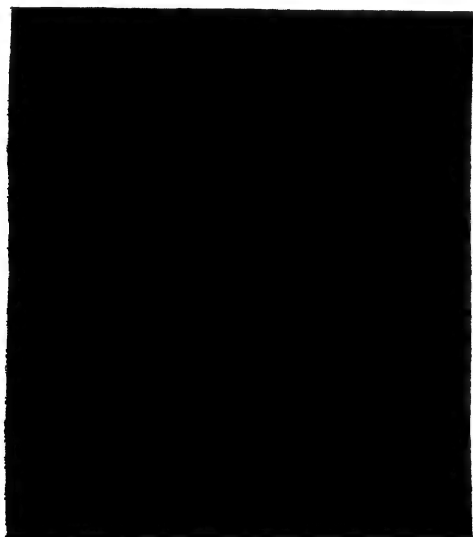


FIG. 539

X-ray appearance in Schlatter's disease.



FIG. 540

X-ray appearance in Keinboch's disease.

that it occurs in the adult and not the child. Injury appears to have some relationship to its occurrence. Pain and limitation of movement with muscular spasm and tenderness over the dorsum of the wrist are complained of. The X-ray shows flattening of the semilunar bone with areas of apparent increased density (Fig. 540). Treatment consists in fixation in plaster until the pain and tenderness subside. Excision of the semilunar bone may be necessary to relieve symptoms, but restoration of function after operation is very delayed.

Osteochondritis has been and continues to be described in many bones, but those given above are most commonly seen.

#### HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY

This is a rare condition which may develop in patients suffering from any chronic pulmonary disease, such as bronchiectasis, though it is

also seen in children with congenital cardiac disease or in adults with a chronic heart lesion. The ends of the fingers become enlarged and swollen or "clubbed," due to thickening of the soft tissues. Later, new bone formation takes place along the shafts of the phalanges. Patients who have suffered from pulmonary or cardiac disease for many years may exhibit similar changes in the bones of the forearm and leg. The condition probably results from some toxic absorption, or is produced by an interference with the return of blood from the extremities, which are in a condition of constant venous engorgement. Treatment has no influence upon this condition.

### **CLEIDO-CRANIAL DYSOSTOSIS**

This is a rare familial condition in which certain membrane bones fail to undergo proper ossification. Sometimes several members of one family are affected. As the result of imperfect development of the frontal and parietal bones the anterior fontanelle may remain widely open until late adolescence. This is not a hydrocephalic condition, though at first sight it may appear to be so, and the child is of normal mental development. The clavicles also are only partially developed so that the shoulders can almost be made to touch in front of the thorax. This partial absence produces very little functional disability. No treatment will hasten in any way the ossification of the bones concerned. The portion of the clavicle which is undeveloped in the child remains so throughout life.

## **TUMOURS OF BONE**

Tumours of bone may be simple or malignant, the latter being either primary in the bone itself or secondary to some other neoplasm.

### **SIMPLE TUMOURS**

Almost any type of connective tissue can give rise to a tumour in bone, but in practice they are few in number, chondromata, osteomata and osteoclastomata being the only ones which are commonly met with.

#### **CHONDROMATA**

A chondroma is a tumour composed of a lobulated mass of avascular hyaline cartilage, the surface of which is covered with a layer of fibrous tissue forming a limiting membrane. The cartilage which composes it differs from the normal hyaline variety of articular cartilage in that its cells vary in size and shape and are arranged in an irregular manner. Two types of chondromata occur in bone.

1. **Multiple Chondromata** or **Enchondromata** are seen in the small bones of the hands and feet, although the latter are much less often affected than the former. The cartilaginous tumour begins in the

interior of the shaft close to the epiphyseal cartilage and as it increases in size produces an expansion of the bone, thus forming a fusiform swelling. After a time these swellings may reach a large size when they are lobulated and irregular in appearance (Fig. 541). The growth of the tumour by its pressure produces an absorption of the osseous tissue surrounding it, but while this is occurring new bone is laid down beneath the periosteum, and in this way the bone appears to have been expanded by the development of the growth within. An X-ray at an early stage will show only a cyst of the bone together with destruction of the cancellous tissue and thinning of the cortical bone (Fig. 542). These tumours seldom give rise to any symptoms beyond deformity of the finger, or if they have been present for a long



FIG. 541

Multiple enchondromata in the finger.

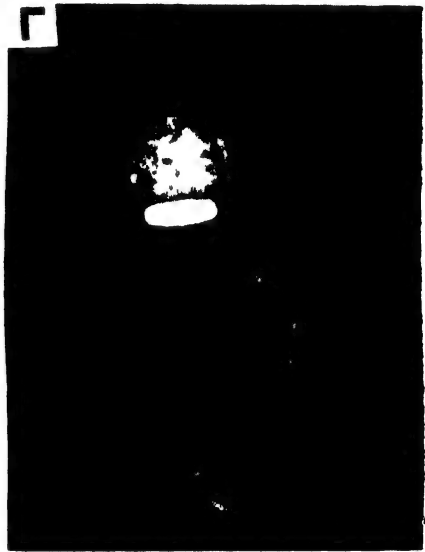


FIG. 542

X-ray appearance of multiple chondromata in the hand.

time advice is sought on account of interference with the function of the hand or for cosmetic reasons. Diagnosis when the tumours are of any size is easy, but when the first one appears it is very similar to either a simple cyst or a tuberculous dactylitis. From the former it may be impossible to differentiate until it is explored, whilst from the latter it can be distinguished by its slower period of development and the absence of any tenderness or inflammatory involvement of the skin, such as is usually seen in a dactylitis.

**2. Solitary Chondromata.**—A solitary chondroma is found most commonly at the upper end of the femur or humerus, or it may arise from the inner surface of the bones of the pelvis. Whilst this tumour commences close to the epiphyseal cartilage it does not appear to be derived from it. Although it may be present in childhood, such a tumour grows very slowly and may not be apparent until adult life, when either from its size or from pressure upon surrounding structures it causes symptoms. Although it is a benign tumour and is limited

by a capsule, it may erode the bone where it is attached. In some, small areas of calcification are dotted about in their substance, but no true ossification takes place (Fig. 543). They may undergo cystic degeneration or in the later stages become sarcomatous. When such a tumour alters its character from a simple to a malignant growth, it shows this change clinically by the onset of pain and a rapid increase in size.

*Treatment.*—Multiple chondromata require incision, the cartilaginous growth being curetted out and, if necessary, the cavity then filled with a bone graft. Occasionally it may be necessary to amputate a finger if several tumours are present and interfering with the function of the hand.

The large single chondroma in theory requires excision together with the portion of the bone from which it arises. Owing to their situation this is sometimes difficult or impracticable, and should they take on malignant characters their removal is certainly impossible. Treatment by radium or X-rays has then to be relied upon.



FIG. 543

The lower end of the femur showing the characteristic features of a solitary chondroma as described in the text.

## OSTEOMATA

Two varieties of osteomata occur, the cancellous which are seen quite frequently, and the ivory which are rare.

1. **Cancellous Osteomata** arise from the ends of long bones close to the epiphyseal cartilage. They are supposed to be derived from islets of displaced epiphyseal cartilage, and thus to

be common in children who in infancy have suffered from rickets. A single tumour may develop or several may be present. The *single* osteoma is found most frequently on the inner side of the lower end of the femur close to the adductor tubercle, or at the upper end of the tibia. It is attached by a pedicle to the shaft of the bone and its surface is covered with hyaline cartilage, beneath which growth continues so long as the epiphysis remains ununited to the diaphysis. Such a tumour is discovered generally by accident or as a result of the overlying adventitious bursa becoming inflamed. Less often a tendon slipping over the tumour may give rise to symptoms and lead to its discovery. The tissues over and around the osteoma are freely movable and are not attached to it, though they may be displaced by its increase in size.

*Multiple* osteomata occur in the condition known as *diaphysial aclasia*, in which tumours develop in childhood in relation to the

growing ends of any of the bones and on the scapula, close to the small epiphysis which appears on the margins of this bone. The condition is often hereditary and may occur in several members of a family. Only one or two tumours may be present or there may be a dozen or more (Fig. 544). The development of the diaphysis of the affected bone from which the osteomata arise is abnormal, for it is much increased in breadth and the condition is evidently unlike the single osteoma, an abnormality of bone growth. It has been suggested that this change is due to failure of the periosteum to exercise its normal restraining influence over the growing bone, which fashions it into its normal shape. In consequence the bones of the limbs are at times much altered in appearance. No symptoms occur unless the tumour by its size interferes with the action of tendons or by pressure causes pain.

In the case of both the solitary and multiple osteomata, removal becomes necessary only when symptoms develop. A *subungual exostosis* is a cancellous osteoma which develops on the inner or outer side of the distal phalanx of the big toe beneath the nail. It pushes the nail up and is liable to become inflamed and septic. When first seen the exostosis is quite commonly covered by a mass of granulation tissue. Such a tumour is considered to follow a chronic septic osteitis of the terminal phalanx of the toe, though there is no proof of this.

*Treatment* consists in removing half of the nail and the exostosis beneath it. If there is any active septic process going on this must be treated first.

2. **Ivory Osteomata** are rarely seen. They originate in those bones of the cranium which are ossified from membrane and may develop on either the inner or outer side of these bones. When they grow from the inner table symptoms may arise from pressure upon the cerebrum. They are also known to develop in the orbital cavity or one of the air sinuses. If causing symptoms they require excision. An area of normal bone around is removed as the ivory osteoma is too hard for it to be possible to deal with the tumour itself.



FIG. 544

Multiple exostoses of the tibia and fibula. An example of diaphysal aclasia.

### OSTEOCLASTOMA

This benign giant-cell tumour develops most frequently at the lower end of the femur, the upper end of the tibia or the lower end of radius, although it can occur in almost any bone. When it begins in the femur it starts as a central growth in one or other condyle and gradually destroys the cancellous tissue. As the tumour enlarges it

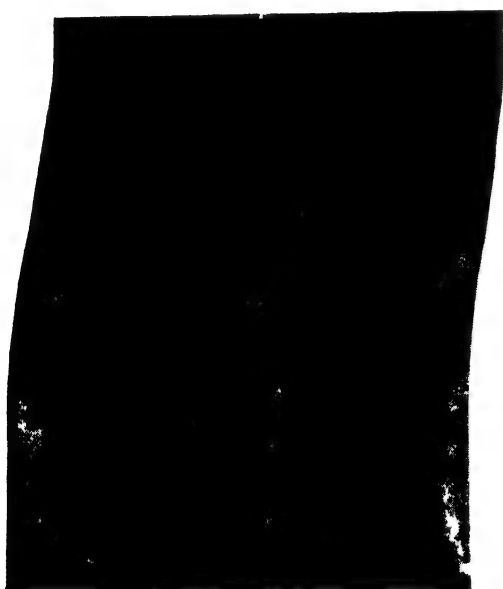


FIG. 545

The lower end of the femur destroyed by an osteolytic sarcoma.

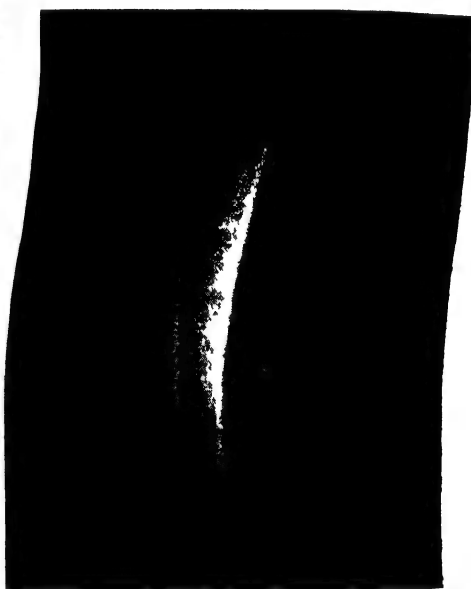


FIG. 546

X-ray appearance of an osteogenic sarcoma.

sometimes present if the tumour is of any size, and this may obscure its presence by directing attention to the joint rather than to the growth itself. Pulsation may be elicited, but the so-called "egg-shell crackling" is rarely, if ever, obtained. A skiagram shows a cystic expansion of the bone with coarse trabeculae running in an irregular manner across the diseased area. Where the growth abuts on to normal bone an attempt at sclerosis is sometimes seen, as though an effort was being made to isolate the tumour and prevent its expansion into the normal cancellous tissue.

*Treatment.*—Under a tourniquet the tumour must be explored and the growth cleared out. The walls of the cavity need to be curetted very carefully with a sharp spoon and cauterised with pure carbolic acid. In some situations it will be advisable to excise completely the portion of bone involved and to replace it with a bone graft. If the tumour has destroyed an extensive area of bone, or the soft tissues are



*Amos Hansen*

FIG 547

The upper end of a tibia replaced by an osteoclastoma, the characteristics of which are well shown.

already involved, an amputation of the limb will be necessary as the only means of obtaining a complete cure.



FIG. 548

A clavicle showing enormous growth of new bone in an osteogenic sarcoma.

## MALIGNANT DISEASE OF BONE

### OSTEOGENIC SARCOMA

This type of tumour is comparatively rare, although it is the most common malignant tumour to commence as a primary growth

of bone. It has long been customary to consider two types of sarcoma of bone, the periosteal and the endosteal. This is a purely artificial classification. The growth may spread more towards the interior (Fig. 545) in one case, the so-called osteolytic type, whilst in another it may spread beneath the periosteum, producing what is regarded as the typical periosteal sarcoma with spicules of bone set at right angles to the shaft (Figs. 548 and 549), thus giving the characteristic fan-like or radiating appearance in a skiagram (Fig. 546). What influences the direction of spread of the growth is unknown. It needs to be appreciated that in only about 18 per cent. of bone sarcomata is this radiating appearance seen, and that therefore it is not a characteristic of the majority of these tumours. The growth is limited by the periosteum so long as this membrane remains intact, but once it is destroyed by the growth or opened by an exploratory incision, invasion of the soft tissues takes place very rapidly and the development of secondary growths is hastened. Osteogenic sarcomata occur in the lower end of the femur more often than in any other situation, over 50 per cent. of all sarcomata of bone developing there. They are essentially seen in the young and seldom develop in an adult over the age of 50 years, except in connection with osteitis deformans. Whilst on some occasions they undoubtedly appear to follow upon some minor injury, the relationship of trauma to the development of the growth is not clear. The traumatic origin of a sarcoma is denied by many, but sometimes the connection

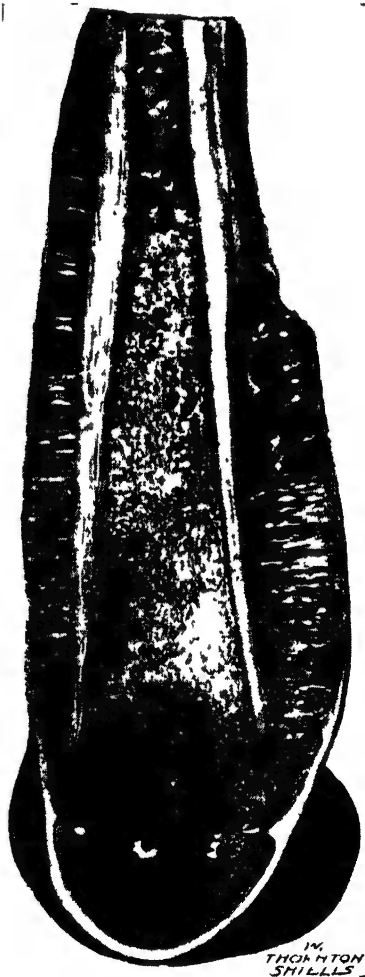


FIG. 549

An osteogenic sarcoma of the lower end of the femur with the typical radiating spicules of bone.



between the two seems so close that it is difficult to exclude the possibility.

The characteristic cell in all osteogenic sarcomata is spindle-shaped and, whilst giant cells not infrequently occur, they are of no importance in so far as the malignancy of the tumour is concerned. Bone sarcomata frequently present mixed constituents, bone and cartilage being commonly found in them. The blood vessels are large and thin-walled, being lined in some situations by tumour cells alone. In consequence of this close relationship, secondary deposits are carried by the blood stream and not by the lymphatic vessels.

*Clinical History.*—The development of an osteogenic sarcoma is very insidious and in the ordinary way, by the time advice is sought, the growth has already reached an advanced stage. Persistent pain in bone in a young person for no apparent reason should always give rise to the suspicion of a growth. But, although this pain may not, as far as the patient is concerned, be an outstanding symptom, there is always some history of pain, if care is taken to elicit it. The tumour, which is situated at the end of a bone close to the epiphysis, has a smooth surface and gives a sensation only of enlargement of the end of the bone (Fig. 550). The superficial veins of the limb may be enlarged and distended. Occasionally, pulsation can be felt in some portion of the swelling, though it may be difficult to detect, and a murmur



FIG. 550

The clinical appearance of the region of the knee showing the swelling produced by a sarcoma of the lower end of the femur. The black marks indicate portals of entry for deep X-ray therapy.



FIG. 551

A young man with a sarcoma of the scapula.

will be heard on auscultation if pulsation is at all obvious. Effusion into neighbouring joints sometimes occurs and may conceal the presence of a swelling of the bone. The general health of the patient is good and does not show any signs of deterioration until a late stage (Fig. 551), when the growth is very large or secondary deposits have begun to appear in the lungs. Spontaneous fracture, which is common with secondary malignant growths of bone, rarely takes place in a primary sarcoma (Fig. 552). X-ray examination, whilst it may reveal the classical radiating appearance, in the majority of instances shows only an osteolytic

process or destruction of bone without any attempt being made to form new bone (Fig. 553). The articular cartilage on the end of the

bone is seldom damaged, having a great resistance to the invasion of any malignant growth either primary or secondary.

The *diagnosis* of an osteogenic sarcoma is either a matter of considerable ease or great difficulty. In infants a subperiosteal hæmorrhage due to scurvy or hæmophilia may clinically be very difficult to differentiate, but an X-ray examination will settle the diagnosis, as in neither of these two conditions do the changes approach those of a sarcoma. In older children a solitary gummatous osteitis starting in the cancellous tissue at the end of a bone may closely resemble an early sarcoma, and only after a Wassermann reaction may it be possible to settle the diagnosis. The effects of treatment on a gumma will result in an improvement in the X-ray appearance within a very



FIG. 552

A pathological fracture through a chondrosarcoma of a phalanx.

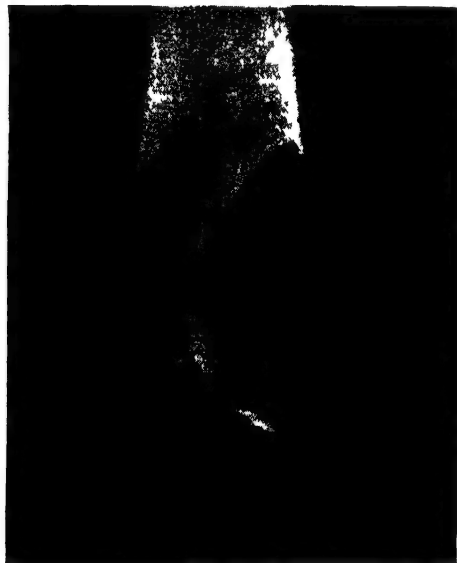


FIG. 553

X-ray appearance of an osteolytic sarcoma.

short period, and thereby prove definitely that the condition is not an osteogenic sarcoma. In adults a syphilitic gummatous periostitis may give rise to doubt, but this again responds to treatment very rapidly. In any patient over 50 years of age a diagnosis of osteogenic sarcoma should seldom be made unless the tumour develops in a case of osteitis deformans or until the possibility of a secondary growth has been ruled out. Few primary osteogenic sarcomata have ever been described apart from osteitis deformans in a patient of this age.

If there is any doubt as to the nature of a tumour which is suspected of being a sarcoma, exploration for the purpose of making a histological examination *should be avoided*. In many cases the biopsy has failed to make the diagnosis certain, whilst an exploration increases the rapidity with which secondary deposits may occur, for incision of the periosteum removes the only restraining force. An osteogenic sarcoma

seldom perforates the skin, save through an area of scar tissue. Instead of exploring the tumour it should be subjected to treatment with X-rays, when, if it is an osteogenic sarcoma, ossification will commence almost at once in the tumour substance.

The *prognosis* of an osteogenic sarcoma is bad. Nearly every patient with such a growth, by whatever method it has been treated or however early the correct diagnosis has been made, is dead within three years. In every case secondary deposits ultimately develop in the lungs and occasionally elsewhere (Fig. 554). Sometimes they appear within a few weeks of the primary growth being discovered and at other times not for two years. No indication can be given as to the length of time which will elapse between the discovery of the primary growth and the appearance of secondary deposits.

*Treatment.*—In view of the almost certain knowledge that amputation of the limb does not save the patient from secondary deposits, there is a tendency at the present day to avoid this method

of treatment and to employ either radium or deep X-ray therapy, both of which will stop the growth for a time. An X-ray taken subsequently will show a considerable amount of ossification in its substance. Unfortunately, the influence of these therapeutic measures does not appear to have a lasting effect, and sooner or later the tumour begins again to increase in size. Amputation then becomes a necessity either for the relief of pain or to avoid fungation through the skin. Disarticulation through the hip joint in cases of sarcoma of the lower end of the femur or of the tibia is an unnecessarily mutilating operation, for the result is no better than an amputation through the middle of the thigh, which enables a good artificial limb to be fitted. Rarely, if ever, does the growth recur in the amputation stump itself.



FIG. 555

The middle of the shaft of the femur showing a Ewing's sarcoma.

This growth is a very rare tumour which starts in the middle of the shaft of a long bone (Fig. 555). The clinical signs are similar to those of a subacute or chronic osteomyelitis, there being attacks of pain and tenderness with rises of temperature, which settle down for



FIG. 554

Widespread cutaneous metastasis from a sarcoma of the ilium.

### EWING'S SARCOMA

an interval only to recur again perhaps several weeks afterwards. The shaft of the affected bone is felt to be thickened and tender. A skiagram shows changes very similar to those seen in a chronic osteomyelitis, except that the subperiosteal bone, which is laid down, is arranged parallel to the shaft in definite layers, whilst the surface of the bone is smooth and not rough as it is in a chronic infective osteitis. The cells of the tumour are small and round, being arranged in intimate relation to the blood vessels. There are no giant cells in these tumours.

The *diagnosis* is very difficult, for exploration is dangerous as the tumour is liable to become more vigorous in growth afterwards and subsequently to fungate through the incision.

*Treatment.*—Radium or X-ray therapy will produce a diminution in the size of the tumour and relief of symptoms so that it appears to be cured, but after a few months the attacks recur. Secondary deposits eventually occur, although amputation is considered to give a better prognosis, provided it is performed early, than it does in an osteogenic sarcoma.



FIG. 556

Extensive replacement of the humerus by secondary carcinoma.

#### MULTIPLE MYELOMA

Myelomatosis is a rare disease in which multiple growths develop in the marrow with destruction of both the cancellous and cortical bone, there being no attempt at the formation of new bone. This condition is seen most commonly in patients of 40 to 50 years of age and is associated with poor general health. It is now believed to be a plasma-celled sarcoma. The most constant symptom is bone pain of a persistent nature. Bence-Jones's protein is found in the urine in only 50 per cent. of cases, and its absence is of little importance in making a diagnosis. Its presence in the blood is quite frequent. The condition as seen in skiagrams

may resemble very closely that of multiple secondary growths. Treatment is of no value.

#### SECONDARY MALIGNANT DISEASE OF BONE

Malignant disease of bone secondary to some other growth is most commonly seen in association with a carcinoma of the breast (Fig. 556), but tumours of the prostate and thyroid often give rise to osseous metastases, as also do hypernephromata of the kidneys.

The clinical signs of secondary deposits in bone fall into two main groups. In one class the presence of the deposit is revealed by pain developing for no obvious reason. The characteristic feature of this

pain is that it slowly but steadily increases in severity and duration until it becomes constant both by day and night, so that relief is obtained only by the administration of constantly increasing doses of drugs. The second class is that in which the presence of a carcinomatous deposit in bone is unsuspected until a pathological fracture takes place.

The characteristic X-ray feature of secondary deposits is absorption of bone by the growth, and absence of new bone formation (Figs. 557 and 558) in most, but occasionally osteosclerosis is well marked (Fig. 559).

Secondary carcinomatous deposits are found more commonly in certain situations than others. Thus, metastases from a tumour of the breast are seen most often in the body of a vertebra, where they give rise to persistent pain and the development, due to destruction and consequent collapse of the vertebra, of a sharp kyphosis which is not unlike



FIG. 557

Secondary deposits in the twelfth dorsal vertebra

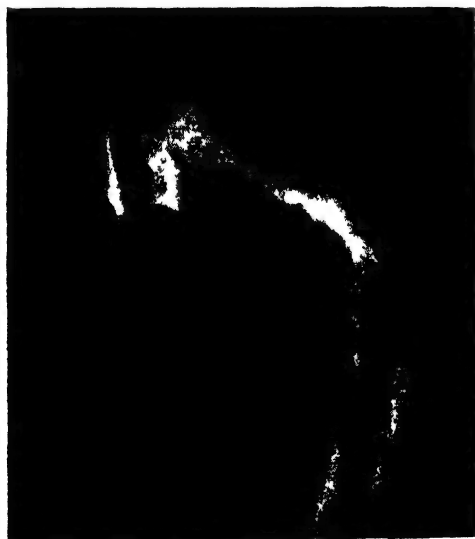


FIG. 558

Secondary carcinoma in the upper end of the femur.

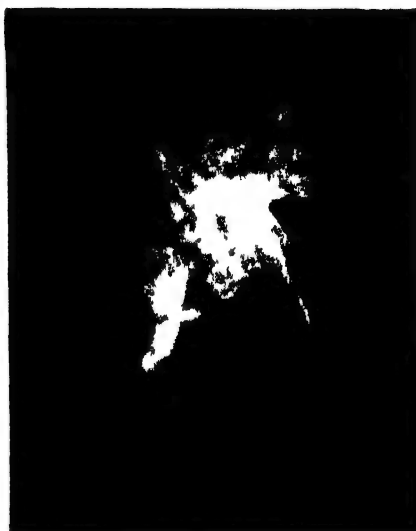


FIG. 559

Secondary involvement of both femur and pelvic girdle from carcinoma of prostate, showing marked sclerosis.

that seen in a tuberculous spine. They are also not uncommonly found in the femur below the trochanters, and this is a common

situation for pathological fracture. Secondary deposits from a tumour in the thyroid or from a hypernephroma often occur without evidence of the primary growth. The secondary growths from a hypernephroma develop most frequently either in the intertrochanteric region of the femur or in one of the bones of the pelvis. They are very difficult to distinguish from other malignant growths, especially as they may pulsate in the same way as does an osteogenic sarcoma, but they occur in older subjects than do primary growths.

*Treatment.*—The treatment of a secondary malignant growth of bone is entirely confined to providing relief of symptoms. Usually this means the administration of such drugs as are necessary. Pathological fractures require splinting in the same way as any other fracture, and in many instances union does take place. Fractures due to the presence of a secondary growth from a hypernephroma do not unite and the patient rapidly becomes gravely ill.

### CYSTS OF BONE

Simple cysts of bone are uncommon, but are seen in the neck of the femur, the upper end of the humerus and the tibia. They give rise to few symptoms and their presence is often unsuspected until a pathological fracture occurs. X-ray examination reveals the presence of a cyst with absorption of the cortical bone, through which the fracture has taken place. Exploration of such a cyst reveals a cavity filled with a clear yellow fluid and lined with a thin membrane which often contains giant cells. These cysts are probably a localised osteitis fibrosa and are seen in children or adolescents (cf. Fig. 531, p. 1041).

*Treatment.*—When a fracture takes place through such a cyst, union readily occurs and the underlying bone lesion is in most instances thereby cured. If the cyst is discovered before fracture, it should be explored and curetted out, whilst the introduction of a graft to fill up the gap left will at times hasten recovery and obliteration of the cyst.

**Hydatid Cysts.**—The presence of hydatid cysts in bone is rarely seen in this country, except in patients who have lived in climates where the *tænia echinococcus* is met with frequently. In consequence the diagnosis is seldom made correctly. The cysts produce destruction of the cancellous tissue and then absorption of the cortical bone. Symptoms are rare, and the occurrence of a pathological fracture is usually necessary to call attention to disease of the bone.

*Treatment.*—Exploration of the affected bone and curettage are required, and after this the disease seldom recurs. When the bone has been fractured the cysts first require removal; when, if the bone is adequately splinted, union readily occurs.

E. P. BROCKMAN.

## CHAPTER XLVIII

### DISEASES OF JOINTS

**G**ENERAL CONSIDERATIONS.—Before considering the diseases which affect the joints of the body, it is necessary to review briefly certain anatomical points, for upon a proper knowledge of these depends the appreciation of the pathological changes which occur in them. Every joint is a potential space between the ends of the bones which form the articulation. Their surfaces are covered with a layer of hyaline cartilage, which is called the articular cartilage. It is avascular and derives its nutrition from the synovial membrane and from the underlying bone. As long as this articular cartilage remains intact it is very resistant to infection, but once any portion of it has been destroyed the remainder is rapidly undermined by the inflammatory process and, a portion having been separated from the bone, it quickly peels off in pieces which disintegrate and disappear. Connecting the two ends of the bones with each other is the capsule, which is strengthened by various ligaments to resist the many strains to which the joint is exposed. The movements of a joint are brought about by muscular action, and if the muscular control of a joint ceases for any reason the capsule and ligaments rapidly stretch. The inner surface of this capsule is lined by synovia, a serous membrane secreting a clear glairy fluid which is normally sufficient only to lubricate the opposing surfaces. The synovial membrane reaches to the edge of the articular cartilage but does not cover its surface. In those joints in which there are intra-articular ligaments, such as the knee, these are also covered with a layer of synovia. In joints where spaces exist between the synovial membrane and the capsule, such spaces are filled with fatty tissue, which permits an alteration in the normal capacity of the joint to occur. In many instances pouches of synovial membrane protrude from the joint cavity between the muscles controlling its movements, and these are liable to become involved and distended in inflammatory conditions of the joint itself. The inner surface of the synovial membrane is covered with a number of small villi, which are not visible to the naked eye under normal circumstances, but in chronic inflammation of a joint they may become enlarged and hypertrophied, giving the synovial membrane a shaggy appearance, known as a *villous arthritis*. The blood supply of the synovial membrane is derived from the *circulus vasculosus* which is formed around the joint from the many articular vessels which are present.

In certain joints, *e.g.*, the hip, knee, elbow and shoulder, the epiphyseal cartilage lies partly within the joint cavity, and an osteitis or inflammation of the metaphysis is thus more likely in these situations to infect the joint.

The nerve supply of a joint is derived from the nerves which supply the muscles controlling its movements, and therefore two joints may receive branches from the same nerve, in which case disease of one joint may cause pain to be referred to the other. Movements are controlled by the muscles which arise from or are inserted into the bones forming the joint, and when-

ever inflammation occurs, either as the result of trauma or infection, the muscles controlling its movement go into an involuntary spasm to prevent this taking place. Such spasm is protective and entirely independent of the will of the patient. But not only do these muscles go into this protective spasm, but they also start rapidly to waste. Thus, in the case of disease of the knee joint the vastus internus may waste to an appreciable extent within a few days.

**Methods of Examination.**—In all diseases of joints the taking of an accurate and detailed history is essential and in particular the details of how the condition first commenced are all-important. Only when this history has been obtained can examination profitably be carried out. In all patients the corresponding joint on the other side of the body must be exposed so that the normal may be used for comparison. Otherwise there is no standard by which to judge the affected joint.

**Inspection** reveals the presence of any deformity, of swelling or redness and of wasting of the muscles, which control the movements of the joint.

**Palpation** will demonstrate any tenderness, increase of local temperature and the presence or absence of an excess of synovial fluid in the joint cavity. The position where tenderness is found must be accurately defined, whether it is over the reflections of the synovial membrane, the edges of the articular cartilage, the capsule, the attachment of ligaments, or on the bone-ends themselves. The examination of the range of movement requires great attention. It is first necessary to ascertain whether limitation of movement, if any, is due solely to an involuntary protective muscular spasm or to any other cause. Very great care should be taken to identify minor degrees of limitation of movement in each direction, for these are the early signs of disease, and for this reason examination of the corresponding sound joint is all-important, for only by a comparison of the two can these minor limitations in movement be appreciated. The protective muscle spasm, in addition to preventing any movement taking place during the acute disease, may at the same time produce certain fixed deformities if it is permitted to act uncontrolled for any length of time. Thus, in disease of the hip in the early stages, this joint may be fixed in a position of flexion, abduction and external rotation. In the later stages the fixed deformity, which is present, is the result of changes which have taken place in the joint during the progress of the disease and also of secondary contractures which have occurred in the muscles, ligaments and joint capsule.

In the early stages the presence of a protective muscular spasm can be demonstrated by making a short sharp movement, when the muscles can be both seen and felt to contract. This protective muscular spasm can be very well demonstrated by forced internal rotation in an early example of disease of the hip joint.

**Measurements** of joints are of little value except for certain exceptions. Any swelling or muscular wasting should be estimated by inspection and palpation, the degree of either being judged by a comparison with the sound joint. In the hip, measurements are of value in that they reveal the presence of any change in the relationship of the head and neck of the femur to the acetabulum. Damage or destruction of any of the components of the hip joint results in a raising of the great trochanter, which can be revealed by measurements. Two particular methods are employed in the examination of all lesions of the hip joint. In one a line is drawn from the anterior superior spine of the ilium to the most prominent part of the tuberosity of the ischium. This, known as *Nélaton's line*, normally passes through the tip of the great trochanter. When any alteration in the relationship of the head and neck



of the femur to the acetabulum has occurred the great trochanter is raised above this line. The same information can be obtained by the measurement known as *Bryant's triangle*, which is formed by dropping a perpendicular line from the anterior superior spine on to the couch on which the patient is lying; a second line is drawn from the anterior superior spine of the ilium to the tip of the great trochanter, the triangle being completed by a third line which passes from the tip of the trochanter at right angles to the first line. A comparison of the two triangles on either side will reveal whether the trochanter is raised or not, and to what extent. Whilst Bryant's triangle is, strictly speaking, a more exact measurement than is Nélaton's line, the latter for all practical purposes is accurate enough and at the same time is more convenient to employ. Lastly, the stability of all joints requires examination, especially in the knee and hip. In the latter this can be revealed by the presence or absence of *Trendelenburg's sign*. When the patient is made to stand on a sound leg with the knee on the other side flexed to a right angle, the buttock on the latter side is raised to a higher level than that of the limb upon which the patient is standing. When the hip joint has been damaged by any disease, standing on the affected limb will result in the buttock of the sound side falling to a lower level. This sign is present in any condition in which there has been an upset of the normal mechanics of the hip joint, *e.g.*, pseudocoxalgia, an old tuberculous or infective arthritis not followed by ankylosis, coxa vara and congenital dislocation.

X-ray examination of all joints suspected of being the site of disease is essential, and to be of any value must include for comparison a skiagram of the sound joint on the other side. Both hips should be taken on the same film, as only thereby can a true comparison be made.

## INFLAMMATION OF JOINTS

When any inflammation develops in a joint, the changes which occur are identical with those produced in any other structure, being modified solely by the structure of the joint. The synovial membrane lining the joint capsule under normal conditions secretes a minute quantity of fluid, which helps to nourish the articular cartilage and provides a lubricant for the joint surfaces. As the result of injury or disease the amount of fluid secreted is increased and an *effusion* is said to have taken place. This increased secretion of fluid is part of the response of synovial membrane to inflammation known as *synovitis*. In this respect the synovial membrane is identical with the peritoneum and pleura, both of which when inflamed secrete an excess of fluid. As a result of this effusion the joint outlines are altered in appearance, being more easily appreciated in some joints than others, depending upon the amount of muscular tissue which surrounds them. The type of effusion varies according to the cause of the inflammation. Thus, it is called a *serous* effusion if only of lymph and serum, whilst if this becomes infected with organisms and contains leucocytes it is known as a *purulent* effusion, and an *empyema* is said to be present when the joint is distended with such fluid. A blood-stained effusion is known as a *hæmarthrosis* and occurs in injuries which either tear the synovial membrane or ligaments, or else fracture the bones entering into the formation of the joint surfaces. A hæmarthrosis may also be seen in hæmophilia, scurvy and certain blood diseases. Effusion into the *hip joint*, which is surrounded by strong muscles, is very difficult to identify unless the collection of fluid is

large in amount. On the other hand, in the *knee* which is not surrounded by large muscles an effusion is readily identified, as it causes disappearance of the normal hollows on either side of the patella and ligamentum patellæ, whilst the subcrural pouch becomes distended and obvious. When the effusion is considerable in amount no difficulty will be experienced in identifying its presence, but when there is only a slight effusion, it can be recognised by means of a "patellar tap." This consists in pressing the patella backwards with a sharp movement when it can be made to knock up against the articular surface of the anterior aspect of the femur, the tap displacing any fluid which lies between the two bones. If only a little fluid is present it can be identified by the same method when pressure is exerted on the subcrural pouch so as to force any effusion, which may have collected here, into the lower part of the joint.

When an effusion is present in the *ankle joint* the limb is held by muscular spasm in a position of equinus or equinovarus. The anterior aspect of the joint is distended and the hollows which normally exist on either side of the tendo achillis are obliterated.

At the *shoulder* the joint appears more rounded and its anterior aspect is filled up, so that the hollow below the clavicle disappears. When effusion in this joint is excessive it may track down the bicipital groove along the prolongation of the synovial membrane accompanying the tendon of the biceps, thus causing the appearance of a swelling in the upper arm.

At the *elbow joint* the grooves on either side of the tendon of the triceps are filled up and the prominence of the olecranon disappears.

At the *wrist joint* swelling appears all round the joint, being more marked on the dorsum and laterally. The hand is held in flexion and the condition can be distinguished from a tenosynovitis by the fact that with an effusion of the joint the tendons can move freely, whilst the characteristic fine crepitus of a tenosynovitis is absent.

When inflammation spreads from the synovial membrane to the other structures of the joint, the condition is known as an *arthritis*. Such an inflammatory process may be caused by a variety of infections, being either acute or chronic in nature. As the result of any type of arthritis the articular cartilage is liable to become softened and eroded. The bones may become the site of an osteitis and the capsule and ligaments be damaged or destroyed. In the process of healing the damaged structures are replaced by fibrous tissue, which forms adhesions between the various parts of the joint. Should the joint be very badly damaged the bone surfaces may be completely exposed and become fused together with osseous tissue. An *ankylosis* is said to have taken place. When movement is prevented by the formation of fibrous tissue, a fibrous ankylosis is said to have occurred. Such an ankylosis is unsound, there always being present a little movement which, if forced, is likely to give rise to pain and swelling. Deformities often develop in this type of ankylosis owing to the stretching of fibrous tissue from the pull of the more powerful muscles. When all movement has been abolished by osseous union between the two ends of the bones, a bony ankylosis is said to have taken place. In some cases of disease of the joint it is possible to retain movement whilst the inflammation is subsiding, but in other conditions, such as tuberculous arthritis, ankylosis is nature's method of attempting to cure the disease, and movement must be prevented by fixation either with some form of external splint or by operation, which aims at removing all the damaged tissue so as to leave two bony surfaces that will fuse together.

*Ankylosis*.—The position in which a diseased joint should be maintained, if there is any danger of it becoming fixed, varies with each individual joint,

for should it become fixed, it must do so in the position in which it will ultimately serve the most useful function ("the position of election").

In the hip the deformity of adduction and internal rotation must be avoided and the joint held in slight abduction. Flexion to 30 degrees is desirable to enable the patient to sit down in comfort.

The knee needs to be fixed with a few degrees of flexion, as full extension of the joint makes it hard to clear the ground with the foot, whilst any marked degree of flexion gives an ugly gait in walking, due to the apparent shortening. Fixation of the ankle must be obtained at a right angle.

In the case of the shoulder, an ankylosis is a considerable disability owing to the inability to rotate the limb in any direction. It should be held in about 45 degrees of abduction and slightly in front of the coronal plane of the body. Movements of the arm with the shoulder fixed in this position may be quite good owing to the movement between the thorax and the scapula. Too wide abduction is a disability, as it prevents the arm being placed in contact with the side of the trunk. Loss of movement at the elbow is serious and the position in which ankylosis should be allowed to take place must depend upon the work of the patient. For most purposes the forearm supinated and flexed to 90 degrees was said to be the position of choice, but such a position is really of little or no value. The forearm should be pronated and the degree of flexion or extension be decided upon in each individual.

The wrist must always be held in moderate extension, for ankylosis in flexion completely destroys the power of the hand.

The type of ankylosis, which follows upon an arthritis, depends upon the organism which is responsible for the infection and also upon the severity of the arthritis. Thus, a tuberculous arthritis uncomplicated by any secondary pyogenic infection results in a fibrous ankylosis, whilst an acute pyogenic infection which destroys the cartilage of the joint produces a true bony ankylosis. If an arthritis is treated in the proper position, it should be possible to prevent the development of deformities, which would later interfere with the patient's activities.

*Operations upon Joints.*—**Arthrotomy** consists in opening the joint cavity to examine the interior or to remove a portion of the synovial membrane for histological examination. This operation is also performed to remove a loose body or a torn cartilage or to let out the purulent fluid in a septic arthritis.

**Synovectomy** signifies the complete removal of the whole of the synovial membrane of the joint, and is sometimes carried out in the knee joint in patients with a villous arthritis, when the articular cartilage is not involved to any appreciable extent.

**Arthrodesis**, or fixation of a joint, entails the removal of all the synovial membrane and any intra-articular ligaments, as well as the cartilage from the bones forming the joint, with the object of allowing the two raw surfaces of bone to come into close apposition so that osseous union may take place between them, and thus movement of any kind be permanently abolished.

After this operation the limb needs to be fixed in plaster until fusion has occurred, but in a few weeks the patient must be encouraged to walk on the limb in this plaster, as weight-bearing is the best stimulus to encourage a satisfactory bony fusion. The period of time, which is necessary before a bony ankylosis occurs, depends upon the form of arthritis.

**Excision** of a joint is similar to an arthrodesis, except that the ends of the bone are widely removed by means of a saw instead of the cartilage being simply removed with a gouge.

**Extra-articular Arthrodesis** is fixation by means of a graft passing between the two bones concerned without removal of the two surfaces of the joint.

It is most commonly employed in dealing with the hip joint, but, though called extra-articular it is not truly so, as the joint in most instances must be opened in order to enable the graft to be satisfactorily fixed in position.

### TRAUMATIC SYNOVITIS

This condition occurs as the result of any injury to a joint either with or without a fracture. In the former the effusion is generally only serous, but if a fracture involves the joint surfaces the fluid may contain a variable amount of blood. The effusion develops rapidly and the joint becomes painful, tender and hot.

It is held fixed in a position of flexion by muscular spasm and any attempt at movement gives rise to pain. The synovial membrane is bruised or torn and becomes inflamed. Unassociated with any other injury a traumatic synovitis should respond well to treatment, the objects of which are to allow the injury of the synovial membrane to recover and to promote absorption of fluid as soon as possible, whilst at the same time guarding against muscular wasting and the formation of adhesions, which later would interfere with the function of the joint.

*Treatment.*—In the first few days rest is essential to permit healing of the synovial membrane, promote absorption of fluid and relieve pain. There is a danger of resting the joint for too long a period. Therefore it is wise not to use any kind of splint. Where possible the joint should be fixed with a pressure pad and bandage which, in addition to providing some fixation, also by the exercise of pressure promotes the absorption of the fluid within the joint cavity. At the same time the muscles controlling the joint must be kept in condition; otherwise, if they are permitted to lose their tone and waste, recovery may take a long time and the function of the joint subsequently be badly impaired.

It is only necessary to encourage the patient to use the joint through as great a range as possible, provided this does not excite muscle spasm or cause pain. Passive movements definitely do harm for they are out of the control of the patient, and massage to the joint itself will often delay recovery rather than hasten it. Any massage which may be given must consist simply in stroking the limb to soothe the spasm which may be present.

Should active movement not be maintained in an acute traumatic synovitis the surfaces of the synovial membrane, where they are in contact with one another, are liable to become stuck together by the formation of fibrous bands and adhesions of varying thickness and strength. However well a traumatic synovitis is treated, these adhesions do occasionally form. They may be quite slender, but nevertheless interfere with movement and hence cause a loss of function. Even minor limitations of movement may produce an interference with function quite out of proportion to the loss of range. The only treatment for such a condition is a manipulation of the joint under anæsthesia. If movement is only slightly restricted, such a manipulation can at times be carried out without any anæsthetic if the patient can be persuaded to relax the limb completely. The manœuvre in these patients consists in moving the joint once through its full range

in each direction. Great force is not required to do this with complete satisfaction. The joint having been moved, the patient must subsequently be made to use it actively. Little, if any, reaction follows and the condition is usually cured in a dramatic manner. If movement is very limited, with adhesions of long standing, it is wiser to move the joint through a limited range and observe how much reaction takes place. If this is considerable, further manipulation must be postponed until it has subsided, whilst if there is none the joint can be put through its full range on the second occasion without further delay or risk.

### PYOGENIC INFECTIONS OF JOINTS

The infection of joints by pyogenic organisms in most instances occurs as the result of a blood-borne infection from some other focus, but occasionally it may be a direct infection from a perforating wound, though this is rare in civil life. Direct extension from a bone, the site of an osteitis, will produce an arthritis or the joint may be infected by a pyæmic embolus. The organism responsible is usually a staphylococcus or streptococcus, other infections being comparatively rare.

Whilst in theory an acute infective synovitis is possible, in actual practice any infection of this nature involves the whole joint to some extent, although its principal effect may be synovial.

Whatever the source of the infection the onset is usually sudden. The joint becomes distended with fluid, is very painful, hot and tender. It is held in a position of flexion by involuntary muscle spasm, and any attempts to move it give rise to great pain and are resisted by the patient, who is obviously in real distress and quickly begins to show signs of toxæmia and fever, and may even in quite an early stage have one or more rigors. If the condition is not treated, or in spite of treatment continues to progress, the general state of the patient rapidly deteriorates. He refuses food and becomes more toxic, the temperature and pulse continuing to rise still further. The skin over the joint becomes red and the surrounding tissues œdematous; pus may burst out of the capsule and invade the overlying muscles, whilst, as a result of the inflammatory changes, the capsule and ligaments may soften or be destroyed and a *pathological dislocation* of the joint take place.

Unless satisfactory treatment is instituted at an early stage of the disease the patient will die either of septicæmia or toxæmia, or will be left with a badly disorganised joint with multiple discharging sinuses and a chronic osteitis, which may lead ultimately to the development of lardaceous disease.

*Pathology.*—When acute pyogenic infection occurs the synovial membrane rapidly becomes red, swollen and congested. From this inflamed membrane there is exuded a large quantity of fluid containing a few pus cells and organisms. If the joint is aspirated at this stage the fluid drawn off may appear quite clear and slightly yellow, but microscopical examination after it has been centrifuged and cultivated will reveal its true contents. This fluid, however, rapidly becomes purulent from the addition of polymorphonuclear leucocytes, which have migrated thence from the blood vessels of the synovial membrane, and

the condition is now known as an *empyema* of the joint. If successful treatment is instituted at this stage permanent damage to the joint may be avoided. Otherwise, the synovia is converted into granulation tissue, and this spreads very rapidly as a *pannus* on to the edge of the articular cartilage, which becomes softened and speedily eroded both at the margins and more especially at those spots where pressure is exerted. It will be stripped up from the underlying bone, granulation tissue having spread beneath it, and may lie loose in the joint in flakes, which are rapidly absorbed. The ligaments and capsule of the joint are œdematous and softened, allowing



FIG. 560

An acute pyogenic arthritis of the knee. The joint has been opened from the front, the patella being turned back. The articular cartilages are seen to be eroded by a "pannus" of granulation tissue.

subluxation or even complete dislocation to take place. The bone beneath the cartilage becomes the site of an acute osteitis, with absorption and necrosis of the cancellous tissue, which is converted into a mass of granulation tissue exuding quantities of pus (Fig. 560).

*Treatment* has two aims : first, to provide relief of tension within the joint, and second, to fix the limb in an appropriate splint with extension until the disease has subsided. A fixed extension rather than a weight and pulley is desirable. The object of fixation and extension is to keep the inflamed joint at rest by maintaining surfaces a little distance apart, thereby avoiding as far as possible any damage to the softened articular cartilage and at the same time relieving the patient of the acute pain, which is so exhausting. In the

early stages, when the condition has only just started, the tension within the joint can be relieved by aspiration, which may be repeated if necessary, but once the effusion has become frankly purulent the joint requires opening to permit free drainage. Rubber drainage tubes are unnecessary. If the capsule is freely incised nothing else need be done. The introduction of drainage tubes within the joint itself is certain to lead to ankylosis subsequently, and if they are employed at all they must be put down only to the capsule and not into it. When the condition is very acute irrigation may be called for, but it is not desirable as a routine. With such treatment a certain number of joints will recover, with a useful range of movement, though it may be many weeks before the inflammatory condition completely subsides, and it is impossible to give a prognosis as to the final result when the patient first comes under treatment.

When the articular cartilage has been destroyed and an osteitis of the underlying bone has started, fixation will need to be maintained in the position of election until ankylosis has occurred; otherwise, deformities will develop. Any sequestra, which may form during this stage, require removal before the sinuses will heal completely. Should the general condition, in spite of adequate drainage, continue to get worse an amputation may be needed as a life-saving measure. During the whole time the patient should be given an easily digested diet, the bowels being kept well opened and large quantities of fluid drunk. It is all-important to emphasise the need of sleep, for otherwise a patient's resistance may break down simply from lack of rest, and appropriate measures must be taken at an early stage to attain this end.

*Prognosis.*—This is always serious, both as regards life, for the patient may die of septicæmia or pyæmia, and also as regards the final function of the joint, ankylosis resulting in the majority of patients.

## ACUTE ARTHRITIS OF SPECIAL JOINTS

### THE KNEE JOINT

This joint is the site of a pyogenic infection more commonly than any other. It may become infected by direct spread from an osteitis of the lower end of the femur or the upper end of the tibia, from perforating wounds or from a blood-borne infection. The effusion is always marked and the limb is held flexed and lying on its outer side. The joint is red and swollen, whilst pain is very severe and the patient, usually a child, will cry out if any attempt is made to move it. A high temperature results and rigors may occur.

*Treatment.*—If there is an osteitis in either the femur or the tibia, which is recognised early, it is possible that the effusion into the knee joint is a sympathetic one at first and the bone lesion must be opened to relieve the tension in the bone and provide free drainage. If taken in time it is possible that the knee joint may be saved from infection and that the effusion will be reabsorbed. Once the effusion is infected the joint must be aspirated with all aseptic precautions in addition to draining the bone abscess. This is done by introducing a needle into the joint on the outer side of the patella. The knee is then fixed with extension in a Thomas' splint. Aspiration may need to be repeated, and if the local or general symptoms do not subside the joint must be opened on either side of the patella, washed out, and left open to provide free drainage, the limb being fixed as before.

When free drainage with fixation has been provided the temperature falls to a lower level very rapidly in most instances.

In a few patients the infection does not subside, and amputation may be called for. However well the condition responds to treatment, many acute pyogenic infections of this joint result in a fixed knee after many months of illness.



## THE HIP JOINT

Acute pyogenic arthritis of this joint is seen generally in young children as the result of an acute osteitis of the epiphysis of the head or neck of the femur ; rarely is it seen in older children or adults. The onset is sudden, with acute pain and fever, the limb being held everted and fixed in flexion and abduction by muscle spasm. There is a rise of temperature with the usual constitutional signs. In the early cases it is difficult to decide whether the condition is an arthritis or a pure osteitis, for there is no fullness over the joint, abscess formation cannot be detected, and an X-ray in this stage will not assist in the diagnosis. If nothing is done the temperature continues to rise and the child becomes obviously more ill. Left to itself the abscess will burst through the capsule and point either anterior to the great trochanter or through the posterior portion of the capsule, when it collects in the buttock ; the head of the femur may dislocate backwards on to the dorsum ilii in consequence of softening of the capsule and destruction of a portion of the epiphysis or lip of the acetabulum.

*Treatment.*—As soon as it is certain that the joint has been infected it must be opened and the tension relieved. The anterior approach is the most suitable unless an abscess has formed posteriorly, when this must be opened as well. Fixation of the patient upon an abduction frame with an extension is necessary and in the early stage nothing else should be done, for a certain number of cases will settle down and incision not be called for. Fixation, besides keeping the inflamed joint at rest, will prevent dislocation. Once the abscess has been opened and tension relieved the general condition will improve.

In infants the epiphysis of the head of the femur is usually damaged to some extent and may be entirely destroyed, whilst the acetabulum always undergoes a certain amount of alteration. When all sepsis has subsided, the joint is often partly subluxated and the patient walks with a limp, though movement is surprisingly good. In older children or adults the final result is a bony ankylosis, and therefore the hip joint must be kept during the active disease in a good position to prevent deformity. Should sequestra form, these will subsequently need removal. If the hip has become ankylosed in a bad position, function can be much improved by an osteotomy below the trochanters, the limb being fixed subsequently in plaster in a good position.

Acute infective arthritis of other joints is rare and needs no special description, the same principles of treatment being applicable.

## PYÆMIC ARTHRITIS

This type of arthritis results from a septic embolus, and is seen generally with an osteomyelitis. It is characterised by the joint becoming very rapidly distended with purulent effusion, often without any pain. The joint must be aspirated at once and the affected limb fixed in a splint with extension. If necessary, the joint may need aspirating a second time, or even opening and washing out. It usually recovers with a fair range of movement.



**SPECIFIC TYPES OF ARTHRITIS****PNEUMOCOCCAL ARTHRITIS**

This type of arthritis is due to a blood-borne infection either from the nasopharynx or from the lung in a definite attack of pneumonia. It may occur in a single joint or be part of a general septicæmia. It is usually a disease of infancy and is rarely seen in older children or adults. The larger joints, especially the hip and knee, are more often attacked than the smaller. The synovial membrane is affected some time before the other structures of the joint and with adequate treatment the infection may subside.

The effusion is usually thick and purulent, but occasionally may be seropurulent or even serous.

The characteristic clinical sign is a sudden painless effusion into the joint, the capsule of which becomes greatly distended. In this respect it resembles a pyæmic arthritis. The skin is usually unaffected, though at times it may be red. The general condition is not disturbed by this occurrence. The diagnosis is obvious if the child already has pneumonia, but otherwise it may be mistaken for a pyæmic arthritis or an early acute rheumatism.

*Treatment.*—The joint should be aspirated and the limb fixed in a splint with extension. In most instances this is all that is required, the infection settling down and leaving little permanent damage to the joint. Should it, however, fill up again it will require incision and washing out. Even then complete resolution may occur, but ankylosis is more likely to follow.

**TYPHOID ARTHRITIS**

An arthritis caused by the *B. typhosus* occasionally occurs in the course of this disease. The inflammation may be almost entirely confined to the synovial membrane, but when it involves other structures of the joint a pathological dislocation is apt to occur; this is free from any great pain and may be discovered by accident. Only very rarely does true suppuration take place, but if this should happen the joint must be opened and treated in a similar fashion to any other acute arthritis. Otherwise, aspiration with adequate fixation and splintage is all that is needed. A persistent effusion is liable to remain, which may be very resistant to treatment and leave a certain amount of permanent stiffness.

**DYSENTERIC ARTHRITIS**

An arthritis may occur in bacillary dysentery caused by Shiga's bacillus, and it is seen towards the end of the acute stage or rarely as a late sequela. It is never found in amœbic dysentery. Many joints may be involved, becoming distended and painful, so that the patient is of necessity confined to bed. It closely resembles a gonococcal or rheumatoid arthritis when seen in young adults.

*Treatment* consists in rest in bed with such splintage as is necessary to prevent the development of deformities. Anti-dysenteric serum,

whilst it may prevent the development of arthritis, has no influence upon it once the condition has occurred. Aspirin will relieve the pain in the joints. A certain amount of permanent stiffness may result, but as a rule there is recovery with normal movement.

### GONORRHOEAL ARTHRITIS

This always results from the gonococcus being transmitted from the primary focus through the blood stream to the joint or joints affected. The primary focus is, of course, the genito-urinary tract in nearly every instance. Although this form of arthritis commonly occurs within a few weeks of the original infection, which may itself be settling down, it is possible for it to start months afterwards, when only a chronic gleet is present. The virulence of the acute infection seems to bear no direct relationship to the development of an arthritis, a mild infection being just as likely to cause it as a more severe one. When one of the affected joints is aspirated the gonococcus can usually be cultivated from the fluid obtained. The patient is as a rule a young adult, but at times the disease commences in older subjects long after the original infection.

The inflammatory changes in the majority of cases start in the periarticular tissues before the joint itself is involved, but when its interior has become affected the pathological changes are similar to those of any other arthritis, except that a purulent effusion is very rarely seen. Whilst any joint in the body may be involved, the knee, wrist and elbow seem to be particularly liable to develop a gonorrhoeal arthritis.

Two main types of the disease are seen :—

1. **Monarticular**, affecting one large joint, such as the knee or elbow, in which the synovial membrane particularly is attacked, though there may be also a good deal of periarticular inflammation.

2. **Polyarticular**, affecting small and large joints alike. Effusion into the joint is not marked, but there is considerable oedema around it, with redness, heat and pain, and the general health of the patient is impaired. A rise of temperature is frequently present, though in both varieties suppuration rarely occurs.

*Clinical Signs.*—The history of onset of a gonorrhoeal arthritis is almost diagnostic in itself. The patient is awakened with acute pain in one or more joints. This becomes rapidly worse and in a few hours the joint may be swollen and so painful that any attempts to examine or move it are resisted by the patient, who lies in bed guarding the affected joint from the slightest alteration in position. The skin becomes red and stretched. Left untreated, the pain gradually subsides after several days, leaving a stiff joint with a certain amount of effusion and much periarticular thickening. Having been kept fixed during this stage deformities are very likely to remain, so that in the case of the knee it may be impossible to extend the leg, or in the elbow to flex or extend the forearm. In the polyarticular variety of the disease the joints of the hands and feet are especially liable to become fixed, as are those of the spine in young adults, where infection

around the intervertebral joints and discs may produce a completely rigid spine.

*Diagnosis.*—In the presence of a urethral discharge little difficulty arises in making a correct diagnosis. The fact that the patient denies any history of urethritis is of no importance, and with a typical history of onset, the urogenital tract must always be examined. The polyarticular form of arthritis may be confused with a rheumatoid arthritis which it much resembles, though this latter condition starts more insidiously and symptoms may affect first one joint and then another.

The prognosis of a gonococcal arthritis is uncertain as regards the ultimate function of the affected joint. In the polyarticular form a considerable degree of permanent stiffness results, especially when the joints of the spine are involved. The polyarticular variety is sometimes only one sign of a general systemic infection with gonococci.

*Treatment.*—Immediate treatment of the urethral infection must be instituted, if this has not already commenced. With rest in bed, adequate treatment of the original site of infection and chemotherapy, the acute pain in the joint begins to subside. Local treatment, except by splintage to keep the joint at rest and prevent deformities, has little effect. As soon as the acute pain has subsided the joint is treated with radiant heat, and every effort made to encourage active movement, for only thereby can function be preserved. A fibrous ankylosis, unfortunately, is only too common a sequela of gonococcal arthritis.

Should suppuration occur, bony ankylosis will result. Any attempts at forcible manipulation to obtain movement after the condition has settled down are to be discouraged, for they are likely to be followed by a recurrence of swelling and pain, the joint becoming more stiff than before. Treatment by autogenous vaccines is sometimes of assistance in gonococcal arthritis, but their value is difficult to assess.

## TUBERCULOUS DISEASE OF JOINTS

The infection of a joint by the tubercle bacillus, whilst not so common as it was fifty years ago, is still sufficiently frequent to require very serious attention. It is essentially a disease of the young, being especially common before the age of 5 years, but it may develop throughout the period of adolescence. In the adult it is a comparatively rare condition. Both sexes are about equally affected and, whilst it is not hereditary, some individuals are certainly more susceptible to infection than others. Bad hygienic conditions may contribute to a lowering of the general resistance of the patient and hence this disease is more common in the less well-to-do members of the population. Whilst injury is not a direct cause, trauma may temporarily lower the resistance of the injured part and provide suitable conditions for the bacillus to develop and multiply.

*Pathology.*—The tubercle bacillus, which has been lying in the mesenteric or thoracic lymphatic glands, passes by the blood stream to the joint, having gained entrance originally via the alimentary canal or tonsils. Children with tuberculous disease of a joint seldom

have pulmonary disease, save as a terminal complication. A tuberculous arthritis may commence either in :—

1. **The Synovia.**—When infection occurs in this membrane it has usually been present for some time before symptoms commence. The synovia becomes congested, thickened and œdematous. Tubercles are scattered over it just below the serous membrane, through which they show as small white spots. Some of these fuse together, after a short time undergo caseation and, ulcerating through the serous membrane, leave small ulcers, which permit the underlying disease to communicate directly with the joint cavity.

2. **The Bone.**—Infection may enter the joint from a pre-existing tuberculous focus in one of the bones, either in the epiphysis or in the metaphysis close to the epiphyseal cartilage (Fig. 561). By whichever route the joint is attacked the prognosis is the same.

Once the joint has been infected the synovial membrane becomes very much thickened, and tiny tuberculous ulcers can be seen scattered over its surface. Granulation tissue forms in it and this begins to invade the other structures of the joint. It slowly spreads on to the articular cartilage, which it first softens and then erodes. Once this has occurred at any one place the tuberculous granulation tissue very rapidly spreads beneath the cartilage and, lifting it up, separates it from the underlying bone. A well-marked *pannus* can be seen growing over the articular surface of the cartilage in the early stage, having spread there from the synovial membrane. As a consequence of this stripping up of articular cartilage the underlying bone is exposed and a tuberculous osteitis develops. The cavities in the cancellous bone which result are first filled with granulation tissue and caseous matter, and then caries of the bone takes place. When the articular cartilage and ligaments have been destroyed pathological dislocation is apt to occur, especially in the hip and knee, if these are not adequately splinted in a correct position during the stage of destruction, which may last many months or even years.

Should the arthritis progress, an abscess is likely to form and either remain within the joint itself or spread outside the capsule and track among the surrounding muscles. Such abscesses are liable ultimately to burst through the skin and form sinuses, which are lined with typical tuberculous granulation tissue and which may become secondarily infected with pyogenic organisms. A tuberculous arthritis under ordinary conditions seldom terminates in bony ankylosis. Repair takes place by the formation of a close fibrous union between the damaged articular surfaces, a bony ankylosis being the result of a secondary pyogenic infection superimposed upon the tuberculous arthritis. At any stage in tuberculous arthritis the patient's resistance is liable to break down and a general infection to occur, when death takes place from miliary tuberculosis. In children the usual manifestation of such a general infection is meningitis.

In disease of the shoulder joint in adults the bone infection may be a quiet process with few symptoms, leading to destruction of part of the upper end of the humerus, a condition known as **Caries Sicca** (Fig. 567). Tuberculous periostitis is seen in association with disease

of certain joints, notably at the lower end of the humerus and upper end of the ulna in an arthritis of the elbow joint.

*Clinical History.*—The onset of a tuberculous arthritis, except in exceptional cases, is very insidious. Attention may be called to the joint by some aching in it after use, or by a limp which is observed by the parents. The child may have been noticed to be off colour for a few weeks without any definite complaint being made. Limitation of movement *in all directions*, the result of an involuntary muscle spasm, is the earliest and most constant sign, and the affected joint is held in the position of greatest comfort, which varies for each individual articulation. Swelling around the joint is apparent in the knee, elbow and wrist at an early stage, but in the hip and shoulder it is difficult to observe owing to the greater quantity of surrounding muscular tissue.

This swelling is due to inflammation of the synovial membrane, and only in exceptional cases is there sufficient excess of fluid in the joint to be appreciated on clinical examination. The joint will feel hotter than the corresponding one on the other side of the body.

Pain is not an important symptom in the early stages of a tuberculous arthritis, though it is often pronounced when an abscess is present under tension, or erosion of the cartilage has occurred rapidly. It may also be present at night in the form of "night cries," which are explained by the fact that as the patient drops off to sleep the muscles, which have been in spasm, at once relax and the inflamed surfaces by being rubbed together again stimulate the return of muscle spasm, which wakes the patient with a cry. Muscular wasting is obvious at a very early stage.

The *diagnosis* is by no means always easy, especially in the early stages, but as a general working rule any subacute arthritis with an insidious onset in a child should be regarded as tuberculous until the contrary is proved, and if this rule is followed faithfully very few errors will be made. Disease in the bone, although not involving the joint, may give rise to symptoms which are similar to those of an arthritis. In the knee Clutton's joints may lead to error, especially if the second knee has not become swollen. X-ray examination of a suspected joint will in the early condition reveal little. It is only when articular cartilage has been destroyed and there is destruction of bone that changes will be shown in an X-ray. Thus the diagnosis must be made in the early stage on clinical signs alone. In certain joints where a definite diagnosis cannot be made otherwise, it is desirable to perform an arthrotomy in order that a specimen of the synovial membrane may be removed for histological examination.

*Prognosis.*—As regards life, the prognosis depends upon the patient being treated under good conditions and the general resistance to the disease improved, but even so there is a mortality whilst under treatment of between 5 and 10 per cent. Patients in whom treatment is commenced early are more likely to respond favourably than others; nevertheless, early treatment of the local lesion does not prevent the disease in the joint progressing. However early a tuberculous arthritis of the hip or knee is treated, the disease under any conditions goes through its cycle of activity with tissue destruction, gradual repair

and finally quiescence, for, except in a few instances, the disease can never be said to be cured, but only rendered quiescent. For all practical purposes this is as good as a cure, but there always remains the possibility of a recrudescence of the disease.

Whilst the prognosis of a tuberculous arthritis varies enormously with different patients and different joints, in general it is bad if more than one large joint is the site of disease, whilst if several small joints are involved the prognosis is usually good.

*Complications.*—1. **Abscess Formation.**—This is an indication of the activity of the disease. The more virulent the infection and the more active the destruction of bone the more likely is abscess formation to take place. It may develop quietly without any symptoms and not be noticed until of considerable size. There is no heat or redness and the skin is involved over it at a late stage. Such an abscess is painful only if it develops rapidly before the joint capsule has had time to soften and stretch, the sudden rise of tension within the capsule being responsible for the pain.

2. **Sinus Formation** results from an abscess either bursting, being incised or repeatedly aspirated. In the old days sinus formation was always looked upon as a serious complication, in that it was certain to become infected with pyogenic organisms, as a result of which the patient would ultimately die of lardaceous disease. At the present day under proper conditions of treatment this risk is very slight, and when the underlying focus of disease has been rendered quiescent, the discharge ceases and the sinus heals up, although this may require several months of treatment.

3. **Generalised Tuberculosis.**—At any stage of the disease the resistance of the patient is liable to break down and a general infection to occur. If this should happen the patient generally dies from a tuberculous meningitis, the onset of which is heralded by a sudden rise of temperature, without any obvious cause, accompanied by headache and pain in the back.

*Treatment*—1. **General.**—In all patients suffering from surgical tuberculosis it must be remembered that the local lesion is only one manifestation of the disease. Although there may be no other signs which can be identified, infection is present elsewhere either in the mediastinal or abdominal lymphatic glands. Pulmonary phthisis is rarely seen in association with a tuberculous arthritis, except in elderly patients or as a terminal condition in children, when it is only one manifestation of a generalised infection.

The object of treatment must therefore be to assist patients to build up their general resistance so that they may be able to cope with the local disease.

All patients with surgical tuberculosis should be treated where possible in the fresh air, and it has long been generally recognised that they improve when removed to the country. The value of sunlight is difficult to assess. It is probable that it does good principally in that it increases the feeling of well-being, as it does in the normal individual. It is unlikely that it has any specific action upon the tuberculous process.



The diet should be a good mixed one which patients enjoy, but there should be no effort to force them to take more than they desire. Living and sleeping in the open air will rapidly develop a good healthy appetite.

**2. Local Treatment—A. Conservative.**—The main principle to be observed is *rest* with fixation of the diseased joint either by splints or plaster until all signs of active disease have completely settled down. The actual length of treatment will vary with each joint and depends very largely on the general condition of the patient. If there is to be any chance of success, fixation for a lengthy period is all-important and any attempt to reduce the period merely courts a recurrence of activity. During treatment care must be taken to watch for the appearance of an abscess, which may develop to a large size without having revealed any sign of its presence.

When the activity of the local lesion is settling down, the general condition of the patient will commence to improve. The child will put on weight and take a more lively interest in its toys and life in general.

**B. Operative Treatment.**—It is now generally recognised that surgical measures are not desirable in the routine treatment of an active tuberculous arthritis, especially in children, on account of the danger of increasing its virulence. Surgery has nevertheless a useful part to play in treatment, provided that its limitations are recognised. It used to be thought that by surgical methods the period of conservative treatment might be shortened, but it is appreciated now that this is not so in practice. In a few instances during the active stage it may be necessary to remove a sequestrum which has formed in the end of a bone, before the sinus leading down to it will heal and the bone disease become quiescent. Before any other kind of operation is performed a prolonged period of conservative treatment must be given a trial. The main principle underlying any operation is the splinting by internal fixation of a joint which has already been damaged by disease.

**Arthrodesis** or fixation of a joint is an operation which aims at the removal of any infected tissue together with the articular surfaces so as to provide two raw bone-ends which will fuse together with osseous tissue and thus prevent any further movement taking place. This operation is chiefly indicated in the hip and knee in children where conservative treatment has failed. In the hip such an arthrodesis is not usually done at the present day, because it generally fails to achieve the desired end, namely, a bony ankylosis; therefore, the operation of so-called *extra-articular* arthrodesis is preferred, the joint being fixed by means of a bone graft placed in close relationship to the joint, a procedure which does not require any interference with the diseased tissue itself or, at any rate, to only a minimal degree. The older operation of **excision** of the joint, whilst it may still be performed under suitable conditions in adults, has been given up in children on account of the danger of damaging the epiphyseal cartilage, thereby interfering with the growth of the limb.

**Amputation** is called for in those patients in whom other measures have failed. Seldom does such a need arise in children, but in adults

amputation has a definite place in the treatment of tuberculous arthritis. It is impossible to lay down any general rules for deciding when it is called for, and each case must be judged on its own merits.

## TUBERCULOUS ARTHRITIS OF SPECIAL JOINTS

### THE HIP

Tuberculous disease of the hip joint commences as frequently in the synovial membrane as in the bone. In the latter it may start either in (1) the upper lip of the acetabulum, (2) the epiphysis of the head or (3) the neck of the femur (Fig. 561). Wherever it starts the disease sooner or later, in spite of treatment, involves the other structures of the joint. If not treated adequately progress is rapid and marked. As with other joints the hip is more commonly affected in children than in adults, but in the latter the prognosis is more serious both as regards the local lesion and the general health of the patient.



FIG. 561

An early tuberculous focus in neck of the right femur, which has not yet affected the joint.

*Clinical History.*—Parents commonly come for advice, having noticed that their child has been less active than normally, has been observed to limp, and tires easily. Pain, except in rare instances when abscess

formation is early and rapid, is not a prominent feature, but when present it may be complained of either in the hip itself or on the inner side of the knee. Whilst the history of some injury can often be elicited, rarely do the parents associate the onset of the limp with this until they are questioned.

Tuberculous arthritis of the hip may be divided into two stages :—

**First Stage.**—The hip is held with the thigh in flexion, abduction and external rotation and in consequence the leg on the affected side appears to be longer than that on the sound side (Fig. 562). The child stands with a lumbar lordosis to compensate for the fixed flexion of the joint. The true position, in which the joint is fixed, can be demonstrated by laying the child on its back and flexing the sound thigh upon the abdomen, thus obliterating the lumbar lordosis, when the affected thigh will be raised from the bed and the true angle of fixed flexion of the diseased hip can be demonstrated. The movements of the hip joint are limited in *every direction*, and in the really acute stage movement is completely abolished. This is the direct result of an involuntary muscular spasm. Muscular wasting is seen very early and can be demonstrated most easily in the gluteal muscles,





FIG. 562

A, front view of a boy with tuberculosis of the right hip. Note the abduction and eversion and the apparent lengthening of the leg.  
B, a lateral view of the same child showing the flexion of the hip.

where the buttock is flattened and the normal gluteal fold is lost. Little swelling is to be noticed around the joint and, though occasion-

ally an abscess may already be present, it is impossible to demonstrate its presence whilst confined within the capsule and until it reaches an appreciable size. In such an early case as this an X-ray will reveal either nothing abnormal or at the most only slight decalcification of all the bones entering into the formation of the joint, while the obturator foramen looks smaller than its fellow (Fig. 563). This sign is the result of muscular spasm rotating the pelvic bones, and indicates that the disease

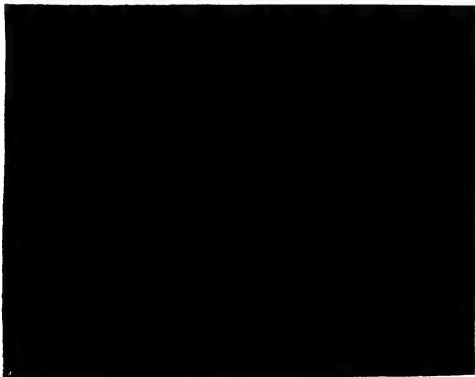


FIG. 563

X-ray illustrating the early changes in a tuberculous hip (left), as described in the text.

has been present for some time. As pathological changes progress, if the condition remains untreated, there may be no other

signs, but in a number of patients pain increases and there are "night cries."

**Second Stage.**—In this stage damage has been done to the joint and the position of the thigh has altered. The hip is now held in flexion, adduction and internal rotation, the degree of flexion being more marked than before. No very satisfactory explanation to account for this alteration in position can be offered. It is usually supposed to be due to stretching of the capsule with destruction of bone, allowing the femoral head to sublunate backwards and upwards, and thereby permitting the adductor muscles (which are the strongest) to pull the thigh over into the position of adduction and internal rotation (Fig. 564). With the hip in this position the limb will now be found to have apparent shortening in contrast to the apparent lengthening, which was present in the first stage. The amount of true shortening will depend upon the degree of bone destruction and (if appreciable) the great trochanter will be found to be raised above Nélaton's line.



FIG. 564

Advanced tuberculous disease of the left hip, the limb being adducted. There is great destruction of bone and an abscess cavity is well defined by its mottled calcification.

the gluteal region, or occasionally it is found on the inner side of the femoral vessels in the adductor region. When the disease commences in the acetabulum, an abscess may form inside the pelvis as a result of perforation of its floor by tuberculous granulations, but such an occurrence is rare. The final condition of the joint will depend upon the amount of destruction of bone and the position of the limb whilst under treatment. If no fixation is employed the diseased bone will be damaged by movement, for the osteitis so softens it that pressure adds to its destruction and the remnant of the head of the femur becomes completely dislocated backwards upon the dorsum ilii. This condition, which should not be allowed to occur, leads to great difficulty in treatment.

**Diagnosis.**—The diagnosis of a tuberculous hip in the early stages may be most difficult. From its clinical signs the only possible diagnosis is that of an arthritis of the hip joint. Since the commonest form of subacute or chronic arthritis of the hip joint in children is tuberculous, such a condition in a child should always be regarded

as such until the contrary is proved. A child of the age, when surgical tuberculous lesions are common, is liable to minor strains or injuries and to develop a limp associated with muscular spasm. This is a mild **traumatic synovitis**, which clears up within a week or ten days with rest in bed.

There are also a certain number of cases of mild **arthritis of unknown origin** in children which clear up completely with rest, though such treatment may need to be continued for several months. These hip conditions cannot be differentiated from a tuberculous arthritis in the early stages. They all have the same signs and symptoms. The only way to arrive at a correct diagnosis is to treat them as though they were tuberculous, until all signs of arthritis have completely subsided. Then, if with increasing activity there is no recurrence of the signs and symptoms, the arthritis is not tuberculous, for if it were, increased activity would soon bring about a return of muscular spasm with resultant loss of movement, evidence of the persistence of the arthritis. **Pseudocoxalgia** should be differentiated from a tuberculous arthritis in most cases with ease. It occurs at an age period somewhat later than a tuberculous hip. Also in pseudocoxalgia, whilst movement is limited by muscle spasm, it is not limited in all directions. Flexion is nearly full in range, whilst abduction in flexion is almost completely absent. So also is muscular wasting, while a positive Trendelenburg's sign is usually present. The X-ray pictures in the early stages may be a little difficult to differentiate, but in the ordinary way typical changes are present in the head and neck, and the diffuse atrophy of the bones, which is present in a tuberculous arthritis, is absent in pseudocoxalgia. The presence of **spinal caries**, which gives rise to spasm of the psoas muscle producing a flexion deformity of the hip joint, may lead to a mistake in diagnosis.

The *prognosis* is uncertain as regards both the joint itself and also life. As with any other tuberculous lesion there is a definite mortality—between 5 and 10 per cent. With regard to the diseased joint it can truthfully be said that early and efficient treatment will give a better result than if the disease is permitted to progress for weeks or months without treatment. But however early treatment starts it will not prevent further damage being done to the structure of the joint, which goes through its cycle of inflammation, destruction of tissue and attempted repair, however early and perfect treatment may be. A child with tuberculous disease of the hip never recovers any useful range of movement. The usual result when the disease is quiescent is a close fibrous ankylosis. Such an ankylosis is unable to resist all the strains and stresses which subsequently fall upon it when activity is resumed, and the thigh in course of time, often in a few months, becomes flexed, adducted and internally rotated, although at the end of treatment it may have been a little externally rotated and abducted.

*Treatment.*—Rest in recumbency, with adequate fixation of the diseased joint in slight flexion and abduction is the underlying principle of all methods, and these must be continued uninterruptedly for a minimum period of two years, whilst considerably longer is necessary

in certain patients. The particular method of fixation employed does not much matter so long as those treating the patient are thoroughly familiar with it. The Robert Jones's abduction frame (Fig. 565) provides the most perfect fixation possible, whilst at the same time allowing the joint to be under observation. The child is kept on this frame until all signs of active disease have completely subsided, all muscle spasm has disappeared and X-ray shows that destruction of bone has ceased and that the affected bones have recovered their density. This may not occur until the end of the third year or even longer, though generally

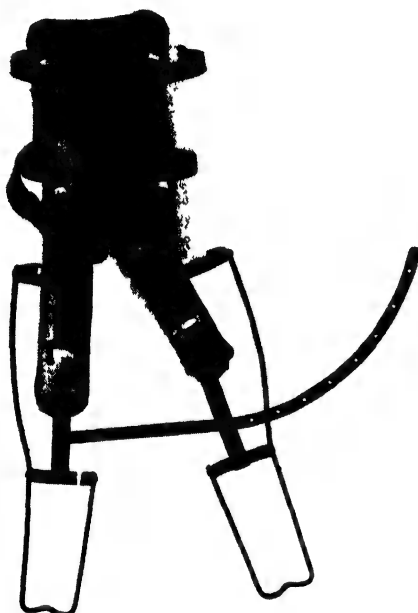


FIG. 565

A Robert Jones's abduction frame.

two years is sufficient. The child having been removed from its frame, a plaster spica, moulded well in to fix the pelvis, is applied to the affected hip down to, but not including, the knee. The child is permitted a certain amount of freedom in bed and later is allowed up, walking in this plaster with crutches. If all goes well, these are gradually given up and active walking in the plaster is permitted. This is continued for a period of another twelve months. If at the end of this time there are no signs of active disease, either clinically or radiologically, the child is allowed to dispense with retentive apparatus and is kept under close observation, X-rays being taken from time to time. Unfortunately, in the majority of patients this does not end the story. The child has been left with an unsound fibrous ankylosis and, although the hip

joint at the end of treatment may be apparently fixed in a position which is functionally satisfactory, *i.e.*, slightly flexed and abducted, within the space of one or two years it becomes more flexed and adducted with some internal rotation, whilst in a certain number a recrudescence of the disease occurs, the most common sign being the development of an abscess. In an endeavour to avoid this troublesome sequela, it is becoming the custom to fix the hip by an extra-articular arthrodesis performed when no signs of active disease are present, the graft being taken either from the tibia, femur or outer wall of the ilium. The joint is then fixed in a short plaster spica which is kept on until the X-ray shows that consolidation has taken place. *It must be clearly understood that this operation is not intended to replace the conservative treatment previously described.* Its sole function is to fix a joint which, having been already damaged by disease, is

no longer capable of standing up to the strains which it may be called upon to bear in the ordinary wear and tear of life.

*Abscess Formation.*—The development of an abscess in a case of tuberculous arthritis is an indication of the activity of the disease. Treatment is necessary as, if pus is permitted to track among the muscles of the thigh, a tuberculous myositis results and this makes it very difficult to obtain any improvement in the joint condition (Fig. 564). If the contents of the abscess are liquid, it may be treated by repeated aspirations under careful aseptic precautions. If, however, the abscess is filled with thick caseous material, this will not pass through the needle when aspiration is attempted. In such a condition various fluids have been injected into the abscess with the object of liquefying the caseous contents, so that subsequently they may be aspirated. It is, however, much more satisfactory to incise the skin over the abscess, remove the mass of caseous material under strict aseptic precautions and suture the skin carefully afterwards. Many abscesses treated in this way heal by first intention and do not re-form. Those which do break down form a sinus discharging tuberculous pus, and this continues to leak out until all signs of active disease in the joint have entirely settled down, when the sinus will heal up rapidly. Under good conditions in a country hospital these abscesses, even if opened, do not become infected with pyogenic organisms, as was the experience of surgeons in the large general hospitals thirty or more years ago.

When a tuberculous arthritis of the hip has been neglected, or if symptoms have never been serious enough to call for treatment (and this does happen), the joint is found to be fixed in about 90 degrees flexion with marked adduction and internal rotation, and a great amount of apparent shortening.

Under these circumstances it is desirable to perform an osteotomy below the trochanters and to fix the limb in the best functional position. About 30 degrees of flexion are necessary to enable the patient to sit down in comfort and a few degrees of abduction to compensate for the true shortening that is present. Too wide an angle of abduction, especially if combined with flexion of the hip joint, will result in an ugly gait.

Tuberculous disease of this joint in an adult offers quite a different problem. The onset is always more acute and the patient's power of resistance is poor in most instances. Treatment by recumbency with fixation is just as important in the adult in the early stage, but the disease seldom is less likely to settle down except with considerable destruction of bone, and upon occasions excision of the damaged head of the femur is required. The prognosis of an acute tuberculous hip in an adult is poor both as regards the joint and the duration of life.

## THE KNEE

Tuberculous disease of the knee in the vast majority of patients starts in the synovial membrane and only at a later stage affects the articular cartilage and bone by direct spread. As in the hip, treatment

does not prevent the spread of the disease, although it may delay its progress. When the disease begins in bone, it does so generally in the metaphysis, and infection spreads to the joint only after an interval. There may be present a sympathetic synovitis which will subside completely if the bone abscess is incised and drained in time.

*Signs and Symptoms.*—The patient complains of swelling or stiffness of the joint, and also that the knee has become bent, so that he is no longer able to straighten the limb and has to walk on his toes.

Pain is a variable symptom and is not a prominent feature in most knees. When a young infant a few months old develops a tuberculous knee, pain may be marked, so much so that it may appear to be a pyogenic arthritis.

Thickening of the synovial membrane is sometimes very marked and its attachments to the margin of the articular cartilage on the lower end of the femur are tender. In young infants the effusion is often great, but in older children and adults there is but little excess of fluid in the joint.

Muscular wasting, especially of the vastus internus, is present, whilst the hamstring muscles are in spasm. In the majority of tuberculous knees the X-ray examination reveals nothing except a general decalcification of the bones. Only after the disease has been present for many months or, if treated, for some years, does it reveal any signs of caries or erosion of bone. In young children the lateral X-rays of the two knee joints on comparison in the early stages will show that the patella on the affected side has started to ossify earlier than that on the sound side and that its ossification has proceeded more rapidly. This X-ray appearance is practically diagnostic of a tuberculous arthritis, starting in the synovial membrane. When a tuberculous knee has been neglected a triple deformity occurs in consequence of the destruction of the joint surfaces and ligaments, and as a result of the spasm of the hamstring muscles. The joint becomes markedly flexed, the tibia subluxated backwards and externally rotated. Such a deformity is seldom seen at the present day, but if attention is not paid to the method of fixation during treatment it is possible for the tibia to rotate externally and subluxate backwards unobserved.

*Diagnosis.*—Any chronic synovitis of the knee in a child with little pain but muscular wasting should be regarded as tuberculous until the contrary is proved. The absence of pain might lead one to consider the possibility of it being a Clutton's knee, a condition which can be excluded by the absence of muscular spasm, and by the presence of more effusion than is usual in a tuberculous knee, other congenital syphilitic stigmata and a positive Wassermann test. An abscess in the lower end of the femur or upper end of the tibia, either tuberculous or pyogenic, may be associated with a sympathetic effusion into the joint. An X-ray will reveal the presence of such a lesion. There occur in children a few cases of chronic synovitis which clinically are indistinguishable from a tuberculous knee. It is therefore justifiable, if no other means will settle the correct diagnosis, to do an arthrotomy and remove a specimen of the synovial membrane for

histological examination. If the condition is tuberculous the diagnosis can be definitely established, whilst it is known that these other cases of chronic synovitis of unknown origin clear up completely after an exploratory arthrotomy. Every now and again a patient will be seen in whom a chronic synovitis is associated with a gummatous osteitis, and therefore it is wise in all children with a chronic arthritis of the knee joint to have a Wassermann test done as a routine.

A chronic effusion into the joint is often the accompaniment of a sarcoma of the lower end of the femur, but the history of pain associated with a sarcoma and the X-ray appearance should reveal its true nature. In an adult the diagnosis from a villous arthritis may be very difficult, especially as it sometimes happens that this joint is the only one affected. An arthrotomy may be the only method by which the diagnosis can be made.

A gummatous synovitis is in many respects similar to an early tuberculous arthritis in the adult and, as in the child, a Wassermann test as a routine is indicated. Pain is a more prominent symptom in the adult with a tuberculous arthritis than in the child, because the destruction of the joint surfaces occurs more rapidly in the former.

*Treatment.*—Rest combined with fixation in a Thomas' knee splint is essential in every patient during the acute stage. Such immobilisation must be combined with a fixed extension, for a weight and pulley allow movement at the joint. If pain is a prominent feature it rapidly subsides with adequate extension and fixation. Rest in bed must be continued until all signs of activity have been absent for at least six months. Then the patient can gradually be allowed up in a walking caliper (Fig. 566), and even if no signs of activity return he must retain this for at least two to three years. Abscess formation is rarely seen in a tuberculous knee. The usual history in a child after conservative treatment is that for perhaps a year or more the disease remains quiescent and that then symptoms recur. After a further period of conservative treatment the condition settles down only to recur again at a later date. By this time the X-ray, which may never have revealed anything definite before, except for enlargement of the patella and a diffuse bone atrophy, will begin to show erosion of the ends of the bones. It has been found that sooner or later all tuberculous knees with very few exceptions require an arthrodesis, which in children can be done without interfering with the growth of the limb by removing only the articular cartilage on the femur, tibia and patella together with the diseased synovial membrane and any remnants of the ligaments.

The limb is then fixed in plaster and after a few weeks the child is



FIG. 566  
A Thomas' walking  
caliper splint.



allowed to bear weight on it. Bony ankylosis will occur, but it may take as long as two years, during which period some form of fixation is called for, as otherwise a fibrous and not a bony ankylosis will result and the knee gradually bend. Once a firm osseous union has taken place the patient is unlikely to have any further trouble.

The treatment of a tuberculous knee in an adult depends very much upon the age of the patient. It is always a more virulent disease than in a child and no surgical methods must be employed which aim at fixation of the joint, while this activity persists. In young adults the same treatment as for children is called for until the knee is quiescent, and then an arthrodesis or excision of the diseased joint should be done. Fixation in plaster or a splint must be continued until bony ankylosis is firm. If an arthrodesis or excision is performed during the active stage, it is probable that the disease will become more active still and multiple sinuses develop, a condition which may necessitate amputation as a life-saving measure.

When a tuberculous knee develops in an adult over the age of forty years, the only satisfactory treatment is an amputation through the middle third of the thigh well away from the disease. People of this age seem to have lost the power of local resistance and any attempt at arthrodesis is likely to fail. Once the diseased limb has been removed the general health of the patient rapidly improves, and he is infinitely better off with a good artificial limb.

In those cases in which a tuberculous osteitis is associated with a sympathetic synovitis of the knee joint, the focus in the bone must be explored and curetted out as soon as it is diagnosed, the incision afterwards being closed. It may heal by primary union or, at the worst, a sinus develops which discharges some thin pus for a few weeks before finally healing up. Left untreated such an osteitis always spreads ultimately to the joint cavity, whereas with early drainage of the bone abscess the joint can be saved and the sympathetic effusion will rapidly subside.

### THE ANKLE

Disease of this joint in most instances begins in the synovial membrane: very rarely does it originate from an osseous focus. The joint becomes stiff and painful. Swelling, which is marked, shows itself on the posterior aspect of the joint on either side of the tendo achillis. Muscular spasm holds the foot in a position of equinus and in a child the development of a limp is often the first sign that may be noticed. The joint surfaces become involved more rapidly than they do in the knee and therefore pain is complained of earlier. In the adult an X-ray will in most instances reveal destruction of articular cartilage by the time advice is first sought. Abscess formation is very common both in children and adults.

*Treatment.*—In the child fixation in plaster without any weight-bearing, if continued for a sufficiently long period, results in a firm fibrous ankylosis. If an abscess forms, in spite of aspiration, it usually breaks down with the formation of a sinus, which heals in time without any difficulty. Such an ankle joint is liable in young adult



life to be a source of further trouble, from a recurrence of activity. When a tuberculous arthritis develops in an adult conservative treatment gives very poor results. Excision of the joint is liable to be followed by increased activity of the disease and in most patients the only satisfactory treatment is an amputation through the tibia, 7 in. below the knee joint; certainly in any adult over the age of 30 years this is the only treatment of any value, though many will hesitate to accept such advice and prefer to try conservative treatment for a year or more.

### TARSAL JOINTS

Infection of these joints is the result of an osteitis of one of the tarsal bones, which spreads infection direct into the neighbouring joints. This is very rare in adults.

*Treatment* consists in fixation in plaster until all signs of active disease have settled down. In nearly 100 per cent. of cases an abscess develops, and as long as there is any osseous disease the sinus which results will continue to discharge. At times a carious sequestrum will form in one of the tarsal bones and necessitate removal by operation before the joint condition will clear up.

### THE SHOULDER

Disease of this joint is seldom seen in children. It is usually secondary to a tuberculous osteitis of the upper end of the humerus from which the joint becomes infected. In young adults it occurs quite often when active pulmonary disease is also present. In these cases pain is not a prominent feature, but there is loss of movement and a considerable amount of swelling. Muscular wasting of the deltoid especially is marked. The pain, if present, may be complained of either in the joint itself or down the front and outer side of the arm, leading the patient to believe that he is suffering from a neuritis. In later life a particular form of tubercle known as *caries sicca* is seen in the upper end of the humerus, and the joint is generally involved by the time advice is sought (Fig. 567). Symptoms are slight, limitation of movement being the most common with aching down the arm, this having often been present for some time. In the young adult in most instances an abscess develops, and in the older patient also this may occur. It may reveal itself either at the anterior or posterior fold of the axilla, but most often tracks down along the tendon of the biceps and appears in the upper part of the arm below the tendon of the pectoralis major, forming a fluctuating swelling



FIG. 567

*Caries sicca* of the left shoulder joint.

above the muscular belly of the biceps. X-ray examination in the adult will generally show destruction of bone, whilst in the older patient with caries sicca an osteitis of the head of the humerus with the formation of small sequestra is revealed. Disease beginning in the tuberosity, if identified in time, may be dealt with by surgical means and the joint saved from infection, but this unfortunately is seldom practicable.

*Treatment* consists in fixation of the shoulder in abduction to 90 degrees and slightly in front of the coronal plane of the body, either in plaster or upon an abduction splint. Any abscess which forms must be aspirated repeatedly, but many of them burst through the skin and form sinuses which may take several months to heal. If the disease in spite of adequate fixation shows no sign of settling down, a formal excision of the joint, removing all the diseased tissue, may be desirable, combined with fixation for a long period afterwards. Disease of this joint in young adults is so often associated with active pulmonary disease that the prognosis is correspondingly poor.



FIG. 568

Bones of the arm showing extreme rarefaction and destruction of the elbow joint. In the actual specimen the bones are translucent.

### THE ELBOW

This joint is principally involved in young children, the disease starting either in the synovial membrane or the upper end of the ulna. It seldom arises either in the head of the radius or the lower end of the humerus. The joint becomes swollen, fusiform in outline and painful, while movement is almost entirely abolished by muscle spasm. The grooves on either side of the tendon of the biceps are obliterated and the swelling of the joint is made more obvious by the amount of muscular wasting. Abscess formation occurs in nearly every

patient, showing itself either on the inner or outer aspect of the joint. Although aspirated repeatedly these abscesses usually break down leaving a sinus, which takes a little time to heal. X-ray shows destruction of the joint (Fig. 568), combined with periostitis of the lower end of the humerus and upper end of the ulna, which latter is especially well marked.

*Treatment.*—The joint must be fixed in flexion by slinging the forearm in a collar and cuff, or if this is not sufficient to relieve symptoms, in plaster. Treated thus in children the prognosis is good both as regards time and function, for a certain amount of movement in the joint is ultimately obtained. In adults excision of the joint may be desirable, but it should never be done until conservative treatment has had a good trial and been found wanting.

## THE WRIST

Disease of this joint is much more common in elderly patients than in children or young adults. Starting usually in the synovial membrane the joint slowly becomes puffy, swollen and painful. There is also gross limitation of movements in all direction and the carpal bones after a time become extensively diseased. Pain may or may not be a common feature.

*Treatment.*—Fixation in plaster for several months is necessary, the fingers and thumb being allowed free to move whilst the wrist itself is held in slight dorsiflexion. If damage to the carpal bones is very extensive and does not settle down with fixation alone, it may be necessary to explore the joint through a dorsal incision and remove with a gouge any obviously diseased bone.

## THE SACRO-ILIAC JOINT

Tuberculous disease of this joint is not uncommon, especially in children. It commences in the neighbouring bone of the ilium and is characterised by the paucity of symptoms to which it gives rise. The patient may complain only of having had a tired feeling in the lower part of the back for some weeks or months and upon examination may be found to have an abscess forming either on the posterior or anterior aspect of the joint. In the latter case, unless it is very large and can be felt through the anterior abdominal wall, its presence is revealed only by rectal examination. The patient may walk with a limp, keeping the hip on the affected side flexed, but the most outstanding feature of disease of this joint is the entire absence of any pain until a late stage.

X-ray examination will reveal caries of the ilium in the region of the joint by the time symptoms are severe enough to call the attention of the patient to the back.

The *prognosis* is good in children and uncertain in adults; whilst the latter usually recover from the local lesion they appear very liable to develop another bone lesion at a later date. Fixation on a plaster bed is all that is required in children, but in an adult the joint may need exposure by cutting a window through the posterior portion of the ilium, in order that all the diseased tissue so far as is possible may be removed. The piece of bone which is removed to give access to the diseased joint, if it is healthy, may be employed as a graft which is sunk down into the cavity, left after the diseased bone has been removed. In some patients a sequestrum may form, which needs removal before a sinus will heal. Subsequently, the patient is fixed on a plaster bed until the X-ray shows that the joint has become ankylosed and the surrounding bone has attained a uniform density.

## TUBERCULOUS DISEASE OF THE SPINE

## PATHOLOGY

In common with tuberculous infections of bones and joints elsewhere, **Pott's disease** occurs most often in children under the age of

10 years. Thereafter its frequency diminishes and, though it may



FIG. 569

A specimen illustrating the early changes in the body of a vertebra in Pott's disease (see text).

occurs at any age, it is a comparatively rare disease in the adult. The male is rather more prone than is the female. Injury has no more association with the development of a tuberculous focus in the spine than it has elsewhere. The dorsal and dorsi-lumbar regions are the most common situations for the disease to develop, probably due to the fact that a greater strain falls upon this portion of the spine. The cervical and lumbar regions are involved about equally, but taken together disease in these situations is only half as common as in the dorsal region. The infection of the spine is commonly believed to be due to the bovine bacillus more often than to the human.

The tuberculous process is identical in its development with lesions in other parts of the skeleton. Except in rare instances it



FIG. 570

A small boy showing marked kyphosis in the dorsal region. This is due to the collapse of not more than two vertebræ.



FIG. 571

X-ray illustrating kyphosis due to the complete collapse of one vertebra and the partial collapse of another.

commences in the anterior portion of the body of a vertebra close to the epiphysis (Fig. 569). A periosteal site of infection, as opposed

to this endosteal type, occurs in the adult, but by the time the patient seeks advice it has usually advanced to a stage at which it is impossible to differentiate the two. As the result of the development of the tuberculous focus caries occurs, and the portion of the bone affected becomes softened and breaks down into a caseous mass. The disease may very rapidly involve the intervertebral disc and, having infiltrated this, spread thence to the adjoining vertebra. It may also spread from one vertebra to another beneath the anterior common ligament.

The anterior portions of the two vertebral bodies, becoming softened, under the weight of the trunk collapse slowly and the typical deformity of an angular kyphos is produced by the projection backwards of the spinous processes (Figs. 570 and 571). The acuteness of this kyphos will depend upon how many vertebræ are involved by the disease. It may spread so that several are affected at more or less the same time, and then a more gradual curve results. Especially in children more than one portion of the spine may be affected, and this has been more appreciated since routine X-ray examinations have been carried out during treatment.

Although disease may commence in any portion of the neural arch, in practice it is rare for it to develop in the lamina or spinous process.

In the cervical and lumbar regions a kyphos is not so marked as in the dorsal vertebræ. In the cervical region, in addition to commencing in the bodies of the vertebræ (Fig. 572) the disease occasionally appears in the joints between the atlas and axis, and the bone is affected at a later stage.

An abscess or its remains can be found in all those patients who come to autopsy. When the disease becomes quiescent and repair takes place in the damaged vertebræ, it does so, in the first place, by a fibrous ankylosis. Later, this is replaced by a bony union, which may take several years before it is complete. Little, if any, callus is formed in the healing process after caries of the spine. If the patient has been allowed to resume the upright position before such repair has taken place, and has not been provided with an adequate support there is a grave risk that the kyphos may increase in extent due not always to progression of the disease but simply as a result of further collapse of the damaged vertebræ.



FIG. 572

Tuberculous disease of the 7th cervical and 1st dorsal vertebræ.

The proximity of the spinal cord to the site of disease leads not infrequently to its becoming involved by pressure from an abscess which has tracked posteriorly, or, rarely, from being stretched over the deformed vertebræ in front of it.

### CLINICAL PICTURE

The onset of spinal caries may be very gradual or quite acute, but the former is the more common. In consequence, the history which the parent gives may vary considerably. The child may be brought because it is limping, has pain in its back or abdomen, or as the result of a lump having been noticed in its back whilst being bathed. Probably the appearance of a kyphos is the commonest early manifestation which is noticed in hospital practice. Very rarely the child will be brought up for examination because of weakness of the lower limbs.

In the adult any of these reasons may bring the patient under observation, whilst occasionally a swelling in the abdomen or back, the result of an abscess, attracts notice by its size or pain.

It must therefore be realised that the early stages of the disease, in many patients, give rise to few symptoms, and that, save in a few instances, the disease is already well established before it can be recognised clinically.

**Pain** is not always an early symptom. It is more common for the patient to complain only of having had an indefinite ache in the back which, especially in the adult, is apt to be regarded as a mild lumbago or back-strain.

Pain, when it is present, may be of two kinds. It may be *local* over the site of disease, made worse by any kind of movement, or it may be *referred*, produced by pressure upon or irritation of the nerve trunks as they emerge from the intervertebral foramina. When due to this latter cause pain will vary in distribution according to the portion of the spine affected. In disease of the cervical region it is complained of in one or other arm; in the dorsal sector it will be referred to the abdomen, whilst in the lumbar region it affects the legs and in the adult may be regarded as sciatica.

**Rigidity.**—Stiffness always occurs in tuberculous disease of the spine. In the early and active stages, before the appearance of a kyphos, rigidity is due to a reflex involuntary muscular spasm, which is nature's method of splinting the inflamed portion of the spine. This spasm may be of such a nature, that it is quite obvious upon examination of the spine that the muscles are contracted and that they are preventing any attempt at movement. In disease of the lumbar and cervical regions the muscles may be seen in spasm more readily than in the dorsal spine, whilst in cervical caries the head and neck will be carried a little forwards and held immobile. In children, when muscular spasm is not as marked as this, it may be difficult to demonstrate its presence. To invite the child to put the spine through its range of movement may be impossible on account of its age. The existence of muscular spasm can, however, be elicited by placing the patient on its face upon a couch, then raising the lower

limbs and noticing the degree of extension of the spine which can be obtained and also the lateral mobility. If rigidity of the spine is present movement will be limited.

Alterations of gait are common in spinal caries and are due to the trunk being held rigid by muscular spasm, so that the patient does not twist his spine when looking round or bend it when stooping; instead he flexes the lower limbs in order to do so. In cervical disease a torticollis may develop, or the child may support the chin upon his hand.

In the late stages of the disease rigidity is, of course, due to the fixation of the damaged vertebræ either by a fibrous or bony ankylosis.

**Deformity.**—It has already been stated that the characteristic deformity in a tuberculous spine is an acute angular kyphos. This is the usual case, where only two vertebræ are affected, but when several vertebræ are involved the deformity may be angular, but more rounded. The deformity is more easily recognised in the dorsal region of the spine, owing to the spinous processes being longer and hence protruding relatively further when their bodies have been damaged. In both the lumbar and cervical regions there may be very little actual deformity, only a loss of the normal concavity which exists in these portions of the spine. In the cervical region in children the contraction of the spinal muscles will flex the occiput backwards, and thus the child will appear to have lost its neck.

In patients, in whom many vertebræ in the dorsal region have been damaged and no satisfactory treatment carried out, the sternum is likely to be deformed and the ribs crowded together, the patient becoming the typical "hunchback," a condition seldom seen at the present day.

#### ABSCESS

In practically every patient an abscess develops at some stage of the disease, but it may never be of sufficient size to be recognised as a clinical entity, though it is revealed upon radiographic examination.

An abscess may grow to a large size before it is recognised, since its production is often painless, and it commonly develops even some long time after treatment has been instituted. This is particularly so in adults, and in caries of the lumbar spine the swelling caused by the abscess may, as has already been pointed out, be the reason for the patient first coming for advice.

In children an abscess does not usually occur until a later stage of the disease. Its size and rapidity of development, as in all other tuberculous lesions, is an indication of the activity of the disease.

The abscess forms beneath the anterior common ligament and may strip this up for a considerable distance from the bodies of the vertebræ. It may remain localised at the site of disease and be gradually absorbed, or it may increase in size and track in various directions, according to the portion of the spine from which it originates, and so become a clinical feature of the disease.

In the *cervical* region a **retropharyngeal** abscess appears in one of two situations. It may show itself behind the posterior wall of the

pharynx and, pushing this forward, cause a fluctuating swelling which interferes with swallowing and breathing. In the early stages the posterior wall of the pharynx is freely movable, but unless the tension of the abscess is reduced, it becomes adherent. Instead of accumulating here the abscess may track laterally behind the carotid artery and jugular vein and appear in the posterior triangle of the neck behind the sternomastoid muscle. Very rarely in cervical disease an abscess may, instead of tracking in either of these directions, make its way down either into the mediastinum or into the axilla.

In the *dorsal* region the abscess commences in the same way and, although obvious as a shadow in the X-ray in the posterior mediastinum, rarely becomes a clinical entity. It does so, however, when it extends backwards between the vertebral ends of the ribs to form a **dorsal** abscess. In this region it is more likely than in other situations to track backwards beneath the posterior common ligament and, owing to the smallness of the spinal canal, to produce pressure upon the cord.

In the *lower part of the dorsal and the lumbar region* the pus finds its way into the sheath of the psoas muscle, producing a **psoas** abscess (Fig. 573). It tracks down, forming first an iliac abscess above Poupart's ligament and then travels on behind the femoral vessels to accumulate in the adductor region. It may even go further down the thigh or pass with the internal circumflex vessels behind the femur and present on the outer side of the thigh below the great trochanter. In very rare instances the abscess tracks into the pelvis, appearing again in the gluteal region, or even rupturing into the rectum.

In some patients instead of tracking down the sheath of the psoas muscle it passes backwards between the latissimus dorsi and external oblique muscles, presenting then as a **lumbar** abscess in the triangle of Petit (Fig. 574).

The development of a large abscess is, of course, a serious complication on account of the risk of secondary infection with pyogenic organisms, but under modern conditions of treatment this seldom happens.

#### NERVOUS SYMPTOMS

Though occasionally weakness of the lower limbs may be the first manifestation, this is rare. Nervous symptoms are more likely to develop during very active and extensive disease or when efficient treatment has not been carried out for a sufficient period of time. They result either from an extension backwards of the disease, a mass of granulation tissue or an abscess pressing upon the spinal cord, or in later stages from stretching of the cord over the posterior aspect of the damaged vertebræ. Owing to the size of the spinal canal in the dorsal region, nervous symptoms are more likely to develop in this situation than in the cervical, where there is plenty of room, or in the lumbar region, where the cord has broken up into the cauda equina. Owing to their anterior position the motor tracks are the most likely to be involved.



The typical nervous symptoms are a true spastic paraplegia with increased knee and ankle jerks, ankle clonus and extensor plantar response, whilst incontinence of urine and faeces, either one or both, is usually present.

The onset of these signs of pressure upon the spinal cord are usually very gradual and may develop even whilst the patient is under satisfactory treatment. When this happens the pressure is due to an abscess, and for a time the symptoms will get worse before improvement commences.

Such symptoms are very often seen in patients who have a

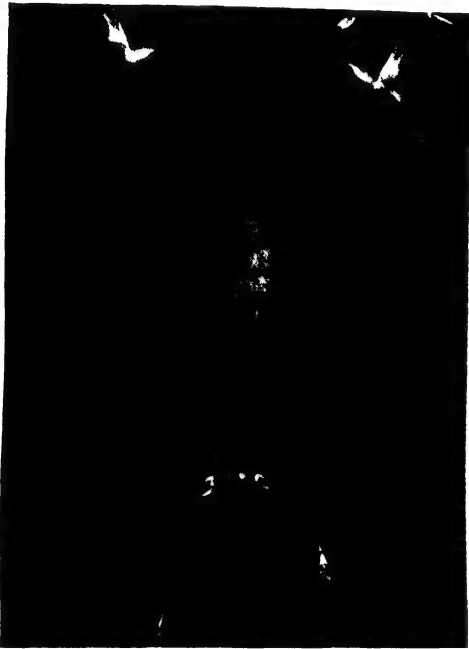


FIG. 573

Extensive dorsi-lumbar tuberculosis of the spine. The calcified outline of a large psoas abscess can be seen on the left side and a slight indication of a similar condition on the right.



FIG. 574

A large lumbar abscess coming to the surface through "Petit's triangle" between the erector spinae and latissimus muscles.

recurrence of the disease, and these seek advice again not on account of the back but because of inability to walk properly.

Occasionally, in addition to manifestations of motor involvement, sensory changes, even to the extent of anæsthesia, may develop in severe and acute cases. Under these conditions the muscular paralysis is flaccid in type and there are diminished and not increased reflexes.

Acute myelitis develops after the signs of pressure upon the cord have been in existence for a considerable time, and results in permanent nervous damage.

*X-ray Examination* —An X-ray of the spine is, of course, essential in every patient with suspected caries, and of the two views the lateral is the more important. In early cases all that can be seen

is perhaps a little mottling of one or more vertebræ, with a diminution in the width of the intervertebral space, showing that some change has taken place in the disc. When the disease is well established the damage to the vertebræ and the disc can be easily seen. The presence of an abscess in the early stages is revealed by a fusiform shadow. Later this can be clearly seen by calcification in its wall.

### PROGNOSIS

Tuberculous disease of the spine is always a serious condition and although the risk as regards life, with adequate treatment, is favourable there still remains a definite mortality.

As in all other bones or joints, even with adequate treatment under the best conditions, the disease usually progresses before the process of repair commences. Because a patient begins treatment without any visible destruction of bone, there is no certainty that appreciable damage will not occur; in fact, some is inevitable. The more acute the onset the more is this likely to occur, and it is always wise to be guarded in prognosis.

The patient who has a well-marked deformity when first examined often does best. The development of an abscess, visible on clinical examination, does not of necessity mean a bad prognosis, but it indicates that the disease is definitely active. In adults the prognosis is not as good as in children, but probably only because they are more liable to lung complications.

Again, the onset of a flaccid paralysis early in the disease is not in itself a bad omen, as it ensures the patient having adequate treatment, but the development of a spastic paralysis whilst under treatment means that this will need to be prolonged and that the lesion is less likely to recover completely.

As in all other tuberculous lesions in children, there is a risk of a miliary tuberculosis developing, and this may occur without warning at any stage of the disease.

### DIAGNOSIS

The diagnosis of spinal caries in a child should be easy because in most patients a deformity has already developed and rigidity is a marked feature. It may, however, not be possible to make a diagnosis at once if no deformity exists. When this occurs it needs to be remembered that any child with stiffness of the spine, with or without pain, should be suspected of caries until the contrary is proved. Often the only way of settling the diagnosis is to treat the child as though it had a tuberculous spine, and watch its progress. If with rest and fixation the symptoms subside and do not return with increased activity, the lesion is almost certainly not a tuberculous spine. Should, however, the rigidity return with activity, even in the presence of a negative X-ray, treatment must be continued.

Other infections of the spine in children are rare, and if they occur give rise to more general disturbance. The differentiation from

scoliosis should give rise to no difficulty, but it does occasionally happen that the side of a vertebra is destroyed more extensively than its anterior aspect, and then it may not be easy without an X-ray to differentiate the conditions.

In the adult diagnosis may be much more difficult. If there is an acute kyphos without any history of injury it may be obvious without the assistance of a skiagram, but often in the adult pain and stiffness exist some time before a kyphos is visible. Here the diagnosis may be difficult, for these symptoms may be due to muscular strains, osteo-arthritis, rheumatoid arthritis or to conditions such as growths or aneurysms pressing upon the spine. Malignant growths, usually secondary, can be differentiated by the history of the primary and by the fact that the pain is more severe and constant, being present at night and when the patient is resting. Syphilitic disease of the spine, either as a gumma or as a complication of tabes, is rare but does occur, and can be differentiated by the presence of other signs. A Charcot's spine develops an acute kyphos very similar to that of a tuberculous spine.

#### TREATMENT

All patients, although they may have no other manifestation of the disease, have other foci and therefore the spine must be regarded as only the local manifestation of a general disease.

Treatment must therefore include both general and local measures.

**General.**—Patients must be treated under the conditions which are most favourable for building up the general resistance to the infection. For this reason they should be moved out into the country away from large cities. Plenty of good simple food is necessary, and it should be varied in detail so as to tempt the appetite. The value of sunshine is much debated, but its chief value is probably the production of that feeling of well-being, which is so essential. Fresh air and cold winds act beneficially by raising the general metabolic rate. Sometimes when a patient is not progressing favourably a change from the country to the seaside will provide the necessary stimulus.

**Local Treatment.**—Rest in recumbency with fixation of the spine is the principle to be followed. There are various means by which this may be achieved, and it does not matter which method is employed provided it is thoroughly understood.

In a child rest is best achieved by some form of frame—either Thomas' or Bradford's or some modification thereof. The child is strapped to the frame, which rests on a bed with large wheels or upon a carriage which can be moved about the grounds. In some children it is advisable during the active stage to fit the lower limbs with extensions to make fixation more secure, and in the cervical and upper dorsal regions a headpiece must be attached to the frame to enable the head also to be immobilised. At intervals it is necessary to examine the spine, and for this purpose a plaster case to fit the front of the child is made (Fig. 575), so that the patient may be turned over with the minimum movement of the diseased spine.

In adults fixation may be achieved in the same way or, if they will not tolerate the frame, a plaster bed is made in which they lie and which is raised upon a low wooden platform, so that no movement occurs during the necessary nursing attention.

In addition to rest and fixation, in the child it is desirable to produce, by hyperextension of the frame, a corrective curve above and below the portion of the spine which is diseased, so that when the erect posture is subsequently resumed there shall be less stress upon the damaged vertebræ. This hyperextension is achieved gradually by bending the frame backwards. It is, of course, not only impossible but also unwise to attempt to straighten out the spine at its diseased segments, as their ultimate fusion is the most satisfactory method of curing the disease.

The period of fixation on a frame varies greatly, but in children a minimum of two years is indicated, even if no abscess develops or no nervous symptoms are manifest. It may be necessary, however, to prolong this period even up to five years or more. Few adults will tolerate fixation in the plaster bed for two years, whilst in the patient of over 50 years of age a period of recumbency of only a few months is often all that can be achieved.



FIG. 575

A girl shown in a carrying or turning plaster, having been taken out of her treatment apparatus for inspection of the spine.

When the general condition of the patient and the skiagram warrant the period of fixation being terminated, some form of support must

be supplied. Of these the Jones's brace or a Taylor support is the most satisfactory, and it must be worn for an indefinite period, certainly until the skiagram shows that the damaged vertebræ have become ankylosed. The child must, when released from its frame, have exercises to develop its spinal muscles, and until these are re-educated it should not be allowed to assume the erect posture. In all it must be expected that the child will be under treatment for three years, even if no complications develop.

In disease of the cervical region a collar must be worn to support the head, one of moulded leather being the most satisfactory.

In consequence of further bending of the spine sometimes taking place even after prolonged treatment, two operations have been practised, the aim of which is to provide a living internal splint. These operations are :—

1. **Albee's Operation.**—The spinous processes of the diseased vertebræ and of two above and below those affected are exposed and split. A large graft is then cut with a motor saw from the tibia and sunk in between the spinous processes. After this operation the patient remains in a plaster bed for at least three months and then gradually gets up with a back brace, which must be worn for a further period of at least a year.

2. **Hibbs' Operation.**—This is more extensive and was particularly

devised for children with disease of the dorsal region. The muscles are dissected off the spinous processes and laminae until the intervertebral joints are exposed. These are opened and the cartilaginous surfaces removed, whilst the laminae and spinous processes are split and turned up and down, forming a large number of small grafts which fuse together.

Patients, upon whom these operations should be performed, require very careful selection and in children there is seldom any reason for their employment. They should really be looked upon as operations to splint a spine which has been damaged by disease and which requires some extra permanent support, such as cannot be given by a brace.

*Treatment of Abscess.*—If this is not increasing in size, not painful or not showing any signs of involving the skin, it can safely be watched, as many are spontaneously absorbed. The exception to this is the chronic retropharyngeal abscess which is likely, if left to itself, to rupture into the pharynx and, even if it cause no other trouble, to become infected.

The great danger of all tuberculous abscesses is that they may be secondarily infected with pyogenic organisms, and for this reason it is wise to treat them by aspiration rather than by incision. In some few instances the abscess must be incised for a variety of reasons, but the skin should always be sutured carefully afterwards. Should a sinus develop after either aspiration or incision the greatest care must be taken to keep it aseptic by cleansing the skin with surgical spirit and then covering it with a sterile dressing. Under the conditions which exist in country hospitals to-day secondary infection of a tuberculous abscess is rare.

**A Chronic Retropharyngeal Abscess** must be aspirated in the neck behind the sternomastoid, and if this does not provide relief it must be incised. Should it rupture into the pharynx it will certainly become infected.

**Dorsal Abscess.**—This abscess can be aspirated very easily and should be repeated when necessary.

Mediastinal abscesses seldom cause any trouble.

**Psoas Abscess.**—This abscess should be aspirated above Poupart's ligament just internal to the anterior superior iliac spine. In spite of this it may continue to accumulate in the adductor region and require aspiration there, in which case it is likely ultimately to involve the skin and form a sinus.

**Lumbar Abscess.**—Aspiration is also indicated, but in adults if it is of great size it is more satisfactory to incise it and suture the skin.

*Treatment of Paraplegia.*—Although the development of paraplegia is a serious complication in a patient with spinal caries, it will be found that, if and when the disease of the spine gets better, the paralysis will likewise improve. This may not be for some months, but if rest with fixation is persisted in the majority of cases clear up, especially if they are of more or less sudden onset. In children the paraplegia recovers, except in those patients in whom there has been a relapse in the local condition or in whom the spine has become bent after walking about for some time. Even in these, if nervous symptoms do

not clear up completely, they seldom persist to the extent of interfering with the patients' ability to walk about.

When it can be established that the paralysis is due to the presence of an abscess and this continues to exert pressure, relief can be obtained by performing a costotransversectomy to enable the abscess to be drained.

Laminectomy should be performed only in those patients in whom the paralysis can be proved to be due to pressure from a ridge of bone. Except under these conditions it does harm and, when it is done, must be combined with a bone graft to splint the spine which has already been damaged anteriorly by disease and is now further weakened by the laminectomy.

### SYPHILITIC DISEASE OF JOINTS

Articular disease produced by syphilis at the present day is uncommon, as are all the late manifestations of syphilis, in consequence of the intensive treatment which, during the last twenty years, has been given for this disease in its early stages. In **congenital syphilis** a diffuse gummatous infiltration of the synovial membrane occurs between the ages of 5 and 15 years. It commonly affects the knees and is known as *Clutton's knee*. One knee generally swells some little time before the other, though both are always affected in spite of treatment. The onset is sudden and the characteristic feature is that of a painless synovitis in a child for no apparent reason. The synovial membrane becomes slightly thickened and there is a large effusion into the knee joint without any limitation of movement, except such as occurs in consequence of the amount of fluid present. There is no muscular spasm or wasting. Other signs of congenital syphilis are present, in particular an abnormal increase in the length of the tibia which is out of proportion to that of the femur. The Wassermann and Kahn tests are usually positive.

*Treatment* consists in the usual antisyphilitic remedies. Even with these the condition of the joint takes a long time to settle down and, if the effusion has been present for a considerable time, a certain amount of laxity of the joint may persist, due to softening and stretching of the joint capsule and ligaments. Occasionally a child with a syphilitic epiphysitis may develop a synovitis of the neighbouring joint.

In **acquired syphilis** the following lesions are seen :—

1. In the later part of the **secondary stage** vague pains in the joint accompanied by synovitis and an effusion may occur. The knee is most often the seat of this condition and in spite of treatment it may persist for a long time.

2. In the **tertiary stage** a gummatous synovitis is occasionally seen, the knee again being the site of such a condition more commonly than other joints. The synovial membrane is thickened and may contain firm nodules. The amount of effusion varies greatly. Pain as a rule is not very marked, and the chief disability results from the considerable degree of muscular wasting which sooner or later accompanies the swelling. In time the stability of the joint is impaired

by stretching of the ligaments. The diagnosis is often obscure, especially when more than one joint is affected. It may under such conditions be difficult to decide whether the disease is syphilitic or a rheumatoid arthritis. The Wassermann reaction is often negative and the diagnosis may be finally settled only by the improvement which follows administration of antisyphilitic remedies.

3. A chondro-arthritis, originally described by Virchow, is seen very rarely. The synovial membrane is the seat of a gummatous synovitis. The cells of the articular cartilage soften and proliferate and then become eroded. These eroded areas do not correspond to the sites of intra-articular pressure. Erosion and eburnation of the bones themselves are not extensive, and whilst there may be a little crepitus upon movement, osteophyte formation or lipping of the articular margins does not occur. The pits which are made in the bone-ends are filled with gummatous material or fibrous tissue.

The tertiary manifestations of syphilis so far as they occur in joints improve rapidly with the administration of potassium iodide and bismuth, probably more so than with some of the new intravenous preparations.

### ARTHRITIS DEFORMANS

Under the title of arthritis deformans is included a variety of joint diseases of which the etiology remains obscure, in spite of innumerable investigations. Whilst such diseases are generally regarded as being produced by either toxic or infective agents, little success has been achieved in defining exactly what these are. Many claims have been made concerning the true causes and for appropriate treatment, but one after another they have failed to stand up to the test. Although this group of joint diseases is so intermingled, it is possible to separate two main classes which differ essentially in their pathology and clinical signs.

### RHEUMATOID ARTHRITIS

Rheumatoid arthritis is the first of these. It is an acute or subacute polyarticular disease of either toxic or infective origin, which finally becomes chronic. The characteristic feature is a thickening of the synovial membrane and the extra-synovial tissues, leading to a chronic fibrosis with the production of contractures and deformities. It is a disease which may have either an acute or an insidious onset. Most commonly it develops in young adults of 20 to 30 years of age, who previously have been quite healthy. Women are more often affected than the opposite sex, in whom it is a rare condition. Little or nothing is known as to its causation.

*Pathology.*—In the early stages the inflammation is confined to the synovial membrane and periarticular structures, the capsule and ligaments of the joint. The synovial membrane becomes much thickened and shows hypertrophy of its villi, so that the surface appears shaggy. Whilst there may be an effusion into the joint, this



is usually small in amount. Histological examination of the synovia shows a low-grade subacute or chronic inflammation. As the condition progresses the synovial membrane and periarticular structures become more and more fibrotic and the neighbouring muscles atrophied and contracted. In the early stages the articular cartilages are not involved, but in the process of time their edges are invaded by a pannus of granulation tissue, which has spread from the inflamed synovial membrane. This granulation tissue gradually erodes and undermines the articular cartilage, so that it becomes softened and necrotic. Its cells are slowly absorbed, being replaced by fibrous tissue, and adhesions are likely to form between the opposing articular surfaces. The underlying bone becomes rarefied and the spaces which are thus formed are filled with fat intermingled with fibrous tissue. In this disease, as compared with osteo-arthritis, no osteophyte formation takes place nor, except in rare instances, does bony ankylosis result.

*Clinical Signs and Symptoms.*—The onset, as has already been said, may either be acute or insidious, but before any joint involvement is noticed or complained of, these patients have for some weeks or months before been in indifferent health.

The disease starts most often in the small joints of the hands or feet, following which signs appear in the larger joints, especially the knees, spreading thence to others so that in acute cases the patient must be confined to bed. The degree of pyrexia during the acute stage varies from just above normal up to as high as 103° F., or even higher. The patient is obviously ill and very rapidly the general health becomes impaired, the pain in the joints preventing any restful sleep at night. The appetite begins to fail, weight is quickly lost and the skin develops a sallow tint. A peculiar odour is associated with such patients, probably owing to the fact that their hands and feet are always moist.

The disease tends to exhibit exacerbations and during the intervals the signs of inflammation in the joints settle down, but after each remission the amount of permanent thickening is increased and, unless steps have been taken to prevent them, deformities are more noticeable. X-ray examination during the active stage shows no bone changes. As the acute signs settle down pain diminishes, but the patient is left with various contractures or deformities depending upon how successfully the limbs have been splinted during the active process. An X-ray taken of the joints at this stage will show a decalcification of the ends of the bones with some translucent areas close to the articular surfaces, and an absence or diminution of the joint line in consequence of damage to the articular cartilage. When all signs of activity have subsided, patients begin to put on weight again and, owing to their activity being restricted by the joint changes, they are liable to become heavier than before.

*Diagnosis.*—This is obvious in a typical example of the disease, but any polyarthritis of sudden onset in a young male adult, in spite of his denials, should be very carefully investigated as to the presence of the gonococcus in the urogenital tract, for rheumatoid arthritis is seldom seen in males. A gonococcal polyarthritis in the



early stages gives rise to more pain than does the true rheumatoid arthritis, and it can also be more crippling in its final end results.

*Prognosis.*—The prognosis as regards complete recovery without any permanent disability is bad. Rheumatoid arthritis, especially in the young adult, tends to continue and progress in spite of treatment, though ultimately after many remissions it seems to burn itself out. This may, however, take several years and the patient will be much crippled from rigidity of many joints if not from actual deformities. In spite of the greatest attention, whilst the disease is active, some deformities may develop.

*Treatment.*—Since it is probable that rheumatoid arthritis is caused by some infection or toxic absorption every endeavour must be made to discover the possible source, for which purpose the teeth, tonsils, genito-urinary tract and alimentary canal require a thorough bacteriological examination with a view to isolating the causative organism, should one exist. The teeth and nasal sinuses need to be X-rayed as it is possible for these to be infected without any symptoms. In few patients with this disease is anything found, but in spite of this every attempt should be made to locate the cause, for should any likely organism be isolated an autogenous vaccine may be of value. The general experience of vaccines is disappointing, as they appear to have little or no influence upon the progress of the arthritis. Nevertheless, even if only an occasional patient seems to benefit, it is worth while employing such a therapeutic measure. The bowels need to be kept regular, but irrigation of the colon is of little value and disturbs the patient. Intestinal antiseptics likewise have no influence upon this disease. Rest in bed is essential with such splintage as may be necessary to make the patient comfortable and to prevent the development of deformities. Considerable relief can be obtained from radiant heat and diathermy. When the more active signs have subsided and the pyrexia has completely settled, treatment at a spa, such as Droitwich or Bath, will sometimes produce a marked improvement in the condition of the joints and in the general health of the patient, but at the start it is impossible to predict which patient is likely to benefit from these measures. One may improve out of all knowledge within a short period, whilst another apparently similar in all respects remains quite stationary. Whilst it has to be admitted that treatment appears to have little effect upon the progress of the disease, this will certainly settle down sooner or later. Often the inflammatory process in all the joints except one subsides, whilst this one remains active and painful in spite of everything that is done. In such a state relief will follow an arthrodesis of the affected joint. In patients where for any reason deformities have developed, correction of these is necessary either by gradual splintage or by operative measures.

#### STILL'S DISEASE

This disease is similar to rheumatoid arthritis. It is a poly-arthritis of childhood associated with some enlargement of the spleen.

The onset is acute and associated with pyrexia of a swinging nature. The child is seriously ill, loses weight rapidly and develops painful swellings of its joints similar to those seen in rheumatoid arthritis in the adult, but the whole condition is much more severe and pain is a more marked feature. Deformities rapidly develop if permitted to do so.

The diagnosis is in doubt at first for the clinical picture is similar to that of acute rheumatism, but enlargement of the spleen and the failure to respond to salicylates leaves very little doubt as to the true nature of the complaint.

*Treatment.*—This is very unsatisfactory and such a child is commonly left with the majority of its joints, both large and small, more or less fixed and rigid from periarticular fibrosis. The only satisfactory method of splintage is to fix the limbs in the correct position in plaster of Paris under an anæsthetic. When this is done the acuteness of the disease will often subside at once and the temperature rapidly fall to a lower level. Such fixation requires to be continued until the general condition is satisfactory, even if this takes several months. Afterwards massage, radiant heat and active movement will restore a surprising range of movement to joints, which may appear at one stage to be completely rigid. The muscular wasting, which is a very prominent feature of this disease, takes many months before it is overcome.

### SPONDYLITIS DEFORMANS

This disease, which results in rigidity of the spine, is probably one variety of rheumatoid arthritis. It is seen in young adults and starts gradually with pain and stiffness of the lumbar spine. An X-ray in an early case will show changes in the sacro-iliac joints. To commence with it is entirely a periarticular fibrosis with which is associated pain and rigidity. The disease spreads up the spine, which in time becomes kyphotic and absolutely rigid, being known as the poker spine. After a time pain and stiffness appear in the hip joints, and these become gradually stiff. Nothing is known as to its etiology, and treatment must aim at the relief of pain and prevention of deformities, especially of a kyphosis (see p. 887).

### OSTEO-ARTHRITIS

This disease usually affects one of the larger joints and is characterised by atrophy, grooving and eburnation of the articular cartilage, associated in the advanced stages with osteophytic formation at their edges. Though as a rule one joint only is the site of this disease, there is no reason why others should not be involved. One of the most commonly affected is the knee joint.

In contrast to rheumatoid arthritis, osteo-arthritis is a degenerative condition and not the result of infection, though this certainly makes it worse. It is liable to develop in the joints of the lower limb, especially the knee and hip, as the result of the strain and trauma to which they are exposed.

*Pathology.*—The synovial membrane is inflamed and thickened as the result of cell proliferation, and an effusion of varying amount, while never great, may be present. Villous hypertrophy of the synovial membrane occurs and as the result of a fatty degeneration taking place in the synovial fringes a condition of “*lipoma aborescens*” may form, fatty masses hanging into the joint. The articular cartilage softens, then degenerates, fibrillation of the matrix takes place, and at those places where pressure occurs it is worn away and the bone-ends exposed. The cartilage cells at the edges of the articular surfaces proliferate and, growing out into the soft tissues, form cartilaginous masses, which undergo ossification and form osteophytes. Some of these may become broken off and form loose bodies (Fig. 576). The bone-ends first become softened and then in consequence of friction hardened, eburnated and grooved. The development of these grooves is best seen in the knee joint. True bony ankylosis as the result of osteo-arthritis very rarely occurs.

Intra-articular cartilages or ligaments are gradually destroyed and disappear. Enlargements of various bursæ in communication with the joint are seen at times and, should one develop in a knee joint which is affected with osteo-arthritis, it is known as a Baker's cyst, though this is very rarely seen.

*Clinical Signs and Symptoms.*—The patient is generally past middle life and symptoms may develop quite suddenly after some minor injury or reveal themselves more gradually. The patient complains of stiffness associated with a dull ache, which is made worse by changes in the weather. Pain is complained of upon making any movement after a period of rest. Creaking in the joint may or may not be noticed, but in the knee it is usually a prominent feature. Upon examination a little swelling may be observed and the temperature compared with the corresponding joint on the other side of the body may be raised. Pressure upon the synovial membrane at its reflection from the edge of the articular cartilage produces tenderness. There is a varying degree of limitation in movement, which produces a fine creaking or crepitus. In some joints, especially the knee, osteophyte formation can be felt. Muscular wasting in the early stages is not obvious, though after a time it may be quite considerable. The symptoms of osteo-arthritis are subject to exacerbations, and during the intervals the patient may be entirely free from trouble, until some minor injury or

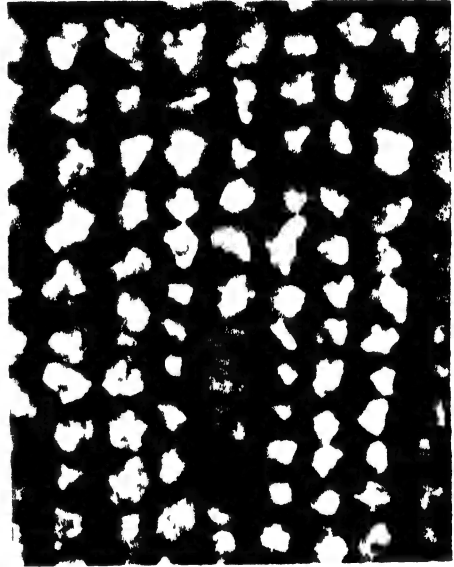


FIG. 576

Multiple loose bodies removed from a hip joint. These formed a small proportion of the whole.

twist produces further symptoms. These intervals of freedom become less frequent and of shorter duration as the condition progresses.

Whilst most of those patients have septic teeth or are habitually constipated, there is no evidence that these conditions cause osteo-arthritis, though there is no question but that the absorption of toxins from such a focus will intensify symptoms in the degenerated joint.

*X-ray Examination* in early osteo-arthritis shows no bone changes. After a time the bones will appear closer together, the joint line being diminished as the result of damage to the articular cartilage. Later still osteophyte formation with lipping of the edges of the bones is visible. The degree of bone changes in such a joint bears no relationship to the symptoms. It is possible for a joint with very definite osteophyte formation to give rise to little or no pain, whilst another with a nearly clear X-ray may suffer intensely in this respect.

The *treatment* of osteo-arthritis can only be palliative, for it is not possible to prevent the joint getting worse. Any definite septic focus must receive attention and the bowels require regulating. Pain in the affected joint can be relieved in the early stages by radiant heat or diathermy, but it must be appreciated that pain is the direct result of limitation in movement and that, if the range can be improved, great relief will be given to the symptoms. In the past such a joint would have been treated with rest, a splint and Scott's dressing. This only made the patient worse, and it is now agreed that mobility of the joint must be improved if relief of symptoms is to be obtained. Therefore, after the application of radiant heat the joint requires manipulation, if possible, without an anæsthetic, a little further improvement being obtained upon each occasion. If this is of no value, one manipulation under full anæsthesia followed by physical treatment and active use may obtain the desired result.

In those joints which are too much damaged for such means to be adopted, relief can be obtained with any certainty only by fixation of the joint to prohibit any subsequent movement. Provided the patient is in a sufficiently good general condition to stand the operation, such a procedure as arthrodesis gives great satisfaction.

#### OSTEO-ARTHRITIS IN SPECIAL JOINTS

The **hip** is quite commonly the site of an osteo-arthritis, which in old people is known as *morbis coxæ senilis* (Fig. 577). Osteo-arthritis of this joint, whilst commonly seen in later life, may develop at any age. In the young it is apt to follow an unreduced separation of the femoral epiphysis or too forcible attempts at its reduction. It follows an untreated pseudocoxalgia in which the femoral head has become misshapen, sometimes within a period of a few years. It may occur about the age of 30 years in an unreduced congenital dislocation of the hip, the arthritis developing between the femoral head and the false acetabulum. In patients of about 50 years of age an osteo-arthritis will sometimes occur in this joint without any definite reason. They may give a history of an accident many years previously, and it is quite often found that in their younger days they were in the habit

of taking exercise, during which this joint might have sustained injury without symptoms developing at the time.

*Pathology.*—The synovial membrane is very much hypertrophied and the capsule of the joint thickened and fibrosed. The head of the femur is flattened like a mushroom, as if its substance had melted and then hardened again at the margin, and shows a large amount of osteophyte formation. The head in an old-standing osteo-arthritis of the hip may be found partly subluxated out of the acetabulum, the upper and back lips of which have melted in the same way as the femoral head and then ossified again. The articular cartilage is almost completely destroyed and the two bone surfaces are composed of sclerotic bone.

*Clinical History.*—The patient complains of stiffness upon rising from a chair, of the leg becoming shorter or of the hip bone sticking out, whilst difficulty in getting the shoe on to the foot of the affected limb is sometimes noticed. Pain varies enormously. It may occasionally be so severe that life is miserable by day and at night sleep is impossible, but whilst pain is always present to some extent, it is usually more of a constant dull ache. Pain along the course of the sciatic nerve is occasionally complained of rather than in the joint itself. By the time advice is sought the hip is fixed in adduction, flexion and eversion, so that sometimes the patient complains of pain in the lumbar region and not in the joint. Such pain is due to muscular

strain in constantly maintaining a lumbar lordosis to compensate for the fixed flexion deformity of the hip, and the patient often has no idea that there is little or no movement in that joint. An X-ray shows the joint line either diminished or absent as the result of erosion of the cartilage, whilst the head has become flattened, irregular in outline and often subluxated backwards and upwards. Large masses of osteophyte formation are usual upon the head of the femur and also on the lips of the acetabulum.

*Prognosis.*—Many patients, after a time, cease to have much in the way of symptoms and learn to adjust their mode of life to the condition which exists in the hip joint, but in others the pain becomes worse and drastic measures may have to be employed to relieve it

*Treatment* consists in such measures as radiant heat, ionisation,



FIG. 577

Advanced osteo-arthritis of the hip joint in an elderly patient showing enormous overgrowth of bone and commencing ankylosis at the upper part of the joint.

diathermy and massage, which give the patient considerable relief, if only for a limited period. Spa treatment does not produce as much improvement as it does in rheumatoid arthritis. Manipulation of the hip joint under an anæsthetic often gives temporary relief. The explanation of this is that the pain is due to fibrosis and contracture of the capsule rather than to formation of osteophytes, and by manipulation the capsule and ligaments are stretched, physical treatment afterwards preventing them, at any rate for a time, becoming contracted again. Manipulation is also a therapeutic measure of value, as by this means the bad position into which the joint may have got can to some extent be corrected. Under an anæsthetic the thigh is first abducted, the adductor muscles (if these are contracted) being divided with a tenotome so as to obtain as wide abduction as possible. The joint is then fixed in plaster in slight abduction. In this the patient can walk in a few days with freedom from pain, for it is not weight-bearing but movement in osteo-arthritis which gives rise to symptoms. Such a plaster may be worn for a period of three months, and this fixation is often a means of relieving the patient from pain and thus enabling him to sleep in peace. Should all these methods of treatment fail, surgical means will have to be employed. While all kinds of operations have been devised and employed for the treatment of a painful osteo-arthritic hip, there is only one method which can be advocated with any certainty of success, and that is the operation of arthrodesis, either intra-articular, or this combined with extra-articular fixation. Whilst this is a severe procedure, the amount of shock is not great, provided it is done quickly and the patient not allowed to lose a lot of blood. The relief of pain is immediate, and after about two months the patient can be permitted to walk in a close-fitting plaster, which is retained in all for about six months. The position in which the hip is fixed must be one of 30 degrees flexion and slight abduction. Recently fixation of the hip joint has been carried out by means of a long Smith-Petersen nail driven through the neck and head of the femur into the thick portion of the ilium above the acetabulum. It is a less severe operation and the patient can walk after a few weeks without any external fixation.

### THE KNEE

This joint is affected with osteo-arthritis more commonly than any other. Women are peculiarly susceptible to degenerative changes of this nature in this joint. They complain of stiffness, pain and tenderness on movement after resting. Their knees are liable to give way, and difficulty is experienced in coming down stairs rather than in ascending them. After any severe injury, such as a fracture of any of the bones forming the knee joint, osteo-arthritis is liable to develop, though the onset of symptoms may be delayed for many years. In the common variety in women the X-ray may reveal few changes, while at times a joint which has only just begun to give symptoms will be found to show marked lipping and osteophyte formation in the film.

*Treatment.*—Rest with a back splint is definitely harmful in the osteo-arthritic knee of the middle-aged woman. Movement, which is limited, especially as regards flexion, must be increased. For this purpose, heat in some form must be employed, and then the masseuse is instructed gradually to increase the range by active and passive movements. The patient must be encouraged to do exercises to maintain movement afterwards. If such methods do not achieve the desired end, manipulation under an anæsthetic will often succeed. In some knees the removal of osteophytes and even both cartilages, which are so swollen and thickened that they prevent full extension of the joint, will result in great permanent improvement in function. When osteo-arthritis follows upon some severe injury, these measures may fail to give relief. Pain is too severe, and the knee may have developed a fixed flexion which prevents the patient bearing weight upon it. Under these conditions an arthrodesis is necessary to relieve pain and provide a limb capable of bearing weight. The only disadvantages of a stiff knee are that the patient is unable to kneel, and the limb is apt to be a nuisance in a bus or train, but the relief of pain and the improvement in the general condition more than compensate for these disadvantages. In a few patients where the arthritic changes are principally confined to the patello-femoral joint surfaces, removal of the patella will cure the symptoms and avoid a stiff knee.

#### THE ANKLE

Osteo-arthritis is wont to develop in this joint after a Pott's fracture which has not been completely reduced, and even sometimes after an apparently perfect reduction. It is a very crippling disability, for the patient is unable to walk on account of constant pain, and an arthrodesis is called for as the only means of relieving the condition. Arthrodesis of the ankle, although it abolishes the movement of this joint, handicaps the patient very little and an active life is perfectly possible, as also is hard manual work.

#### THE SHOULDER

In this joint osteo-arthritis is uncommon, but is often diagnosed as neuritis, since the patient complains of pain down the front and outer surfaces of the arm. Examination of the shoulder joint under such circumstances reveals limitation of movement in all directions, internal rotation being restricted more than any other movement. X-ray examination shows little, if any, osteophyte formation.

*Treatment* consists in manipulation under an anæsthetic, the arm being subsequently fixed on an abduction splint at an angle of 90 degrees to the trunk. Physical treatment with exercises, to maintain the movement obtained by manipulation, is employed and the abduction splint is worn until the patient can voluntarily hold the arm abducted at a right angle and can lower and raise it from the trunk to this position.



### LOOSE BODIES IN JOINTS

Loose bodies occur in several joints, but it is the knee in which they are most often encountered. A single one may be found, or as many as two or three dozen (Figs. 578 and 579). They may be either entirely free to wander about within the cavity of the joint or shut off in synovial pouches. Sometimes they are attached by a pedicle to the synovial membrane. Several varieties are found.

1. **Cartilaginous Loose Bodies**, which originate from cartilaginous nodules in the synovial membrane, the so-called synovial chondromata. To commence with, such loose bodies are pedunculated, but after a time they may become free and then give rise to symptoms. They have a smooth cartilaginous exterior, with a nucleus of ossified

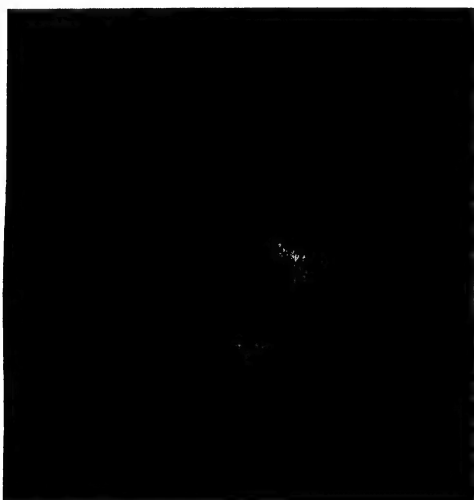


FIG. 578

A single loose body in the knee joint.



FIG. 579

Multiple loose bodies in the knee joint.

bone in the centre, and gradually increase in size, deriving their nourishment from the synovial fluid. Such a loose body may develop in a joint which is otherwise normal, or, if they are multiple, an osteo-arthritis is commonly present (Fig. 580).

2. **Ecchondroses** may be broken off and become free in the joint, forming irregularly shaped loose bodies.

3. A portion of the articular cartilage may be torn off as the result of violence and form a loose body. This is seen in the knee joint in the condition known as **osteochondritis dessicans**. The fragment is separated from the lower end of the femur and leaves a hole at the place whence it came. Such a loose body is composed of articular cartilage covering a portion of bone, the other surface of which is smoothed over by a layer of fibrous tissue.

4. **Fibrinous Loose Bodies** occur in joints after a hæmarthrosis, or in a tuberculous arthritis. In the latter condition there may be many hundreds of them. They are flattened and elongated, and from their appearance are called "melon-seed bodies."



The *symptoms* of a loose body are produced by it becoming caught between the articular surfaces. The joint is locked for a moment with acute pain, the loose body then slips out of the way and freedom of movement is restored to the joint. After the first attack a synovitis results, but this becomes less frequent and of minor severity after each attack of locking. The patient can often isolate the loose body, especially in the knee joint. Owing to their characteristic habit of wandering from one part of the joint to another, these bodies are sometimes known as "Gelenkmaus" or joint mice.

The *diagnosis* is usually confirmed by an X-ray, as these typical loose bodies contain an osseous nucleus. In the knee the diagnosis between a loose body and a torn semilunar cartilage is not always easy, unless the former appears in the subcrureal pouch, when it can be felt.

The history is all important, as locking with a loose body is momentary, whilst, in the case of a cartilage, this will remain out of position until it is reduced either by the patient or his doctor.

*Treatment* consists in removal through a small incision. In the knee, if the loose body wanders about very freely, after the joint has been opened it may get lost; in such a case the cavity should be flushed out with sterile saline, when the loose body will often float out of the incision and obviate the necessity of a wide exposure.

The *prognosis* with a single loose body, if no arthritic changes are present, is good, the joint being capable of full activity. If an osteo-arthritis is present the removal of the loose bodies will improve the patient's function, but the underlying arthritis may continue to cause trouble, though often it ceases to do so when the loose body has been removed.

When resulting from injury it requires removal, but the joint takes longer to recover than in other cases.



FIG. 580

Two loose bodies in the elbow.

### HÆMOPHILIAC JOINTS

In the condition known as hæmophilia, any minor injury or twist is likely to result in a sudden effusion of blood into a joint cavity. The knee is most often affected in this way. The joint becomes suddenly distended, hot and painful, for no very obvious reason. After the first attack it may recover its normal function, but further attacks are likely to occur, and in a very short time the synovial membrane becomes permanently thickened and movements restricted. With repeated attacks of hæmorrhage adhesions develop, and ultimately a chronic arthritis with destruction of the articular surfaces follows, the joint becoming so lax that its stability is impaired.

*Diagnosis.*—At the first attack this may be difficult, but the painful and sudden distension of the joint without any serious trauma

should always give rise to a suspicion of the true nature of the condition; also, if careful enquiry is made into the history, some information of previous hæmophilia in the family can be obtained.

*Treatment* consists in absolute rest in bed, with a pressure pad and bandages to help the absorption of fluid. Later, gentle massage and exercises to keep the muscles in condition are desirable. Under no circumstances must the joint be aspirated, as this is likely to start the hæmorrhage again.

The *prognosis* as regards the affected joint is not very good in consequence of the disorganisation, which ultimately may occur as the result of repeated hæmorrhages. A splint of some kind is necessary to enable the patient to get about.

## NEUROPATHIC ARTHROPATHIES

### CHARCOT'S JOINTS

A neuropathic arthritis is a condition in which the joint undergoes

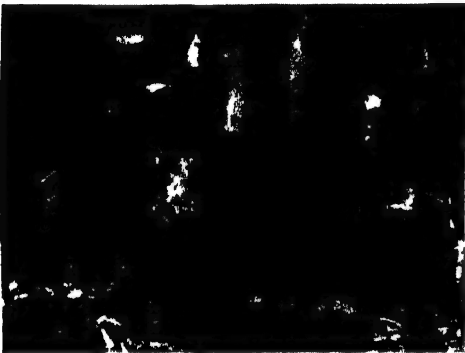


FIG. 581

Charcot's disease of the ankle joint.

certain degenerative changes, the patient being the subject of some disease of the nervous system. It is most commonly seen in tabes dorsalis, when such an affection is known as a **Charcot's joint**. A similar type of arthritis occurs in association with syringomyelia, and, apart from any other sign, if it is present in the upper limb the underlying disease is probably this latter.

Two distinct clinical types are met with:—

1. The **hypertrophic**, in which the joint becomes disorganised and enormous masses of new bone are formed around the edges of the articular surfaces. Bony masses may also form in the synovial membrane or in tendons as, for example, in that of the quadriceps when the knee is affected.

2. The **atrophic** type, which is not as common as the previous variety. In it the joint becomes distended with fluid, there is some thickening of the synovial membrane and the bone-ends become rarefied and absorbed (Fig. 581).

Nothing is known of the pathology of this condition except that it occurs in tabes with degenerative changes in the posterolateral columns of the spinal cord; but why in one patient the hypertrophic form is developed and in another the atrophic is unknown. The knee is most often involved, though any joint may show these changes. Occasionally more than one joint may be affected. In both varieties the onset may be quite sudden, without any previous history.

*Clinical History.*—The patient either seeks advice because the affected joint is unstable, or may complain of lightning pains in the limb without having noticed any pain in the joint. This is swollen,

with a varying amount of effusion, which in the atrophic type is more marked than in the hypertrophic. Abnormal mobility is present, especially in the atrophic type in which, owing to softening of the ligaments, the bones can be moved on each other in all directions. In the hypertrophic variety much creaking may be felt, and large masses of bone are palpable in and around the joint. Other signs of tabes are usually present, such as an Argyll-Robertson pupil, loss of knee-jerks and ataxia, but in a few patients the development of the joint condition is the first sign of any lesion of the central nervous system.

An X-ray examination will reveal the nature of the joint disorder, if this has not already been decided by a clinical examination.

The *prognosis* varies greatly with each individual. In one the condition will remain unchanged often for a year or two, whilst in another, especially if the lesion is of the atrophic type, the absorption of bone may take place very rapidly and the stability of the joint become impaired within a very short interval.

*Treatment* can aim only at providing some form of splint which will enable the patient to bear weight on the limb by rendering the joint more stable. Great care must be taken to avoid pressure sores whilst using this splint, for the sensation of the limb is impaired. Any attempt to render the joint stable by operation is undesirable on account of the danger of its becoming infected. Very rarely amputation may be called for, but, except in selected cases, this should be avoided owing to the danger of the flaps not healing properly.

As has already been pointed out, a similar articular condition may develop in **syringomyelia**, a disease in which there is a gliomatous degeneration of the spinal cord. It occurs generally in the cervico-dorsal segment and is characterised by loss of the sensations of pain, heat and cold, together with muscular atrophy. Owing to the site of the cord lesion, an arthropathy in syringomyelia is most commonly seen in the upper limb. The atrophic form is nearly always found in this disease. The patients are usually much younger than those who develop the typical Charcot's joint of the lower limb.

*Treatment* is unsatisfactory, and the wearing of any apparatus is especially difficult, as the danger of trophic ulcers is very great. Joints of this nature may develop in other nervous diseases, such as myelitis, disseminated sclerosis, anterior poliomyelitis or spina bifida, but they are so rare as to require mention only.

### HYSTERIC JOINTS

These joints are very much more common than is sometimes realised. No age is immune, even very young children being liable, especially after a minor injury, and often the most unlikely people develop troubles of this nature. The characteristic clinical feature of all of them is that the affected limb and joint are held in a grossly exaggerated position, such as is seldom seen in any organic disease. Great pain is complained of and the joint concerned is held rigidly fixed. Any attempt, however slight, increases the pain of which the patient complains, and any endeavour to move the joint is firmly resisted. Clinical

examination will fail to reveal any signs of disease in the joint. During sleep the limb may be seen to lie in a normal position, and under an anæsthetic may be moved normally in every direction. The greatest care needs to be taken before a diagnosis of hysteria is made, for to treat a joint with an organic disease as hysterical would be a disaster.

*Treatment* is always very difficult. In young children, when it follows some minor trauma, if no notice is taken and the child is sent to school and allowed to play games the condition clears up. But in adults the problem is quite a different one. The patients are usually introspective and surrounded by troublesome relations who, in their attempts to be kind, often make it harder to obtain a cure. The patients may show every eagerness to be cured and yet at the same time resist treatment. They should be removed from proximity to relations and friends and placed under the care of a psycho-analyst. Unfortunately, even if he is able to cure the particular condition from which they are suffering, there is a great liability for some other manifestation of hysteria to develop, perhaps months or years later, should a favourable emotional state arise.

E. P. BROCKMAN

## CHAPTER XLIX

### DEFORMITIES

**A** DEFORMITY may be defined as a morbid alteration in the form of a part or organ of the body. This does not mean that there is necessarily any visible alteration in the outline of the part, but the morbid alteration is such that its presence is revealed by a certain loss of function. Modern orthopaedics are directed towards the study of the form and function of the human frame ; their attack is trained on those affections which deform the architecture or arrest the balanced mechanism of man's body. Injuries of bones, joints, muscles, nerves and soft structures which result in loss of form or function are thus their legitimate objective.

Prevention is always better than cure, and if the principles and practice of preventive orthopaedics were more liberally applied to-day many of the more severe degrees of flat foot, scoliosis and like deformities would never occur. It must be realised that many deformities are the end result of postural or static anomalies acquired as a consequence of bad habit rather than the result of a definite pathological lesion. An elaborate mechanism is required to control and maintain the upright position of the body. When this mechanism fails, the body processes are upset and many obscure and distressing maladies may result. In such cases the body posture must be corrected.

Congenital deformities are either primary or secondary, the latter differing in no way from similar conditions in post natal life. The primary congenital error is due to some defect in the fertilised ovum, while the secondary error arises from some such mechanical cause as the continued fixation of a part in such a position as to give rise to structural moulding. A congenital deformity is not necessarily obvious at birth. The tendency to congenital dislocation of the hip may be present, but the actual dislocation may be delayed until the child attempts to walk.



FIG. 582  
Congenital deformity of the hand.

Apart from congenital conditions, deformities fall into one of five groups :—

Affections of bones.

Affections of joints.

Affections of muscles, tendons and other soft structures.

Affections of the nervous system.

Static deformities.

In the affections of the nervous system are seen the mechanical effects of attempting to support weight or carry out function with a more or less flail-like part, to which are added the results of trophic wasting and retardation of growth of the part. In spastic paralysis the deformation is largely due to the overpowering of weaker by stronger muscles.

In the production of deformity the earliest factor is usually gravity. Thereafter there is shortening or contracture of muscles and soft tissues, and ultimately deformity of bone from alteration of pressure or of growth. It is clear, therefore, that the subject of deformity is no narrow speciality but a most important branch of surgical science worthy of the closest study.

## DEFORMITIES OF THE UPPER EXTREMITY

### SPRENGEL'S SHOULDER

Congenital elevation of the scapula was first described by Sprengel. The deformity is characterised by an abnormally high and permanent elevation of the shoulder girdle. It is frequently associated with other deformities, such as absence of vertebræ, fusion of ribs or cervical rib.

The shoulder girdle first appears as a cervical appendage, and gradually descends by the end of the third month of intra-uterine life to the level of the upper part of the thorax. The failure of the normal descent is the cause of this congenital deformity, but the reason for this failure has not yet been properly explained.

The condition is often bilateral, and the scapulæ are fixed in an abnormal manner to the vertebral column, either by a cartilaginous bridge or by a band of stout fibrous tissue. The muscles which elevate the shoulder girdle are usually deficient or absent.

*Clinical Features.*—The scapula may be as much as 4 in. higher than normal and is tilted forwards so that the whole shoulder appears to be displaced upwards and forwards. Abduction of the arm is restricted in many cases, since the usual degree of scapular movement is absent. The shoulders are asymmetrical and the neck appears short. Torticollis and scoliosis are present when the case is unilateral. The X-ray appearances are characteristic, the unduly high situation and small size of the scapula being the principal features.

*Treatment.*—Where the scapula is anchored to the spine by means of a bone or fibrous tissue bridge, the latter should be removed and any shortened muscles tenotomised at the same time. In some cases,

where the scapula cannot be properly mobilised, it may be wise to suture its tip to one of the lower ribs, thus securing it in position and preventing a recurrence.

### CONGENITAL ABSENCE OF THE RADIUS

This is a rare developmental error, but important because it is the commonest cause of "club-hand," the hand being permanently deviated from the normal axis of the forearm towards the radial side. In many cases the condition is bilateral. It is often hereditary and may be associated with other forms of congenital anomaly, notably harelip and certain forms of club-foot.

It is believed that the cause lies in some inherent abnormality of the developing mesoderm which goes to form the forearm bones. Whether the abnormality is the result of disease, or of some inflammatory process, it is not yet possible to say.

*Pathology.*—Frequently the radius is completely absent. Occasionally, however, the defect is partial, in which case a small part, usually at the upper end, remains. The ulna is curved, short and thick, and sometimes of considerable size. There are further abnormalities to be found in the musculature of the arm. The biceps often finds a new insertion, but may be absent or fused with the brachialis anticus. The supinator longus is absent in about half the cases, and, even when present, is usually short and stout.

*Clinical Features.*—The arm as a whole is atrophied and weak. The forearm is short, stubby and bowed posteriorly. The hand is small and atrophic. It is deviated to the radial side and slightly palmar-flexed. The thumb is often absent, but in spite of these deformities the limb may retain a surprisingly good function, though grasping power is usually impaired.

*Treatment* is not satisfactory; indeed, most surgeons are pessimistic in their outlook towards this deformity whatever treatment is carried out. This is not surprising, since the anatomy of the arm is so profoundly disturbed. Albee suggests the insertion of a small tibial bone graft into the forearm, mortising its upper end into the ulna about the middle of that bone, while the lower end is inserted into a specially prepared bed on the radial side of the dorsum of the carpus, after the latter has been manipulated as far as possible towards the ulnar side. Prior to the manipulation it is usually necessary to divide many of the shortened structures. If the graft is not autogenous there is a great risk of its complete absorption. The donor of a homogeneous graft should be of the same blood group as the recipient.

### MADLUNG'S DEFORMITY

Congenital subluxation of the wrist, or Madelung's deformity, is in many cases not a true congenital condition, but is the result of some occupational strain or even severe injury. It is often found in washer-women, following the continuous rotatory strain to which the wrist is subjected by the wringing of clothes. It is due to a tear or dislocation

of the attachment of the triangular disc of fibrocartilage, which is responsible for the strength of the inferior radio-ulnar joint, and, as a result of this, the lower end of the radius dislocates forward, carrying



FIG. 583



FIG. 584

Bilateral Madelung's deformity with X-ray appearances.

with it the carpus and leaving the lower end of the ulna prominent on the dorsum of the wrist.

*Clinical Features.*—The wrist appears enlarged and dor iflexion of the hand is impaired. In severe cases rotation of the forearm is limited. The wrist is loose, insecure and irritable. In older cases the lower extremity of the radius is bent or curved forward. The deformity is easily reduced, but recurs immediately the pressure is released. Not infrequently the condition is bilateral (Figs. 583 and 584).

*Treatment.*—In recent cases reduction of the deformity and the maintenance of the reduction by a short plaster case offers great prospects of relief. In cases of longer

standing, operation is indicated. Attempts have been made to stitch the torn triangular fibrocartilage into place, maintaining the position after reduction of the deformity by a plaster, with the hand in a position of dorsiflexion and full pronation. Osteotomy of the lower end of the radius is also recommended, the articular surface being rotated backwards into its normal position and the correction maintained by a plaster case.



FIG. 585

#### CONGENITAL CONTRACTURE OF FINGERS

This condition is often hereditary and is most frequently seen in

the little finger of one or both hands (Fig. 585). The finger is flexed but, in contradistinction to Dupuytren's contracture, the proximal phalanx is hyperextended. Extension of the middle and terminal phalanges is checked by what appears to be a congenital shortening of

Congenital contracture of little finger. The hyperextension of the metacarpo-phalangeal joint is not as marked as usual.



the soft parts on the flexor side. In some cases several fingers may be similarly affected.

In infancy, manipulation and stretching may overcome the deformity by lengthening the contracted tissues. In later life the prospect of cure is slight, because of the recurrence of contraction after the finger has been straightened, even by operative means.

### TRIGGER FINGER

This is a condition in which some obstacle to full movement is present in the affected finger so that the movement can only be completed by very considerable effort on the part of the patient, or with assistance. When an attempt is made to extend the fingers from the fully flexed position the affected finger lags behind in the flexed position, but jerks into extension when the obstruction has been overcome. When passively moved, slight resistance is encountered until a certain point is reached, after which movement is free.

It usually affects the middle finger of the right hand, particularly in women. The obstruction is caused by abnormal narrowing of the tendon sheath from thickening of its wall (tenovaginitis stenosans) or contraction of the volar accessory ligament.

The most satisfactory method of treatment is an exploration of the tendon sheath, when the volar accessory ligament should be divided or the thickened sheath incised and left unsutured.

### DEFORMITIES OF THE ELBOW

**Cubitus valgus** is the name given to the deformity in which the forearm is abnormally abducted at the elbow joint while in **cubitus varus** the inclination is in the other direction. Both of these conditions may frequently be seen as congenital deformities. They are, however, much more common as a result of a supracondylar fracture of the humerus, which has united without being properly reduced. They are usually associated with a considerable degree of laxity of the ligaments of the elbow, especially in a third type of case which is associated with rickets.

Normally, when the supinated forearm is extended there is an angle of 170 degrees, opening outward. This is called the "carrying" angle, because the hand is thus held at some distance from the body while the arm is in contact with the trunk. The angle is not apparent when the forearm is pronated. There is thus a certain degree of cubitus valgus which is normal. Among women, however, this normal angle may be exaggerated to produce a deformity. Each of the deformities may be treated by osteotomy of the humerus just above the articulation, after the method used to correct deformities of a similar nature at the knee. After osteotomy the deformity is corrected and the arm and thorax fixed in a plaster case. In most cases, however, the deformity is so slight and the function of the arm so little limited that it is unnecessary to carry out any treatment.

## DEFORMITIES OF THE SPINE

### SCOLIOSIS

Scoliosis is a distortion of the spine characterised by rotation and lateral bending. In addition to the permanent deviation of one or more vertebræ from the midline of the body, there are alterations in the relative positions of the ribs and pelvis and adaptive changes in muscles and ligaments. The condition occurs chiefly amongst the hospital classes and is rarely seen in private practice. It is more commonly observed in girls—perhaps because they are more conscious of slight degrees of deformity and consequently complain of them.

*Classification.*—The curvature may be spoken of as right or left, according to the side of the convexity. It is also named according to the anatomical region involved, while occasionally curves are referred to as being primary or secondary, but it is sometimes impossible to decide which is which. A single curve is also occasionally called a C-curve, and a double curve an S-curve.

There are two types of scoliosis: (1) functional or postural and (2) structural, rigid or fixed.

The distinction between the two types is quite definite.

1. **Functional Scoliosis.**—A functional curve is one which (1) can usually be corrected voluntarily, (2) is common in children of school age and (3) is caused by faulty posture in occupation, especially during the period of growth.

The extent of the deformity is within the range of normal spine movement, but the attitude is so frequently adopted that it becomes habitual or perpetual. Structural changes occur only in late stages. They are compensatory, and essentially similar to the secondary changes which follow the structural type. When definite changes occur in the tissues in the course of a functional scoliosis, the case may then be regarded as having gone over to the rigid, or structural, type. This occurs in a small proportion of cases.

The typical features of a total scoliosis are as follows (Fig. 586):—

1. A general curve, convex to the left.
2. Elevation of the left shoulder.
3. Backward displacement of the right shoulder girdle and forward displacement of the left.
4. Undue prominence of the right side of the back when the patient bends forwards.
5. Exaggeration of the hollow at the waist-line on the right side.



FIG. 586

Adolescent scoliosis.

Functional scoliosis may be associated with other evidences of muscular weakness, such as round shoulders, lax abdomen and weak feet.

**2. Structural Scoliosis.**—This organic type presents a completely different picture, as the trunk is grossly misshapen. The patient is unable to correct the deformity voluntarily, as the distortion is due to structural alterations in the vertebræ, ribs, ligaments and muscles, and is consequently permanent. The degree varies from a gentle convexity to a sharp angulation known as "razor back." The vertebral column is curved in the anteroposterior plane and, since it is a flexible, weight-bearing rod, it cannot yield in another plane without its constituents simultaneously undergoing rotation. The vertebræ turn away from the area of maximum stress on the concave side of the lateral curve, and are rotated towards the side of the convexity.

*Etiology.*—In many of the cases the cause of the deformity is self-evident, as in the types due to congenital malformation, torticollis, short legs, or other pathological affections of the body; in others, however, it is by no means easy to assign a cause to it. Faulty attitudes at school or at play may be important factors in the etiology, but it is probable that even in these cases there is some error in the neuromuscular mechanism, resulting in unilateral weakness and subsequent deformity. This is especially so in the functional type which makes its first appearance at the school age of life, the period when the strain of mental work first begins to be appreciated.

*Pathology—Changes in the Vertebræ.* The vertebræ at the apex of the curve are compressed to a wedge-shape and are called the wedge or apical vertebræ. The bones above and below the apical vertebræ also show characteristic distortions, in that they are twisted on their vertical axes to form the so-called "lozenge-shaped" vertebræ. The spinous processes are deflected towards the convexity of the lateral curve. The intervertebral discs are compressed and squeezed out beyond the edges of the body. The ligaments on the side of the concavity are dense and thick, while on the convex side of the curve they are thin and lack a definite lateral border. The muscles show evidence of deformity, in that on the convex side they are atrophied, while on the concave side they are hypertrophied.

**Changes in the Thorax.**—The thorax is displaced towards the side of the convexity and distorted, undergoing a twist in a direction opposite to that of the spine, so that its horizontal diagonal is altered. Frequently the development of the internal organs is prevented and their functional activity seriously impeded so that both the pleural and abdominal cavities are deformed.

*General Symptoms.*—In the earlier stages of this condition the patients are comparatively healthy and suffer little inconvenience. They are able to attend school, and their physical development is almost equal to that of a normal child. Usually the mother consults the doctor owing to some outstanding feature in the child, such as a high shoulder, high hip, prominent shoulder-blade or a slant in the waist-line. Usually the deformity is well established before treatment is sought. Pain is seldom the complaint under the age of 10 years. It

usually takes the form of a mild backache, increasing on exertion. At a later stage there is pain as a result of pressure of the lower ribs against the iliac crest. Occasionally, too, referred or root pains are experienced in the limbs, chest or abdomen. Gastro-intestinal disturbances may occur from pressure on the abdominal organs, while similar pressure on the chest causes dyspnoea and tachycardia, especially on exertion. Later in life, painful secondary arthritis of the spine arises.

*Clinical Examination of the Thoracic Organic Curve.*—1. The curve is usually to the right, and some or all of the thoracic vertebræ are involved.

2. There are compensatory curves in the opposite direction above and below the primary one.

3. The right shoulder is higher than the left, and the right scapula so elevated and rotated that its inferior angle projects and is situated much farther away from the midline than on the left side.

4. The ribs on the right side are prominent, projecting backwards, and their angulation is decidedly increased. Their downward inclination is greater, and the intercostal spaces wider than normal.

5. The right arm hangs away from the body, and is farther from the midline than the left.

6. A transverse furrow or crease at the junction of the chest and lumbar region on the left side is noted, while on the right side the normal contour of the waist-line is either entirely obliterated or considerably filled up.

7. When the patient bends forward, the posterior projection of the right side—the rib “hump”—is rendered more prominent, as is the asymmetry of the back.

In an anteroposterior radiogram, where the curve is limited to the thoracic region, the shape of the spine can be aptly likened to that of a question mark. The two vertebræ at the apex of the curve are wedge-shaped, their bases to the right. The abnormal position of the spinous processes, and the appearance presented by the articular facets, are evidence of the rotation of the vertebral bodies.

*Diagnosis.*—A lateral curvature, occurring before puberty and not associated with pain, suggests a diagnosis of scoliosis. When scoliosis is present it must be decided whether it is (a) postural or (b) structural, and the exact type of curve defined. The cause should be ascertained, if possible, as this may have some bearing on the treatment.

The condition may be differentiated from Pott's disease, which presents pain on movement and loss of spinal mobility with impairment of general health, and from arthritis deformans, which usually occurs in adults and is characterised by pain and stiffness, a diminution or loss of the lumbar convexity, and a gradual curvature showing little or no rotation.

*Treatment.*—**A. Functional or Postural Scoliosis.**—As this type is due in great measure to faulty posture, the treatment should be directed to the removal of the cause. In this respect the effects of improperly adjusted clothing, which pulls unevenly on the shoulders, of ill-designed school furniture, of bad habits of carrying, or reading in poor attitudes,

and of defects of sight, must all be carefully considered and their importance assessed. Where the child is weak and pale the diet may be found to require adjustment, fatigue avoided, and an adequate amount of fresh air and sleep prescribed.

The corrective part of the treatment consists in the employment of gymnastic exercises which will develop the muscles of the body. Particular attention is paid to re-education of the special muscle groups which hold the body erect and in its normal symmetrical attitude. The type, vigour and duration of the exercises are regulated according to the patient's ability to complete them without fatigue. Where the patient tends to return to the faulty position between the exercises it is advisable to fit a light temporary corset.

**B. Structural Scoliosis.**—The treatment of this type is a complicated problem since there are serious and advanced alterations in the shape and internal structure of the various parts of the trunk. An attempt is made to stretch the shortened and contracted tissues, to re-establish or increase the spinal mobility and to overcome the malposition and deformity of the vertebræ.

This may be carried out under one of four heads, or combinations of them: (1) gymnastics, (2) corrective jackets, (3) gymnastics plus retention corsets and (4) operative treatment.

1. *Gymnastic Exercises.*—Exercises are useful only in the mildest types of structural scoliosis. If they do not cause any improvement, either the exercises are not being properly performed, or, what is much more likely, the spinal error is too exaggerated. Exercises increase the strength of the muscles and the mobility of the spine, improve posture, and have a wholesome effect upon the patient's general condition. Progressive improvement is the only criterion of efficient gymnastic treatment.

2. *Corrective Jackets.*—Before fitting a corrective jacket, the spine should be mobilised by some method of passive stretching. Lovett uses a special table on which the patient lies face downwards with the legs hanging over the end. Three canvas straps are fitted to the table and work through pulleys. One strap circles the shoulder girdle and one the pelvis, these being respectively above and below the primary curve. When pulled on, they tend to straighten out the spine. A third strap surrounds the chest, at or about the level of the deformity, and traction on it in the opposite direction enhances the corrective force. The lateral deviation may benefit, but the manipulation has little effect on the rotation of the vertebræ. When the maximum correction has been obtained by this method a jacket of the Abbott or the M'Crae Aitken type is fitted (Fig. 587).

The features of the Abbott method of forcible correction are as follows: The patient is placed in a position of flexion on a special frame. The pelvis and shoulders are fixed by canvas bands, and



FIG. 587

Type of plaster jacket for the treatment of scoliosis

corrective pressure exerted on the deformity by a further canvas band, as on the Lovett table. A jacket is then applied in this position of flexion, and is longer on the side of the concavity. Large windows are cut over the flattened ribs, both behind and in front, and small oblong gutters over the rib "humps." Through these latter openings pads of felt are inserted to exert pressure on the bulging part of the ribs. Additional pads are inserted about a week after the application of the jacket, and thereafter at weekly intervals up to the limit of the patient's resistance. During treatment the results are judged by the clinical appearance of the back and chest, and especially by the X-ray appearance of the spine.

3. *Gymnastics plus Retention Corsets*.—As soon as the scoliosis has been corrected, the plaster jacket should be discarded and replaced by a removable one. Gymnastic treatment should also be begun at this stage. The jacket is either a quadrilateral iron type, with pelvic and chest bands, or a removable turn-buckle pattern.

The turn-buckle jacket is of celluloid, made over a plaster cast of the patient's torso. It is divided into upper and lower halves, both of which are open in front to allow of their application. The jacket is strengthened with steel bands and the turn-buckle applied, together with the necessary straps. Gradual tightening of the turn-buckle straightens out the spinal curvature by slow degrees.

4. *Operative Treatment*.—The great majority of cases of rigid scoliosis either cannot be completely corrected or tend to recur whenever treatment is stopped. In these, spinal fusion is indicated to prevent the deformity increasing. The operation is particularly recommended in patients who, on reaching adult life, seem doomed to wear jackets or some other form of heavy apparatus all their lives.

When the maximum amount of correction has been obtained by passive stretching and by plaster-jacket correction, a fusion operation is carried out to prevent relapse. The operation may be of either the Hibbs or Albee type. Ordinarily, a modification of Albee's method is used, and three or four grafts applied along the spinous processes and in contact with the laminae and transverse processes on the concave side of the curve, the ends being embedded between the split segments of the upper and lower spinous processes. After operation the patient is placed on a Whitman frame for about six months, by which time ossification should be complete and the vertebræ securely ankylosed. Thereafter a light plaster-of-Paris jacket is applied and the back supported for at least another six months. If no relapse has occurred at the end of this time, the patient is given a corset which is worn day and night at first, later left off during the night, then for a few hours during the day, and finally altogether. During this period of treatment gymnastic exercises are unnecessary.

## KYPHOSIS

**Adolescent Kyphosis**.—The normal anteroposterior curve of the spine is subject to many variations, so that it is difficult to say when

a borderline has been crossed and a pathological kyphosis produced. The commonest form of adolescent kyphosis is the type known as "round shoulders" (Fig. 588). While the error may become apparent any time after the erect position is assumed, the majority of cases occur at adolescence, and considerable interest centres round the question of their etiology. Some may be due to congenital factors, others to conditions associated with muscular weakness and consequent bad posture, others again to rickets or vertebral epiphysitis.

Vertebral epiphysitis was described by Scheuermann in 1921, when he found definite radiographic alterations, consisting of irregularity and deformity of the epiphyseal discs. Scheuermann considered the

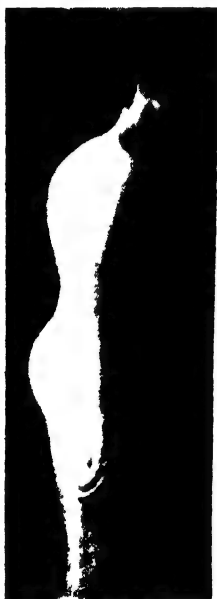


FIG. 588

Adolescent kyphosis.

process analagous to that of juvenile osteochondritis. X-rays demonstrated an epiphyseal irregularity at the anterior edges of the bodies, together with a definite wedge-shaped deformity of the somewhat atrophied vertebral bodies. The intervertebral space was lost, having become clouded and mottled.

*Clinical Features.*—Adolescent "round back" is commonest between the ages of 12 and 18 years, and usually affects the thoracic region. Symptoms are remarkably few; indeed, the only one of note is the deformity. Occasionally, however, there may be vague backache or a history of easily induced fatigue.

In the type due to epiphysitis there may be pain, local and referred to the legs, and the spinous processes of the vertebræ may be tender.

In the postural or muscular type the child lacks vigour and is usually much below the average in muscular development. His movements and gait are clumsy, and there may be other evidences of defective muscular tone, such as prominent belly and flat foot. The attitude is characteristic; the head is flexed, the thoracic curvature and the lumbar lordosis increased, the shoulders droop, the chest is narrow and flat, and the scapulæ are prominent. If it is possible to undo the deformity, such a case is referred to as of the *flexible* type. In many cases, however, the attitude is fixed and permanent—the so-called *resistant* type.

*Treatment*—**A. Postural Type.**—When the deformity can be reduced, either actively or passively, treatment should take the form of mild stretching and of supervised exercises designed to develop the spinal and abdominal muscles. Where the condition is resistant, thorough mobilisation of the vertebral column by stretching is necessary. This is most efficiently accomplished by stretching the shoulders over a padded roll. The forcing back of the scapulæ tends to stretch the contracted soft tissues. When the spine is flexible, gymnastic exercises are employed. A certain number of patients will



require a corset or brace until their muscles have developed sufficiently to maintain the improvement.

**B. Organic Type.**—In this form the spinal error, if untreated, is progressive. Treatment follows similar lines to that of early tuberculosis of the spine, *i.e.*, absolute recumbency on a Whitman frame, with traction applied to the head or legs, or both. After three months of recumbency the child is allowed up, but the spine should still be protected by a plaster-of-Paris jacket.

**Adult Round Back (Senile Kyphosis).**—Increasing spinal deformity commonly accompanies advancing years, and is associated with a variety of pathological changes in the spinal components. Many of these cases have hitherto been classed as osteo-arthritic, and certainly the vertebral changes often bear a close resemblance to this condition. Others are ascribed to occupation, the demands of which have led to certain adaptive changes, rendered permanent as the years go on. The bowed back of old age has been regarded as almost physiological, and few attempts have been made to separate or classify the varying pathological types.

Various groups may nevertheless be recognised :—

1. **TRUE SENILE KYPHOSIS**, in which the spinal curvature is the characteristic process, the intervertebral discs being for the most part well preserved, and the vertebral bodies practically normal.

2. **SPONDYLOSIS DEFORMANS**, in which there is usually some curvature, but the intervertebral discs are degenerated and the vertebral bodies profoundly altered, while there is a marked tendency to the production of marginal osteophytes and ankylosis. These changes have hitherto labelled such a spinal condition as osteo-arthritis or spondylitis deformans.

3. **SENILE OSTEOPOROSIS.**—Here there is a slighter increase in the spinal curvature. The main incidence of the degenerative changes has fallen on the vertebral bodies, the discs being relatively normal, at least in the early phases of the disease.

4. **VON BECHTEREW'S DISEASE.**—Here the main change is in the small intervertebral joints, with resulting ankylosis of the whole column

**Diagnosis.**—In addition to distinguishing between the different members of this series, senile kyphosis must be distinguished from those diseases of the vertebral body which result in deformity. The most important of these are tuberculosis—rare at this age—Paget's disease and Kummel's disease.

Senile kyphosis is seldom amenable to treatment, but if pain is a prominent feature the use of a spinal brace or a chin support may give some measure of relief by preventing the constant drag produced by the weight of the head.

## LORDOSIS

This is the name given to an abnormal degree of the normal anterior curvature of the spine, *i.e.*, where there is an undue anterior convexity. It is almost invariably compensatory in nature and is



found in association with a kyphosis at another part of the spine, as in tuberculosis, with a marked posterior gibbus. It occurs above and below the diseased area to correct in part the forward flexion caused by the gibbus. In stout females there is usually a considerable degree of lordosis in the lumbar region, as there is also in those who are pregnant or have abdominal tumours of any size. There are instances, however, where the condition is not compensatory—in such affections as rickets, spondylolisthesis, and rarely in infantile paralysis. It is sometimes seen in people whose occupations necessitate the carrying of heavy loads suspended from the shoulders, as in street hawkers with a heavy tray of goods.

*Treatment.*—The treatment of these abnormal anterior convexities is the treatment of the underlying cause, or its elimination. Rickety lordosis demands general treatment of the disease itself—local support for the weakened musculature, and massage, exercises and electricity to increase muscular tone.

## DEFORMITIES OF THE LOWER EXTREMITY

### CONGENITAL DISLOCATION OF THE HIP

Congenital dislocation of the hip joint is one of the commonest and most important of congenital deformities. It is a partial or complete displacement of the head of the femur from the acetabulum, probably as a result of some congenital malformation of the parts entering into the formation of the joint. The number of cases in girls greatly exceeds those in boys. It may be hereditary, and accompanied by other anomalies of development.

Primarily there is no socket present on the wing of the ilium, but the acetabulum is formed by condensation and growth of cartilage round the head of the femur. In congenital dislocation the growth of this cartilage does not keep pace with the growth of the head. This retardation in the development of the postero-superior quadrant of the acetabular rim may be temporary, provided that the area in question is carefully protected from any pressure such as would result if the head of the femur were dislocated from the acetabulum. Congenital dislocation of the hip is therefore a symptom of hypoplasia of the acetabular rim, the latter condition being the primary error.

*Pathology—Changes in the Bones.*—The acetabulum is abnormally shallow, owing to the failure of growth of the cartilaginous rim. The cavity is converted from the normal circular contour into a triangular depression, with its base in front and below and its apex above and behind. The deficiency in depth is most apparent in its postero-superior quadrant. On examining the pelvis from the front it will be seen that the outer surface of the ilium and the floor of the acetabulum lie practically in a straight line, owing to the absence of the usual projecting rim at the upper part of the cavity. Above the acetabulum on the dorsum ilii there is a depression, lined with periosteum, in which the head of the femur rests insecurely, separated by a fold of capsule.

The head of the femur is small, atrophied and flattened on its medial and posterior aspects (Fig. 589). In some cases it is absent.



FIG. 589

Congenital dislocation of left hip. The head of the femur is displaced upwards and outwards and is less well developed than on the right side.

The neck of the femur is short, depressed, and sometimes anteverted, so that the normal angle of 12 degrees is increased until in late cases it may be almost 90 degrees, *i.e.*, the neck appears to project straight forwards from the shaft. As a result of this, when the dislocation is reduced, the limb is rotated medially and the patella looks directly inwards.

In bilateral cases the pelvis is tilted forwards and the normal lumbosacral lordosis increased (Fig. 590). The innominate bone is small and atrophied, and lies more vertically than normally, so that the iliac crests are approximated. In unilateral cases the bone on the affected side is imperfectly developed, while the

whole pelvis has a lateral inclination and the shape of the inlet is obliquely ovoid.

**Changes in the Soft Parts.**—The capsule is said to assume an hour-glass shape, one cavity containing the head, the other covering the acetabulum, the constriction between them being produced by the iliopsoas tendon which crosses the capsule at this level. The capsule forms a suspensory ligament for the pelvis and, indeed, supports most of the weight of the body. It accordingly becomes hypertrophied, particularly at its anterior and inferior aspects.

There is considerable alteration in the muscles. Those running in the same axis as the femur are shortened and form a formidable obstacle to reduction. The transverse muscles (the obturators, quadratus femoris and psoas tendon) are stretched and elongated, and become functionally incompetent. The gluteal group show little organic change, but since they are without their fulcrum their power is considerably diminished.

**The Dislocation.**—Since the primary condition is a hypoplasia of the acetabular rim, the dislocation is a secondary effect, and in certain minor degrees of hypoplasia may not be present. Nevertheless, the mechanism for retention of the head within the acetabulum is insecure, and dislocation may take place with the slightest trauma, or even without it.



FIG. 590

Diagram illustrating the increased curve of the lumbar spine in congenital dislocation of the hip.

Where the defect in the rim is pronounced, the child is born with a dislocation, but in the lesser degrees an X-ray will show evidence of the primary error and, after birth, dislocation may occur when the legs are extended at the hip joints for the first time. In mild degrees of the defect, dislocation may not take place until the erect position is assumed. The first attempts at walking are then followed by a gradual upward displacement of the head on the dorsum ilii. Thus, three types of congenital dislocation may be described: (1) the antenatal type, (2) the pre-ambulant type and (3) the post-ambulant type.

*Symptoms.*—Advice is seldom sought until the child begins to walk, when the mother notices a slight limp. Every child, therefore, should be examined for such a condition at an early stage of life by the nurse, midwife or doctor. The presence of dislocation at this early stage might be suspected from broadening of the perineum, or an abnormal position of the head of the femur in the gluteal region.

In the early stages there is neither pain nor tenderness, but movements of abduction and external rotation are limited. The child walks with a distinct dip to the affected side, and when the condition is bilateral the double dip is such that it may be described as a "duck-like waddle." The gait is the result of the mechanical disadvantage of the gluteal muscles, the shortening of the femur, and the displacement of the head, combined with the lordosis and abnormal lateral mobility of the lumbar spine. The lordosis is particularly marked in bilateral cases, but is present to a lesser degree in the unilateral.

In bilateral cases the legs appear to be too short for the body, the perineal space is broadened, the trochanters are unduly prominent and the buttocks broad and flat.

*Examination of the Patient.*—After the child is stripped the alteration in the figure is at once visible. There is a marked prominence of the great trochanter, increase of the lumbar lordosis, lack of development of the limb on the affected side, asymmetry of the groove between the labia and the thigh and, in bilateral cases, broadening of the perineum.

On palpation the femoral artery is less evident than normal, since the supporting femoral head is absent (Fig. 591). Posteriorly, the head can be felt in its abnormal situation.

Movements of abduction and external rotation are limited, but pain is absent. In early cases the femur can be moved up and down in its long axis, because there is nothing to restrict the movement of the head in an upward direction. The name "telescoping" is given to this abnormal movement.

In unilateral cases the affected leg will be found to be from 1 to



FIG. 591

Diagram illustrating the reason for the pulsation of the femoral artery being less apparent on the affected (right) side

1½ in. shorter than the other. There is a positive Trendelenburg's sign (Fig. 592). This is elicited by asking the child to stand on one leg and then on the other, while the buttocks are examined from the back. When the child stands on the sound side the buttock of the opposite side rises as the foot leaves the ground; in other words, the pelvis on this side is raised and the weight is tilted over the leg sustaining the body-weight. If, however, the child is asked to raise the normal leg from the ground, the body-weight being borne on the affected side, the buttock of the sound side drops and remains stationary at a lower level than the other, because of the inability of the hip abductors to support the pelvis and body-weight when the fulcrum formed by the head of the femur on the ilium is unstable.

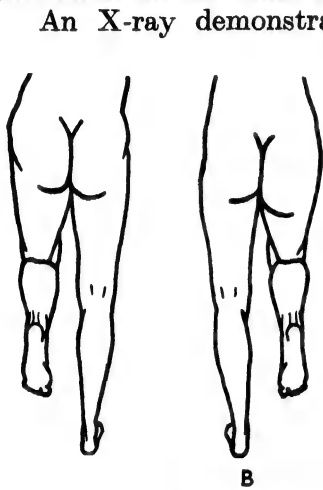


FIG. 592

Trendelenburg's sign. A, patient standing on sound leg, and B, on affected leg. Note the falling of the right buttock when patient stands on left, *i.e.*, the affected side.

An X-ray demonstrates the dislocation of the head on to the dorsum ilii. The outline of the femoral head should be noted. The epiphyseal shadow is usually smaller than normal, and displaced outwards in relation to the neck. The neck is foreshortened and may be twisted. This anteversion is investigated by taking two plates, one with the patella pointing forwards, the other with it pointing inwards. Any anteversion is noted by the superimposition of the head on the trochanter in the first plate, while the second shows the outline of the head quite distinct from the trochanter. The acetabulum appears less excavated than normally.

*Diagnosis.*—Where the patient is a young child, the history of a painless limp, with no history of injury or disease, should suggest a congenital dislocation of the hip. Examination of the case reveals the dislocation, and this is corroborated by an X-ray photograph.

The condition must be differentiated from (1) coxa vara, (2) pathological dislocation of the hip and (3) paralytic dislocation of the hip.

A carefully taken history and a thorough examination, together with an X-ray picture, make this differentiation easy.

*Treatment.*—The aims of treatment are to reduce the dislocation, maintain the reduction, and conserve as far as possible while so doing the function of the joint.

The treatment should be initiated at the age of a few months, before the joint has been subjected to the harmful influences of weight-bearing. The importance of early treatment lies in the fact that in such cases the retarded acetabulum acquires fresh vigour and grows to normal dimensions. In other words, a perfect restoration of the acetabular margin is obtained. This region must be carefully examined by a series of X-ray photographs at intervals until the complete re-formation is visible. Whenever the acetabular margin

remains shallow, even after successful reduction, operative replacement is indicated.

In the early stages reduction is obtained by manipulation.

**The Method.**—Under complete anaesthesia the child lies on its back, with the pelvis and the opposite thigh fixed by an assistant. In the case of a right-sided dislocation, the operator grasps the leg with his right hand, flexes the knee to 90 degrees, and then flexes the thigh to more than a right angle. The thigh is then rotated inwards and slowly abducted, while counter-pressure is applied behind the trochanter with the left hand, to push the head forwards. The reduction is recognised by the occurrence of an audible snap as the head slips over the rim into the acetabulum. The hamstring muscles are now found to be taut and the knee cannot be completely extended. Stability should be tested by releasing the limb and observing with what ease or difficulty the hip redislocates. If reduction cannot be carried out by the above method after two or three attempts, open operation is undertaken.

**After-treatment.**—After reduction the thigh is abducted to a right angle with the body until the inner surface of the knee lies on or behind the plane connecting the anterior superior spines of the ilium. A plaster-of-Paris case is now applied from the nipple line to include both legs, even in unilateral cases. A large pad of wool is put over the front of the lower end of the thigh, and when the plaster is set the area covering the knee may be cut out as shown in Figs. 593 and 594. The pad of wool is removed and a degree of movement is allowed at the hip joint.

The day after the manipulation a radiogram is taken and the position of the hip verified. If the head is not in position, and the case has proved difficult, it may be better to leave the plaster on for about a week, in order to stretch the muscles thoroughly, and then make a further attempt at reduction. The first plaster is retained for three months. At the end of that time the angle of abduction and flexion is lessened, the thigh rotated inwards and a fresh plaster applied for a further three months. When the plaster has finally been removed treatment in the form of radiant heat, massage and exercises will accelerate movement at the joint.

After reduction, progress is followed by means of repeated X-ray examinations, and the state of the acetabular rim noted. In many early cases the rim grows and approaches that of the normal side in architecture. Unfortunately, in the majority of cases treated after the age of 2 years this does not take place, and the hip remains in an

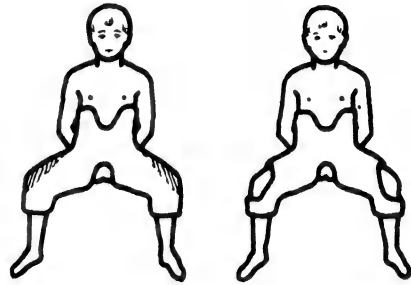


FIG. 593.

FIG. 594.

FIG. 593.—Type of plaster used to retain the hip in position after reduction. The shaded area is padded with cotton-wool, and the prominence thus formed in the plaster is removed as shown in Fig. 594.

FIG. 594.—Plaster after removal of areas over knee joints.

unstable state and will almost certainly redislocate. These cases require a reconstruction of the acetabular rim.

**The Operation of Open Reduction.**—The joint is approached through a Smith-Petersen incision. When the capsule is exposed it is incised parallel with the neck, and the narrow constriction is then seen leading downwards and inwards to the shallow acetabulum. The incision is prolonged through the constricted area to allow the femoral head to be levered or lifted into its socket. After that is done, the capsule is reefed and sewn at right angles to the incision. The muscles are carefully sutured together and the limb put up, in abduction and inversion, in a plaster-of-Paris spica.

The after-treatment is similar to that following manipulation, but the period in plaster may be shorter.

**The Reconstructive Operation.**—The region of the hip joint is approached by the Smith-Petersen incision, as in the open operation for reduction. If the hip is not already reduced, this is done as the first stage of the operation. It is an advantage to open the capsule, thus enabling the exact site of the shelf to be ascertained with precision. A gouge is applied to the ilium a few millimetres above the acetabular margin, and an incision made in a crescentic fashion corresponding to the outline of the upper half of the acetabulum. This is then dug out of the ilium with the gouge, and levered down over the head of the femur, forming a hood. It is quite unnecessary to have any gross projecting ledge; rather the normal size of the acetabular roof is restored. The outward displacement of the upper half of the acetabulum is maintained by the insertion of a piece of bone removed from the posterior part of the dorsum ilii. This piece is cut to form a crescentic plate which will bridge the gap between the ilium and the restored acetabular margin, and its position accurately secured by driving a pair of ivory pegs through it into the ilium.

A plaster spica is then applied with the hip abducted to prevent undue pressure by the head on the newly formed roof. The plaster need not be retained as long as after manipulative reduction. The child may be allowed to walk six or nine months after the operation.

**The Treatment of Old Unreduced Cases.**—Such cases come to hospital at ages between 20 and 30 years, complaining of pain in the lower back and in the dislocated hip, shortening, limp, and with their general endurance and capacity for work greatly reduced.

**A. The Treatment of the Old Unilateral Case.**—In such cases reduction is usually impossible, and the best palliative treatment is the "bifurcation osteotomy" of Lorenz (Fig. 595). The femur is exposed from the upper lateral aspect of the thigh, and divided obliquely from the outer side upwards and inwards, to produce a broad overlapping surface of raw bone. The limb is then abducted, and the upper end of the lower fragment approximated to the ischium,



FIG. 595

Diagram illustrating  
Lorenz-bifurcation or  
osteotomy in Y.

the position being maintained by a plaster case. After union has taken place, the upper end of the femur is Y-shaped, or, as the name of the operation implies, has a bifurcated end upon which the pelvis rests. The abduction diminishes the apparent shortening considerably, while the weight of the body is now supported by bone instead of by soft tissue.

**B. The Treatment of the Old Bilateral Case.**—In such cases an upper rim to the false acetabulum may be constituted by either a shelving or a reconstruction operation. This is carried out on each side. In some cases a bifurcation operation has been done on both sides, with good functional results.

### COXA VARA

In the adult femur the neck is set on the shaft at an angle which varies from 120 to 140 degrees. A decrease in this neck-shaft angle is known as coxa vara, while, if the angle is greater than 140 degrees, coxa valga is said to be present. The depression of the neck in coxa vara results in obvious mechanical disadvantages, as the normal apposition between the joint surfaces is lost, abduction is limited from the upward displacement of the trochanter, and the limb is shorter than normal.



FIG. 596

Adolescent coxa vara due to slipping of the upper femoral epiphysis.

Coxa vara may result from fracture of the neck or trochanteric region of the femur, destructive arthritis, or certain developmental or constitutional diseases. Such types will be described under the headings of the various diseases. It is the type which is called idiopathic, or epiphyseal, coxa vara which will be described here (Fig. 596).

**Epiphyseal Coxa Vara.** This condition commonly occurs in boys between the ages of 10 and 17 years, when the capital epiphysis of the femur is actively growing. It is, as a rule, unilateral.

**Symptomatology.**—There is usually no history of preceding illness or constitutional disturbance. The onset is gradual, and in many cases the earliest symptom is the easy onset of fatigue after walking or standing. In some cases, however, the patient has had a fall or received a blow some time before, and this type is sometimes called traumatic, although the trauma is in most cases very trivial. Not

infrequently the history is elicited that the patient had some disturbance in the affected hip even before the injury.

Complaint is made of pain which may be confined to the hip, but may radiate down to the lower thigh or to the knee joint. The pain is evanescent and disappears for a time, only to reappear with increased severity. It is relieved by rest, and the patient is not troubled at night.

There is a limp and, as the error progresses, this may be present even when pain is absent. The affected leg gradually becomes shorter and smaller than its neighbour and tends to turn outwards, while its movements are restricted.

There is no doubt that trauma and static influences are important factors in the development of epiphyseal coxa vara, but it is probable



FIG. 597

Bilateral congenital coxa vara

that they can act only upon a femur in which the epiphysis is less firmly attached than normally. The pathological conditions which cause the loosening of the epiphysis are not definitely known, but Key points out that they may be neither in the bone nor in the epiphyseal cartilage, but in the periosteum of the femoral neck. In adolescence this periosteum begins to atrophy and to approach the adult type, thus tending to produce a point of weakness at the epiphyseal line (Fig. 597). Many of the cases give a history of very rapid growth previous to the epiphyseal disturbance, and it is possible that this weak periosteum, which normally contributes considerably to the strength of the union between the epiphysis and the neck, is subjected to excessive strain from rapidly increasing body-weight.

*Physical Signs.*—The patient walks with a waddling gait, the body swaying over to the affected side. The pelvis on the sound side tends to drop when weight is borne on the affected extremity. The leg is rotated outwards and somewhat adducted. A slight scoliosis towards the affected side may be present in the lumbar region, and towards



the sound side in the thoracic region. The buttock is atrophied, and the gluteal fold lower than on the normal side.

On palpation of the groin a hard mass can often be felt, which moves with the femur. It is the thickened head and neck. There may be  $\frac{1}{2}$  to 1 in. of shortening on the affected side.

Flexion is limited to about 90 degrees and, as the thigh is flexed, it passively rotates outwards. Adduction and external rotation are free, but abduction, internal rotation, and hyperextension are greatly restricted. In very early cases it may be possible to elicit a soft, muffled crepitus.

An X-ray will show that the head of the femur lies in the acetabulum, but is rotated so that its lower and posterior borders are displaced downwards and outwards. The head is slightly displaced in relation to the neck, its lower border projecting as a beak-like process below the lower margin of the neck. The upper margin of the head is thinned out and separated by a short distance from the prominence made by the upper angle of the metaphysis.

The *diagnosis* is suggested by the characteristic history, the age of the patient, and the adducted, externally rotated position of the limb. In addition, the radiographic appearance is so characteristic that in these days the condition should not be missed.

Coxa vara is to be distinguished from: (1) tuberculosis of the hip, (2) Perthes' disease and (3) congenital dislocation.

*Treatment.*—The obvious treatment is to reduce the displaced epiphysis, and to maintain alignment until a new union takes place. In very early cases it may be possible to reduce the displaced epiphysis. The manipulation, however, must be carried out with great gentleness, otherwise damage will be done to the joint surfaces. The writer believes that heavy traction should be exerted on the leg for about a week before the actual manipulation is carried out. The further manœuvres are simple flexion and internal rotation, followed by traction and abduction of the thigh. The result should be checked by an X-ray examination.

The hip is fixed in the corrected position by a large plaster case, which is retained for six weeks.

In dealing with an advanced case of some months standing, but with an obvious line of demarcation still present between the head and the neck, the hip should be exposed through a Smith-Petersen incision, the head freed from the neck, correct alignment secured, and the reduction maintained by the insertion of a trifin nail.

In a healed case in a young adult, an osteotomy of the femur just below the trochanter is the operation of choice.

### COXA VALGA

This, in contrast to coxa vara, signifies an abnormal elevation of the neck of the femur, the angle which this forms with the shaft being greater than 140 degrees. It is usually a congenital condition, and is not infrequently observed in limbs which have never supported weight.

The gait may be a little awkward, the limb being rotated outwards and abducted. The deformity is uncommon and of minor importance. In most cases function is very little altered.

Where adduction is limited, in some cases manipulative treatment is carried out to overcome this limitation. An osteotomy has been described for the condition, but is very rarely indicated.

**Snapping Hip.**—During certain movements of the hip joint an audible sound, or click, may be heard or felt. In some cases the cause is intra-articular, while in others it is due to factors outside the joint. The former type is not uncommon in children, and results from slight voluntary displacement of the head of the femur over the upper border of the acetabulum.

The more common extra-articular type is analagous with the dislocation of the peroneal tendon commonly seen at the ankle joint. The snap is felt and heard when the knee is flexed and the hip rotated inwards. A tight band is sometimes seen to slip backwards and forwards over the great trochanter.

Where the condition is causing distress, division of the offending band or tendon is usually sufficient to give a complete cure. The operation should be carried out under local anæsthesia, since it is essential to recognise which band is at fault. To prevent post-operative recurrence, the band should be sutured behind the trochanter. After operation, early movement is essential.

#### GENU VARUM

Bow-leg is an outward bowing through the upper and middle thirds of the shafts of the tibia and fibula. Occasionally the femur is also involved. Where the leg is curved in a forward direction the condition is termed anterior bow-leg. The existence of this deformity is presumptive evidence of some degree of rickets, and is the result of the superincumbent body-weight transmitted through soft bones. There is usually an inward rotation of the lower end of the tibia on the long axis of the femur, with the result that the toes are turned in, and, when the child stands with the feet together, the knees are widely separated. On walking, an obvious waddle is present.

If the condition is observed while the process is still active, correction is carried out by repeated manual manipulations. Both extremities of the leg are grasped and bent outward for a few minutes three or four times daily, care being taken that no strain is placed on the epiphysis while the manipulation is being carried out.

If this is not successful, a Knight's brace is used. This consists of two steel uprights attached to the shoe, with a soft leather pad fixed to the upper end of the medial upright to prevent undue pressure on the inner condyle of the femur. The outer upright extends to the head of the fibula, and the two are joined by a calf-band. The bowed leg is drawn inwards towards the inner bar by a broad leather cuff, laced about the leg inside the outer bar. The inner bar may be gradually bent until over-correction is secured.

Operative procedures are carried out only after the active process

has subsided, and consist in fracture without incision through the medium of an osteoclast, or open osteotomy performed at the greatest prominence of the tibia.

#### GENU VALGUM

The deformity of knock-knee develops as a rule in early childhood, and is usually due to rickets. There is an inward projection of the knees, and the leg deviates from the long axis of the femur at an abnormal outward angle. In walking, the feet usually turn in, in a compensatory effort to clear the knees, and the gait is unsightly, as the knees rub together and the line of gravity is transposed to the outer side of the knee joint. The gait is also lurching, with an exaggerated side-sway of the body at each step to preserve balance. The deformity disappears when the knee is flexed, because only the lower ends of the condyles are affected and not the posterior surfaces with which the tibia articulates in full flexion.

*Treatment.*—As in genu varum, daily manipulation is indicated during the active stage, and consists in grasping the lower extremity of the femur with one hand and the ankle with the other, and bending the leg inward, with the internal condyle of the femur as a fulcrum. Corrective splints are also applied during rest periods in the day and at night.

After the active process has subsided, deformities are corrected by operative procedures. The femur is fractured just above the epiphysis with the aid of an osteotome. The limb is then immobilised in a plaster case in the over-corrected position until union is solid. Osteoclastic procedures are not suitable in this type of deformity, since they so easily cause a separation of the lower femoral epiphysis.

#### GENU RECURVATUM

This deformity develops frequently, following paralysis of the quadriceps, when the patient, fearing that the knee will give way, attempts to make the leg stable by locking the joint in hyperextension before bringing weight to bear on it. The posterior part of the capsule is loose and the posterior muscles stretched.

The condition may also be congenital when it is caused by imperfect development of the quadriceps muscle, and may be associated with rudimentary development or absence of the patella. In congenital cases the legs bend forwards instead of backwards at the knee joint, and there is often a forward displacement of the tibia on the femur. In infants, correction may be obtained by gradual flexion of the knee, a malleable posterior splint being applied when the best possible position has been achieved. At weekly intervals the knee is further flexed and the splint bent to conform to the new position. It must be retained until the right angle flexion has been maintained for at least a month.

In older children, operative procedures are indicated to lengthen the contracted structures and are followed by prolonged immobilisation and support by braces. Campbell has recently devised a bone-check operation in which the patella is secured to the anterior aspect of the

upper end of the tibia. When satisfactory union has occurred between the patella and the tibia, it will be found that the former forms a complete check to any hyperextension at the knee joint.

### RECURRENT DISLOCATION OF THE PATELLA

The patella may subluxate as a result of trauma, rickets or congenital anomaly. The displacement is usually lateral. The congenital type is often accompanied by other abnormalities. In the traumatic type the internal part of the capsule is ruptured and the defect is filled in by scar tissue which stretches, allowing a gradual increase in laxity of the capsule on the inner side of the joint. This obviously relaxes the inner ligaments to the patella and permits it to be displaced externally.

*Treatment.*—Reduction in most cases is easy. With the knee fully extended the thigh is flexed to relax the quadriceps and the knee-cap manipulated into position by pushing it medially, while at the same time correcting any rotation.

Conservative measures are only of value before the displacement has become habitual and consist of the wearing of a knee-cage, with an inner wedge on the heel of the shoe. The operative measures which may be employed are notable for their wide variety of choice, depending to some extent on the cause of the error.

Gallie suggested the use of a living suture of fascia lata to form a ligament between the patella and the medial femoral condyle, thus anchoring the patella.

A somewhat similar effect is obtained by transplanting the tendon of the gracilis or the semi-tendinosus to the inner border of the patella.

Krogius transfers a long strip of the redundant internal capsule to the outer side of the knee. It is made to occupy the gap between the edges of a long, vertical, lateral capsular incision. The tense outer part of the capsule which tended to draw the patella outwards is thus divided, and a portion taken from the relaxed medial part to fill the gap produced between the edges by the relief of tension.

Albee believed that the condition is due to a failure of development of the external condyle of the femur, and to correct this he raised the anterior part of the external condyle forwards and kept it forwards by inserting a bone graft under it.

**Transplantation of the Tubercle of the Tibia.**—In most cases this is the operation of choice. An incision is made vertically downwards from the outer border of the patella to the outer side of the tubercle of the tibia, from which point it deviates medially to end over the inner aspect of the tibia. The ligamentum patellæ is defined and, along with the small block of bone into which it is attached, is separated from the tibia. A new bed is now made on the antero-medial aspect of the tibia, its shape corresponding to that of the bony block at the end of the ligament. This is inserted into its new bed and secured in position by a screw-nail. The operation may with advantage in certain cases be supplemented by a soft tissue operation such as that of Krogius.

## CONGENITAL TALIPES EQUINOVARUS

Talipes is the term applied to a deformity of the foot and requires a qualifying adjective to denote the particular type. Talipes equinovarus is a deformity in which there is a persistent plantar flexion and inversion of the foot, with adduction of the forefoot and internal rotation of the tibia. It occurs usually in healthy boys and is more frequently bilateral than unilateral. The condition is now believed to be a primary developmental anomaly associated with hypoplasia of all the bones and muscles of the foot. Not infrequently there are abnormalities in the number or development of the bones. Bohm believes that a primary endogenous disturbance of the embryo, with arrest of its normal development, accounts for the great majority of cases.

In its early stages the condition is a persistence of the normal foetal position of adduction, inversion and plantar flexion. The muscles are poorly developed and the tendons are delicate. The plantar muscles, especially on the inner side, are tensely contracted, while the anterior group of leg muscles is elongated.

The ligaments on the medial and inferior surfaces of the joints between the os calcis, astragalus and scaphoid are contracted, as is also the internal lateral ligament of the ankle joint. Bone changes occur chiefly in the astragalus. A large portion of its upper surface escapes from between the malleoli and becomes prominent on the dorsum of the foot. This part, freed from pressure, becomes broadened and, in severe cases, is an obstacle to passive dorsiflexion of the foot. The neck of the astragalus is longer than normal and is deflected downwards and inwards. The os calcis is plantar-flexed and tilted so that the medial process of the tuberosity approaches the medial malleolus. The anterior extremity is deflected inwards, following the direction of the neck of the astragalus.



FIG. 598

*Congenital talipes equinovarus.*

*Clinical Features.*—The leg is smaller and less well developed than that on the healthy side. The skin of the foot is usually stretched and thin on the dorsum and thrown into creases along the inner border and on the sole. There are evidences of external pressure on the dorsum in the shape of callosities and scars. The head of the astragalus is felt on the dorsum of the foot, which faces downwards and forwards, while the plantar surface is now rotated so that it looks upwards and backwards. The outer border of the foot is convex and the inner concave. The forefoot is plantar-flexed upon the hind foot. The heel is rotated inwards and drawn upwards, throwing the whole foot into equinus (Fig. 598). There is some internal rotation of the tibia on its long axis and, in many cases, a well-marked genu valgum. The child walks with a markedly stumbling gait which lacks elasticity. Bursae and callosities develop over the weight-bearing areas.

With early and continued treatment all cases should be cured and a useful and properly shaped foot obtained. In older children the shape of the foot cannot be completely restored, but the condition should be greatly improved.

*Treatment.*—Whatever type of treatment is advocated, it is important to remember two cardinal rules :—

1. The deformity must be over-corrected.
2. This over-correction must be maintained until the patient's muscles are sufficiently strong to prevent relapse.

The mode of treatment varies with the age and the extent of the deformity.

1. **Treatment of an Early Case.**—The treatment of this type starts about the tenth day of life and consists in manipulation. This may be carried out by a capable nurse or masseuse, who can quickly be trained in the proper method and in the amount of force to be used. It is well to recall that there are three separate deformities which require correction : (1) adduction and inversion ; (2) equinus ; and (3) internal rotation of the tibia. They are attacked in this order and the maximum result of manipulation is maintained for a few seconds at each sitting. The movements should be carried out as often as possible—at least three times a week. They should be repeated until the deformity can be easily over-corrected, and until the foot muscles have developed the power of holding it in the corrected position. They may then be gradually discontinued. In all cases manipulation will have to be continued until the child begins to walk.

After the manipulation some form of retentive apparatus may be used. The best type is the bilateral splint devised by Denis Browne. Both feet are incorporated in the apparatus and a good leverage for maintenance of correction is got from the good foot.

At a later stage, about the sixth month, if the equinus deformity has not been completely overcome, it may be necessary to divide the tendo achillis. This is done by subcutaneous tenotomy. While the child is under an anæsthetic the opportunity should be taken to manipulate the foot freely and to over-correct any remaining deformity. The foot is thereafter put up in plaster of Paris in the over-corrected position. The plaster is applied with the knee bent, is carried up to the middle of the thigh and is retained for three or four weeks.

Supervision should be continued until the child has begun to walk. The deformity may be considered as cured when there is no adduction or inversion deformity, when there is a hollow on the dorsum of the foot in the position previously occupied by the head of the talus, and when the child is able to evert the foot and dorsiflex it to about a right angle.

2. **Treatment of Older Patients, Previously Untreated.**—At this stage manipulation requires to be carried out under a general anæsthetic. The foot is manipulated with a Thomas's wrench, but the various movements are the same as those effected manually in a younger child. Once the deformity is corrected, physiotherapeutic measures are used to mobilise the joints and to develop the muscles.

In the intervals the foot is kept in the corrected position by means of a Jones' club-foot splint of light aluminium. When the child is allowed to walk, the outer side of the sole of the shoe is raised to maintain the foot in good position. The use of the aluminium splint should be continued during the night for many months.

**3. Treatment of Old and Relapsed Cases.**—In many cases manipulative treatment is not sufficient because of rigidity or a constant tendency to relapse. In this type of case an operation is necessary.

After a tourniquet has been applied to the leg, an incision is made on the outer side of the foot, through which the plantar fascia and muscles are exposed and detached from their origins as far backwards and inwards as possible. A second incision is then made on the inner side of the foot and the remaining attachment of the muscles completely erased. The tendon of the tibialis anticus may require lengthening. The inferior surface of the tarsal bones is then completely stripped of soft tissues. The scaphoid is mobilised by dividing the ligaments on its medial, inferior and, if necessary, its superior surfaces. The internal lateral ligament of the ankle and the calcaneo-scaphoid ligament are also divided. The foot may then be manipulated to bring the scaphoid in front of the head of the astragalus. The incisions are closed and plaster applied with the foot just short of full correction. Complete correction may be secured a fortnight later, when the plaster is removed and the foot manipulated without removing the stitches. The tendo achillis may be tenotomised subcutaneously at the time should this be necessary.

**4. Treatment in an Adult.**—In the adult, an operation on bone is necessary in most cases to obtain any satisfactory degree of correction. A wedge of bone is removed from the os calcis behind the midtarsal joint, a similar wedge from the cuboid in front of the joint, and finally a curved wedge with its base upwards and outwards from the head and neck of the astragalus. After division of the astragaloscaphoid capsule and the internal lateral ligament, it is usually possible to manipulate the foot into the correct position without undue force. The foot is encased in a plaster reaching to the knee for six weeks.

In many cases stabilisation of the foot by arthrodesis of the midtarsal and subastragaloid joints after the manner of Naughton Dunn will be followed by good results (p. 1151).

## PES PLANUS

Flat-foot is a condition in which there is a persistence of the passive, or resting, attitude of pronation during periods of active movement. There is a consequent loss of the longitudinal arch of the foot. The attitude of passive pronation becomes more persistent and movements restricted, until eventually there is a fixed deformity. The foot is not flat because its key-stone has sunk, but because its arch is lowered as a consequence of lateral displacement, or so-called abduction. Every grade of severity in the disability and deformity is found.

Flat-foot may be hereditary, but it may supervene on any condition



associated with weakened muscles. The predisposing causes are ill-fitting shoes, bad methods of walking or loose methods of standing, while among the intrinsic causes are congenital and acquired abnormalities, as in rickets in early childhood and muscular weakness in quickly growing adolescent girls. A common cause is strain of the foot from prolonged standing or walking, as is so frequently seen in nurses. Over-weight may be caused in two ways ; either by obesity, the common way, or by the carrying of excessive weights, as is seen sometimes in the brewer's drayman.



FIG. 599

The abduction of the foot in pes planus showing how the os calcis deviates outwards.

If a straight line is prolonged downwards from the centre of the leg, most of the astragalus and os calcis will be lateral to it ; hence the body-weight, pressing on the medial side of the foot, tends to flatten the arch and cause outward rotation—tendencies which are antagonised by the flexors of the toes and by the tibialis posticus muscle. The os calcis is pronated and pushed laterally. The astragalus moves over the os calcis and the ligaments are stretched

*Symptoms.*—In the lesser degrees of the deformity it will be noticed that the feet are hot and uncomfortable and that they perspire freely after use. Stiffness and lameness follow. After a heavy day's exercise patients are particularly unhappy, while they are comfortable after a day of rest. The method of walking becomes inelastic and clumsy. Patients walk with their feet everted and are unable to rise on the toes.

Pain is severe when standing and may be experienced in several places—under the tubercle of the scaphoid, from stretching of the inferior calcaneo-navicular ligament ; below the internal malleolus ; along the astragalo-calcanean joint ; and down the inner surface of the os calcis. In some cases the tip of the external malleolus and the outer surface of the os calcis may be painful, as well as the dorsum of the foot. Pain is due to stretching of ligaments and compression of the tissues below the external malleolus. Synovitis is not uncommonly present in the tendon sheath of the tibialis posticus or of the peroneus longus. Localised swelling of the foot is common and in some cases oedema occurs.

The gait is clumsy, since the patient commonly avoids raising the heel, so preventing the thrust on the tarsal and metatarsal ligaments. Outward rotation of the feet and legs is a favourite method of preventing strain on the plantar ligaments (Fig. 600). He shuffles forwards and the foot is turned over its inner border instead of over the balls of the toes, thus further flattening the arch.

Muscle spasm is common, especially in the peroneal muscles, which may be tightly contracted and can be seen standing out under the skin.

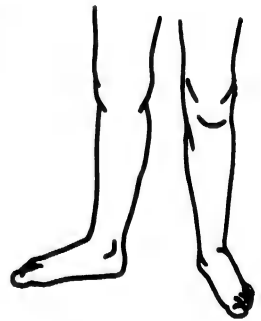


FIG. 600

Pes planus showing the outward rotation of the leg—the position used in walking.



Attempted correction induces pain along the course of these muscles. Painful corns may form in the distal weight-bearing areas, *i.e.*, under the heads of the metatarsals.

*Types of Flat-foot.*—Flat-foot may be divided into four stages, although these merge into one another without any sharp line of demarcation (Fig. 601).

1. **Voluntary Flat-foot.**—In this type the deformity is corrected when the foot is raised from the ground.

2. **Resistant Flat-foot.**—In this type the deformity may be corrected by manipulation.

3. **Rigid Flat-foot.**—In this type an anæsthetic, and possibly even a Thomas' wrench, is required before the deformity can be corrected.

4. **Permanent Flat-foot.**—In this type no amount of manipulation, even under an anæsthetic, will restore the arch. If correction is essential it has to be obtained by an operation, usually implying the removal of bone.

*Treatment*—1. **Paralytic Flat-foot.**—In this type, treatment should be directed towards the strengthening of the weakened muscles by means of exercise, electricity and massage. To maintain a correct position suitable braces are applied. In some cases of paralytic flat-foot it may be advisable to ankylose the ankle joint by operation. Such a bone operation, however, is not indicated before the twelfth year. It may be possible to transplant some actively functioning tendon to help restore the balance of the foot.

2. **Static Flat-foot.**—The object of treatment in this case is to correct the abnormal centre of gravity and to remove the pressure symptoms, the indications being pain and impaired function.

*Methods of Treatment.*—1. **The footwear** of the patient should be carefully examined and, where necessary, corrected. The shoe should have a slightly concave inner border, an accurately moulded waist and in most cases the body-weight should be transferred to the outer border of the foot by means of an inner wedge of leather. Care should be taken in the case of growing children that they do not outgrow, rather than outwear, their shoes.

2. **Physiotherapeutic Treatment.**—Exercises, both in the sitting and weight-bearing positions, form an important part of treatment. They are performed twice daily, and in children may be modified and carried out to music, so that they are less tedious than they would otherwise be. The exercises are directed towards the stretching of shortened structures and to the strengthening of weakened muscles. In some cases faradic stimulation of the small muscles of the foot is most effective in increasing their tone. Contrast foot-baths of cold and hot water are useful and stimulating.

3. **Supports for the Arch.**—Sponge rubber pads afford a resilient support and increase the spring of the gait. They are easily cleaned, retain their resiliency for a long time and are comfortable to wear.



FIG. 601

Imprints of various degrees of pes planus.

In some of the minor degrees adhesive plaster strapping is a desirable means of support. It should be changed every week until the symptoms disappear. The adhesive strapping is carried from the outer side of the foot under the central part of the longitudinal arch and fixed to the antero-internal surface of the leg in such a way that its anterior edge can be made smoothly adherent to the dorsal surface of the instep.

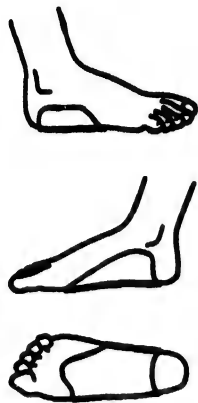


FIG. 602

Whitman's valgus spring.

One of the most efficient appliances is the Whitman valgus brace (Fig. 602) which, since it clasps and holds together the weak part of the foot, serves effectively to restrain deformity and to ensure such an attitude that the patient cannot avoid using the muscles which adduct and invert the foot. The brace is made for each individual from a cast of the foot, and consists of duralumin or aluminium.

**4. Instruction in Walking.**—The patient should be taught to walk with the feet parallel, as the muscles supporting the arch are then more active and produce adduction and inversion of the foot. The heel-and-toe walk also brings strong muscles into play and should be cultivated.

**5. Manipulation.**—Many cases of spastic, or rigid, flat-foot must be manipulated to produce mobility before other treatment is instituted. In some cases the manipulation has to be carried out forcibly with a Thomas' wrench, but in other cases the operator's hand is sufficient. The foot is forced downwards, then inwards, then upwards into extreme varus and an attempt is made to get the outer border of the inverted foot up to a right angle with the leg. The arch is completely restored and a plaster-of-Paris case applied from the toes to the tibial tubercle, with the foot in a position of talipes equinovarus. Walking in the plaster case is allowed as soon as the patient wishes.

**6. Operative Treatment.**—Spastic flat-foot is improved by tenotomy of the peroneal muscles, followed by manipulation and a plaster-of-Paris case. In the very extreme degrees an astragalo-scapoid arthrodesis may be carried out and a wedge removed from the prominent navicular and inserted into the region of the calcaneo-cuboid joint, so shortening the inner side of the foot and lengthening the outer (Fig. 603).

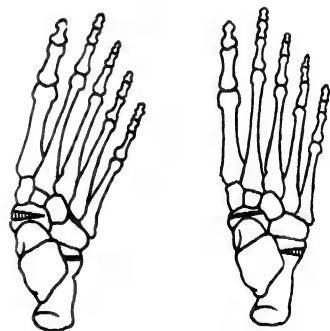


FIG. 603

The corrective operation for pes planus as explained in the text.

*Treatment of the Various Types*—1. **The Voluntary Type.**—Any cause that may be operating in the production of the deformity is removed, *e.g.*, obesity, strain, faulty attitudes. If there is extreme tenderness and œdema the patient should be put to bed for two or three weeks. Thereafter the footwear may be inspected and properly fitting shoes prescribed. He should be taught to walk properly. Physiotherapeutic exercises are ordered and the feet sup-

ported for a few weeks by adhesive strapping, after which a sponge-rubber arch support may be worn.

2. **The Resistant Type.**—The treatment of this is similar to the last, but instead of the sponge rubber a Whitman valgus brace may be recommended.

3. **The Rigid Type.**—The foot is manipulated under anæsthesia until it is flaccid and mobile. It is then put up in plaster in a position of talipes equinovarus for five weeks and thereafter treated as the second type.

4. **The Permanent Type.**—This requires operative interference, usually a bone operation, to correct the deformity. The treatment thereafter is on the usual lines.

#### CLAW-FOOT

The term claw-foot, or **pes cavus**, is applied to a deformity in which there is clawing of the toes combined with a raising of the longitudinal arch of the foot and shortening of the tendo achillis. It may be either congenital or acquired, but even in the former it is not usually apparent until the child is 6 or 7 years old. It may be associated with spina bifida occulta.

Claw-foot frequently follows an attack of poliomyelitis or progressive lesions of the central nervous system, but most commonly no cause for it can be assigned, and in these cases it is called idiopathic claw-foot (Fig. 604).

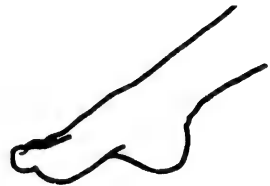


FIG. 604  
Pes cavus

In the early stages the principal sign is a relative weakness of the dorsiflexors, but as the condition advances the plantar fascia appears to become too short for the bony structure of the foot, so that the arch gradually rises. The plantar fascia is felt to be tense and contracted and the deformity becomes visible. All the toes are dorsiflexed at the metatarso-phalangeal and flexed at the interphalangeal joints, so producing a well-marked hammer-toe deformity. The chief complaint is often directed to the painful corns which form on the flexed interphalangeal joints or on the points of the toes. The patient is easily tired.



FIG. 605

Tenotomy of the shortened plantar fascia in pes cavus.

In the minor degrees tenotomy of the plantar fascia (Fig. 605) and stretching of the tendo achillis, followed by the fitting of a metatarsal bar to the shoe, may give adequate relief. In more severe degrees the operation of Steindler is used. This is a muscle-slide operation whereby the structures attached to the under aspect of the os calcis are erased as far forward as the calcaneo-cuboid joint on the outer side and the astragalo-scapoid on the inner side, where they take up a new insertion. After erosion of the structures the foot is manipulated and the arch flattened. Plaster is then applied. An ingenious operation has been devised by Lambrinudi and is very successful. He believes the condition is due to a paralysis of the lumbrical muscles so that

the unopposed flexors flex the toes at the interphalangeal joints and the metatarso-phalangeal becomes hyperextended. Lambrinudi arthrodeses the interphalangeal joints and so allows the flexor to flex the whole toe and act as a sling to the head of the metatarsal. In the most severe degrees the high crooked arch can be corrected only by dividing the bones at the level of the midtarsal joint. Sometimes it is essential to remove a wedge of bone with its base at the dorsum of the foot. The wedge includes a considerable part of the head and neck of the talus. If the deformity is very rigid, with marked bony deformity, a reconstruction operation after the manner of Naughton Dunn is advisable.

### PAINFUL CONDITIONS OF THE HEEL

Pain in the heel is most frequently found in persons who stand or walk a great deal; hence the term, "policeman's heel." The pain is usually aggravated by use, and may be entirely absent during rest. The painful area can always be elicited by digital pressure. The condition may be traumatic in origin, the result of disease or merely static.

1. **Traumatic Disturbances.**—Pain in the heel resulting from trauma may be situated in the region of the insertion of the tendo achillis or on the plantar aspect. In the first instance it may be due to a tenosynovitis of the tendo achillis, in which case there is swelling from effusion and often a fine crepitus.

A bursa situated close to the insertion of the tendo achillis is liable to inflammation from the friction of ill-fitting shoes. In this case there is localised tenderness at the site of the bursa and sometimes fluctuation may be detected.

An adventitious bursa may be produced over the prominent part of the os calcis posteriorly, usually on the outer side.

In all these conditions rest is essential. In some instances it may be sufficient to raise the heel about half an inch more than usual, so preventing any strain on the tendon. Bands of adhesive plaster are also useful adjuvants.

In chronic cases, further treatment may be required in the way of excision of the bursa.

*Calcanean Spurs.*—The plantar fascia arises from the tuberosity of the os calcis. Where there is an undue strain or pull on this fascia slight separation of the periosteum may result. From the stimulation of trauma or infection, or both, osteogenesis occurs more actively, with the formation of new bone leading to the production of a spur. An adventitious bursa may develop over this spur.

The clinical features are pain, tenderness, swelling and a limp. Usually the onset is gradual, but it may be sudden, as when a spur is broken by violence. An X-ray photograph may, or may not, reveal the spur, depending on the duration of the condition and the density of the bone. Many painful heels are seen in which X-ray examination fails to reveal any abnormality.

The differential diagnosis usually rests between osteoma, flat-foot and subastragaloid arthritis, but periostitis, bursitis, epiphysitis, and simple injuries (fractures) must also be considered.

Any obvious focal lesion should be dealt with, such as gonococcal infection or infected tonsils and teeth. If pain is acute, rest in bed and fomentations are prescribed. After pain and tenderness have lessened, proper shoes should be ordered and felt or sponge rubber pads inserted to relieve weight-bearing on painful areas.

In some cases operative removal of the spur may be necessary.

**2. Painful Heel Due to Disease.**—Apart from trauma, pain in the heel may have its origin in organic disease of the bone or epiphysis. The infection may be tuberculous, syphilitic, pyogenic or may follow a general gonococcal or rheumatic toxæmia.

*Epiphysitis of the Os Calcis* may occur in boys between the ages of 9 and 13 years, and has to be remembered in the differential diagnosis.

**3. Static Disturbances.**—Many static disturbances produce pain on the inferior surface of the heel. Where unilateral, it is frequently caused by taking too much weight on one foot, as, for example, where the limbs differ in length. In a claw-foot the posterior part of the os calcis may be painful, due to the fact that the bone is much more perpendicular than usual and acts less as a resilient support for the arch of the foot and more as a direct continuation of the leg bones. Pain in this region is common in policemen and nurses. It is often found associated with a weak, flat foot.

### HALLUX VALGUS

In this deformity there is extreme abduction of the great toe. Moderate degrees are often seen, owing to the prevalent use of badly fitting shoes, but the condition is not usually considered a deformity until the metatarso-phalangeal joint has become greatly enlarged and a bunion has formed. There is then, usually, a partial subluxation of the joint. A bursa develops over the prominent head of the metatarsal, while a corn or callosity forms in the covering skin. The projecting bone, the bursa and the thickened tissues are collectively known as a *bunion*.

The other toes are usually displaced outwards, the forefoot is broadened and the metatarsal arch depressed. The deformity may be combined with a weak foot, although in many instances the longitudinal arch is of a normal height.

The chief sign is the outward displacement of the great toe with resulting prominence of the head of the first metatarsal. The deformity is aggravated by the pull of the tendon of the extensor longus hallucis, which is displaced outwards and lies stretched along the lateral border of the great toe like a bowstring. The symptoms are pain, swelling and redness. The pain may be due to bursitis, to arthritis, or to a digital neuritis.

The condition is often bilateral, although the pain and discomfort are frequently more marked on one side.

*Treatment.*—In mild cases relief is obtained by the provision of properly fitting shoes, which relieve pressure on the tender joint. None of the devices for holding the toe in an improved position have any

curative value, nor do they usually relieve the symptoms. If these are at all severe, operation should be advised.

Where the joint is moderately healthy and in the absence of arthritis, removal of the exostosis and bursa is usually sufficient to effect a cure, but where there is any degree of arthritis an arthroplasty of the joint should be carried out. A resection of the base of the proximal phalanx is done, thus preserving the weight-bearing part of the tripod of the foot. The extensor longus hallucis tendon may require to be lengthened. A flap of deep fascia containing the bursa is then turned into the space between the metatarsal and the phalanx and fixed there with catgut. Pulp traction is applied to the toe for about three weeks. Passive movements are begun after the removal of the plaster, and the patient is allowed to walk at the end of the fourth week.

### HALLUX RIGIDUS

This is a painful affection of the first metatarso-phalangeal joint, characterised by limitation of dorsiflexion. When a flexion contracture of the joint is present the name *hallux flexus* is applied. The joint is usually swollen from peri-arthritis and attempts at passive movement produce pain. Pain is also experienced when standing, and more particularly on walking. There is often a history of injury, such as "stubbing" the toe or kicking a hard object.

In minor degrees of the affection, great relief is obtained by restricting the movement of the joint by the insertion of a narrow strip of tempered steel between the two layers of the sole.

Where the condition is associated with a "weak foot" this deformity may first be corrected under anæsthesia and the foot retained in a corrected position by a plaster bandage. When arthritis is present, however, an operation on the lines of that used for hallux valgus is necessary, where the base of the phalanx is excised and an arthroplasty carried out by turning in a flap of soft tissues between the bone-ends.

### HAMMER-TOE

The deformity of hammer-toe consists of dorsiflexion of the proximal phalanx, plantar flexion of the second and flexion or extension of the distal. The second toe is usually affected, the head of the first phalanx being subjected to pressure by the toe-cap of the shoe, as a result of which it frequently shows a painful corn. Underneath this there is often an inflamed, or even suppurating, bursa. The condition is in many cases bilateral and may be associated with hallux valgus or pes cavus.

In young children the distortion may be overcome by repeated manipulation, the corrected position being maintained by strips of adhesive plaster passing over and under the affected toe and its neighbours. The use of digitated stockings and of wide boots is also beneficial.

In adults, operation is indicated in order that recovery may be certain and quick. Amputation is never performed, but an excision of the head and neck of the proximal phalanx allows the toe to be

straightened. The operation is carried out through an elliptical incision which excises the corn and underlying bursa.

### METATARSALGIA

Paroxysmal pain under the heads of the metatarsal bones is known as metatarsalgia or Morton's toe and is associated with a falling of the transverse arch (Fig. 606). It was first described by Morton as a neuralgia of the digital nerves from their compression between the heads of the metatarsals.



FIG. 606

A badly fitting shoe is the main offender in predisposing to weakness of the transverse arch and in producing pain. Women's shoes with high heels and narrow toes are particularly bad.

Metatarsalgia. On the left is a section of a normal foot, on the right a section showing how the heads of the 2nd, 3rd and 4th metatarsals sink with the dropping of the transverse arch.

The pain occurs in paroxysms and is often severe in character. It is situated beneath the arch and usually opposite the third metatarsal head, although its site varies. The most constant and reliable symptom is the desire to remove the shoe during the paroxysm. Callosities form under each of the metatarsal heads.

The object of treatment is to strengthen the arch, maintaining it in a corrected position while this is being accomplished. Any coexisting defects in the mechanics of the foot are treated, especially a weak foot, an abducted foot or a shortened tendo achillis. Some support is usually necessary for the arch, either a metatarsal bar or a metatarsal crescent (Fig. 607). In some cases a pad of piano-makers' felt or sponge rubber may be inserted under the arch and secured just behind the metatarsal heads by adhesive straps circling the foot.

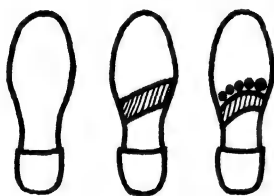


FIG. 607

Types of metatarsal bar. The crescent should fit under the necks of the metatarsals.

After the symptoms have been relieved, regular exercises, forced flexion of the toes to elevate the anterior arch, and massage and manipulation of the foot and toes should be practised.

In cases resisting all other forms of treatment, resection of the head and neck of the metatarsal bone has been recommended.

### INFANTILE PARALYSIS

Infantile paralysis is an acute infectious disease due to a filterable virus. It is seen most frequently in children, resulting in many cases in paralysis, especially of the extremities, abdominal wall and back. It occurs both in epidemics and sporadically, and appears to be more common in the summer months than at other times of the year. Although commonly termed "anterior poliomyelitis," a better term, in view of the cerebral infection which is frequently present, is "anterior poliomyelo-encephalitis." The infective agent gains entrance through the respiratory tract to the blood vessels or lymphatics,



by which it is carried to the spinal cord, resulting in injury to or destruction of the anterior horn cells. Toxæmia, œdema and hæmorrhage are the sequelæ of the infection with resultant damage to the nerve cells. These degenerate, so that there is often a complete loss of motor control in the parts enervated from the affected areas. The changes are most marked in the anterior horns of the lumbar and cervical enlargements.

Pain and tenderness, which may be severe, are due to lesions of the sensory ganglia. Replacement of the motor cells by scar tissue follows, with shrinkage of the whole anterior horn in severe cases.

*Stages of Attack*—1. **Acute Stage**.—The stage of onset usually lasts about a week, during which constitutional symptoms predominate.

2. **Subacute Stage**.—This lasts from one to four weeks, when the general symptoms subside but paralysis persists.

3. **Stage of Partial Recovery**.—This continues up to the end of the second year.

4. **Chronic Stage**.—This begins with the cessation of recovery.

The general symptoms are those of an acute infectious disease with fever, vomiting, constipation and prostration, accompanied by pain and tenderness in the arms and legs. If the lesion is situated high in the cord, the condition may resemble that seen in meningitis with retraction of the head. The child may go to bed, however, feeling perfectly well and wake up in the morning with an arm or a leg paralysed.

Suspicion is directed towards the condition from the period of the year at which it occurs, general indisposition, headache, sore throat, fever amounting to  $100^{\circ}$  or  $101^{\circ}$  F., drowsiness, vomiting and flushed face. There is usually an upper respiratory tract infection; irritability, hyperæsthesia, neck rigidity, convulsions, opisthotonos or unconsciousness may be present. Slight weakness of a limb may be the first sign of an impending paralysis. Kernig's sign is often present. The patellar reflex may be exaggerated at first and diminished or lost later.

Spinal puncture shows a clear, colourless fluid under moderately increased pressure. Cells, increased up to 1000 per c.c., are at first polymorphonuclear and later mononuclear leucocytes.

The paralysis is of the flaccid type; the part which is paralysed is limp and lifeless, but there is no alteration of sensation. It is purely a motor paralysis. One or both legs is most apt to be attacked, in this order, but any part may be affected.

When the infection spreads to the cerebrum, spastic paralysis may be present. The reaction of degeneration of nerves and muscles appears usually within a fortnight following the onset of paralysis. The reaction to faradism is reduced, while that to galvanism is sluggish and is greatest at the closing of the positive pole.

*Differential Diagnosis*.—This has to be made between encephalitis, peripheral neuritis, diphtheritic paralysis, cerebral paralysis in childhood and various local conditions such as epiphysitis, osteomyelitis, etc.

The *prognosis* will depend upon the extent of the area destroyed in the cord, the resistance of the patient and the treatment of the weakened parts.



*Treatment.*—1. **Prophylaxis.**—The condition is now notifiable, so that it is probable that large epidemics will in future be rare. Ordinary care and cleanliness, especially as regards nasal secretions, should be observed, particularly during the progress of an epidemic. Patients should be isolated and crowds avoided. One attack confers immunity. Treatment by convalescent serum and by vaccines is still in the experimental stage.

2. **Treatment During the Acute Stage.**—Intravenous injections of hypertonic salt solution cause a reduction in volume of the brain and cord, and are useful in the acute stage for lessening pressure on important nerve centres. Ten cubic centimetres of a saturated solution of magnesium sulphate may also be used with the same effect. Lumbar puncture is frequently employed to relieve pressure.

Complete rest is enforced during the early part of the paralytic period. Any movements or manipulations prejudice the chances of recovery. The patient lies recumbent and it may be advisable to apply a plaster shell to his body. Pain and tenderness may be relieved by local hot baths. Every effort should be made throughout this stage to prevent deformity, and every affected joint or limb is placed in a position of optimum functional utility and splinted to maintain this position.

When the acute symptoms have subsided an attempt is made to restore the greatest amount of efficiency to the atrophied muscles. Where there is any paralysis of the trunk it is wiser to keep the child lying on his back, but where the paralysis is limited to one, or even two limbs, the circulation may be favoured by allowing him to get up. To avoid deformity, however, some form of apparatus must be used. It not only prevents deformity, but may correct it. It certainly prevents the stretching of paralysed muscles and in many cases permits of or improves walking. An abduction splint may be used for the shoulder, a cock-up for the wrist, a walking caliper for hip and knee cases and other splints of similar function for other parts of the body.

The **active therapeutic measures** which are employed are massage, heat, electrical stimulation of muscles and muscle training. These should be carried out under a trained masseuse, who realises that the main essentials of treatment are the avoidance of stretching and fatigue of muscles and the prevention of deformity. Unfortunately the surgeon is often not called in until deformity is present.

Some of the various types of established deformity must now be considered.

**Hip-flexion Contracture.**—This disabling contracture is a result of contraction of the tensor fasciæ femoris, iliopsoas, sartorius and rectus femoris. It is usually associated with some adduction of the hip, a flexion deformity of the knee and often a shortening of the tendo achillis. In the early stages it may be cured by putting the patient in the prone position for some hours daily, but usually an open operation has to be carried out. The operation of choice is that described by Souttar, whereby the flexors of the hip are stripped subperiosteally from their original position and allowed to slip down the side of the pelvis. The thigh can then be extended and the

deformity completely corrected. It may in addition be necessary to divide the iliopsoas muscle. Where the anterior spine projects through the wound after the muscle slide has taken place, it should be cut off flush with the surface and the wound closed. The patient is afterwards placed in a plaster spica with the hip hyper-extended for two or three weeks until the wound has healed and the muscles have become united to their new attachment.

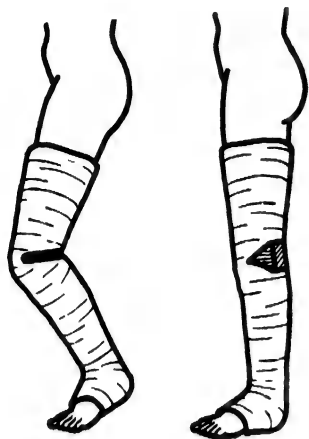


FIG. 608

Flexion deformity of the knee treated by a wedge plaster.

**Flexion Contracture of the Knee.**—This occurs commonly in association with flexion contracture of the hip and is often present in paresis of the anterior thigh muscles and overaction of the posterior group. The deformity can be prevented by a Thomas' knee splint, but when it has become established it is often possible to reduce it by means of a wedge plaster (Fig. 608). A circular plaster is applied to the leg, from the toes to the groin, in the position of the deformity and allowed to harden. Thereafter a transverse section of the plaster is made through the posterior three-quarters at the level of the knee joint. Thin pieces

of wood are then inserted into the slit to force it open, the leverage being so favourable that the knee can be gradually straightened out. When completely straight a few turns of plaster are applied to maintain the corrected position for some weeks.

**Deformities of the Ankle Region.**—Drop-foot is the commonest deformity and develops in cases of paralysis of the extensors of the ankle. It has to be remembered, however, that a slight contracture of the tendo achillis is an advantage in some cases of infantile paralysis where secondary shortening of the leg has taken place, as it compensates to some extent for this (Fig. 609). It is also useful in cases of quadriiceps insufficiency. When weight is borne in such a limb the strain on the gastrocnemius muscle locks the knee joint and increases the stability. The tendo achillis, therefore, should not be divided without careful consideration lest the stability of the leg be imperilled. The operation of lengthening is carried out through a vertical incision, about 6 to 8 in. long, on the medial aspect of the tendon. The synovial sheath is opened and the tendon exposed and divided into anterior and posterior halves by a long, lateral, vertical splitting incision. The anterior half is detached from its insertion into the os calcis while a transverse incision is made through the posterior half of the tendon at the upper end. In this way two broad flaps are secured. The foot is then brought into a right-angled position and the

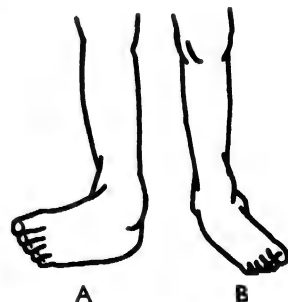


FIG. 609

Types of deformity in infantile paralysis.

A, left talipes calcaneo-varus; B, right talipes equino-varus.

two flaps of the tendon stitched together with chromic catgut. The sheath is stitched over the tendon and the skin incision closed. Immobilisation should be maintained in a plaster-of-Paris case for about six weeks, by which time the tendon should be united. Thereafter support may be obtained from the use of a strong boot.

**3. Treatment in the Chronic Stage of Infantile Paralysis.**—The object of treatment in this stage is to improve the function of the limb, and the operations used are grouped into two main types—those which improve muscle balance and those which secure stability. These operations are not carried out until two years have elapsed from the onset of the disease, as there is always a chance of some recovery of muscle power until that date. Muscle balance is improved by tendon transplantation—a normally functioning muscle taking the place of one which has become paralysed. In this way muscle balance is restored, deformity may be corrected or prevented and stability improved. An instance of such transplantation is in the case of quadriceps paralysis, when one of the flexor group is carried forward to take the place of the paralysed extensors.

In addition to using tendon transplantations, great improvement may be gained by bone operations—or stabilisation operations as they are called—whereby a flail joint is arthrodesed. In this way the shoulder, elbow, wrist, hip and knee may all be stabilised. There are many stabilising operations in use on the foot which from lack of muscle power has become flail, partially or completely. Often these operations are supplemented by tendon transplantation.



FIG. 612

Whitman's operation showing the backward displacement of the foot after removal of the astragalus.

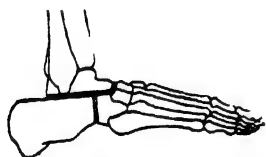


FIG. 613

Naughton Dunn's reconstruction of the foot.

talus, the whole of the scaphoid and the proximal cartilaginous surfaces of the cuneiform bones are then removed (Fig. 613). In this way there is a reconstruction of the forefoot in the nature of a shortening. The foot is displaced backwards at the subastragaloid joint. The



FIG. 610

The incision for arthrodesis of the tarsus



FIG. 611

Exposure of the ankle joint. The foot has been wrenched inwards and the upper surface of astragalus is shown.

**Whitman's Operation.** This operation is occasionally indicated in some deformities and particularly calcaneo-valgus. It is simply the removal of the talus through a lateral curved incision and the displacement of the foot backwards. Results are moderately good, but there is a definite shortening of the limb afterwards a serious disadvantage in a limb already short (Figs. 610, 611 and 612).

**The Naughton Dunn Operation.**—This is carried out through a lateral incision and consists of an arthrodesis of the subastragaloid and midtarsal joints. A portion of bone with the articular surfaces is removed from between the os calcis and the cuboid. The head of the talus, the whole of the scaphoid and the proximal cartilaginous surfaces of the cuneiform bones are then removed (Fig. 613). In this way there is a reconstruction of the forefoot in the nature of a shortening. The foot is displaced backwards at the subastragaloid joint. The

foot is put up in plaster for three months. In cases where there is a loss of power in the calf muscles an arthrodesis of the ankle joint may also be done.

### SPASTIC PARALYSIS

Spastic paralysis of infancy and childhood is due to disease or injury of the cerebral motor centres, affecting the upper motor neurones which control the muscles of the extremities. It is also known as Little's disease or infantile cerebral spastic paralysis. If one extremity is involved, the term *monoplegia* is used; when half the body is involved, *hemiplegia*; both legs, *paraplegia*; both arms and legs, *diplegia* or *quadriplegia*. The two latter types are usually congenital. The hemiplegic type often occurs during the first few years of life and usually follows disease. This may be antenatal in origin, but more often dates from the time of birth, which has usually been difficult and often carried out with the aid of forceps.

Frequently the first sign of any serious disturbance following a difficult birth may be a convulsion, indicating the cerebral origin of the disease. The mother may notice the child's difficulty in controlling movements of the extremities.

**The Paralysis** is of the upper neurone or spastic type and is characterised by the hypertonic condition of the affected muscles and the exaggerated reflexes. There is no wasting or reaction of degeneration. Muscular rigidity is marked and leads to spasm, particularly of the adductors of the lower limbs, when the child begins to walk. Any attempt to straighten the limbs is resisted, but they can be gradually stretched if pressure is maintained. Whenever the pressure is released the spasm returns.

The deformities which result depend upon overaction of the stronger groups of muscles. In the lower limbs the hips are flexed, adducted, and rotated inwards, the knees are flexed, and the feet are usually in a position of *equino-varus*. In the upper limb there is flexion of the elbow, the forearm is pronated, the wrist flexed and the thumb adducted and pressed into the palm by the flexed fingers.

Certain curious involuntary movements often develop and interfere greatly with the function of the limb. These are often in the form of rhythmical athetoid movements or may belong to the "perverse movement" group. They are limited to the affected limb and are usually more troublesome in the arms than in the legs.

Mental deficiency varies with the severity of the limb affections, but is present in all degrees. Epileptic seizures are commonly associated with hemiplegia.

There may be some impairment of growth in the limb.

A diagnosis has to be made between poliomyelitis, idiocy, cerebral tumour and hydrocephalus.

Without treatment the prognosis is poor, but mild cases improve considerably with treatment and even in severe degrees some amelioration may be expected.

*Treatment.*—Massage is worse than useless as it increases the tone of the already hypertonic muscles. Muscle re-education is the most

important part of the non-operative treatment. The patient is taught to use the weaker muscles. The hand is assisted by passive movements until the maximum of normal voluntary movement is reached, and then gently stretched before the limb is passively replaced in the flexed position. This cycle is repeated frequently. The patient is taught to carry out coarse movements of the limb before attempting the finer movements of the fingers. Movements may be performed to the accompaniment of a metronome or music.

**Operative Treatment.**—One does not usually operate until the age of 5 or 6 years, as children are unable to co-operate before that time. Mental enfeeblement, athetoid movements and epileptic seizures to some extent contraindicate operation.

The operations are divided into those on the nervous system and those on muscles and tendons.

The only operation of real value on the nervous system is that of Stöffel. This relaxes the spasm in a certain number of muscle fibres in each muscle by cutting out part of its nerve supply. Stöffel demonstrated that the various tracts run independently in a large nerve and that the position of any bundle is remarkably constant. Surgeons, however, do not now operate after this method but prefer to follow the nerve down till its individual branches can be traced. It is then easy to resect a number of the fibres going to any particular muscle and in this form Stöffel's operation has become firmly established.

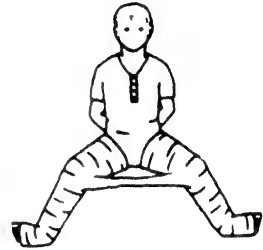


FIG. 614

Type of plaster applied after the operative treatment of adduction deformity of the legs

The operations on muscles and tendons include tenotomy and myotomy, tendon lengthening, excision of portions of tendons and muscles and transplantations.

**Adduction Deformity of the Hip.**—Although improvement may be secured by tenotomy of the adductor muscles, better results are obtained by division of the nerves after the method of Stöffel. The obturator nerve is approached either in the upper part of the thigh or through an abdominal incision. In the latter case the nerves on both sides may be operated on at one time. If the thigh approach is used, both the anterior and posterior branches may be divided through one incision. The type of operation depends upon the extent of the spasticity. After the operation the legs are manipulated into an abducted position and fixed in this position in a plaster-of-Paris case for some weeks (Fig. 614).

**Flexion of the Knee** may be treated by lengthening of the hamstring tendons when there is a permanent contracture of these muscles, but when this is due to spasm alone and can be corrected by pressure, a modified Stöffel operation may be carried out on the sciatic nerve.

**Pes Equinus.**—Here one must know the type of muscular shortening, whether it can be overcome by pressure or whether actual adaptive shortening of the tendons has supervened. In cases of organic contracture the tendo achillis must be lengthened, but if the

deformity is due to spasm alone, a Stöffel operation may be carried out on the tibial nerve, to paralyse to some extent the gastrocnemius and soleus.

A vertical incision is made down the centre of the popliteal space and the nerve isolated and traced down to the calf. Two branches leave the main trunk to supply the outer and inner heads of the gastrocnemius. There are also the branches to the dorsal portion of the soleus and the plantaris. The ventral portion of the soleus is supplied by a branch coming from the anterolateral aspect of the main nerve, while the nerve to the tibialis posticus is found on the posterolateral aspect.

In moderate cases of pes equinus the nerves to the heads of the gastrocnemius should be resected, and in more severe cases, one-half of the nerve to the dorsal portion of the soleus should also be excised. In severe cases the entire tract may be removed. The knee should be kept extended on a splint, with the foot at a right angle, for three weeks to allow complete healing of the wound.

## CHRONIC LOW BACK PAIN

Pain in the lower part of the back is so variable in its causes, its character and its treatment that it forms quite a problem to the examining surgeon, to elucidate which a very comprehensive method of investigation has to be undertaken.

This investigation will start with a careful description of the history of the complaint, particulars being recorded of the method of onset, the origin of the pain, its situation, radiation and duration. Accompanying complaints in other parts of the body are noted—such as in the feet, other joints or the genito-urinary system. Thereafter the patient is stripped and examined in both the erect and recumbent positions. Any abnormalities of posture, curves or movement are readily seen. Points of tenderness in the lower part of the back are very helpful in localising the anatomical position of the probable site of the pain. Finally, an X-ray examination is made of the suspected site and its neighbourhood, both an anteroposterior and a lateral view being necessary. In addition, in many cases an examination of the urine, blood and the cerebrospinal fluid may disclose significant features.

### LOW BACK PAIN ASSOCIATED WITH CONGENITAL ERRORS

The vertebral body is occasionally the site of some congenital error but it is unusual for this to be associated with low back pain. This error is usually developed from some deviation from the normal in ossification. The body may develop in two halves as there are two centres of ossification, and in some cases one half may entirely fail to grow with the production of a hemivertebra, but this is much more common in the upper part of the spine. Sometimes two vertebral bodies fuse together. This condition is usually symptomless.

The articular facets of the lumbosacral joints vary in shape and in

the plane of their surfaces, and when one lateral facet is directed backwards and the other medially it is thought by some that this may be a cause of low back pain.

**The Neural Arch.**—The principal anomaly occurring in this part is a lack of fusion between the two halves of the arch: this occurs commonly in the fifth lumbar or the first sacral vertebra, constituting the error of spinal bifida occulta. This is sometimes clinically evident by the presence of a small tuft of hair or a dimple in the skin at the region. Such a defect may produce instability in the lumbosacral region and low back pain.

**Spondylolisthesis** is the name given to the condition where one body—usually the fifth, occasionally the fourth or even the third lumbar—slips forward on the lower body, carrying with it the superincumbent lumbar spine. This is thought to be due primarily to a variation in the ossification of the affected vertebra where, instead of one primary centre for each half of the arch, there are two centres in each, the two parts being united by a plate of cartilage set obliquely between the superior and inferior articular processes. The apposition of the inferior articular process of the upper vertebra with the articular process of the lower one checks any forward displacement of the vertebra, and if there is any solution of continuity between the two articular processes displacement easily occurs. It is quite likely that the slip is initiated by trauma, either single or repeated.

The patient complains of backache and pain occasionally in the lower extremities, but in a great number of cases no such symptoms are present, and indeed the condition is usually symptomless and therefore often discovered by accident. The pain, when present, is relieved by rest and aggravated by hard work. It is of a dull aching character and the back feels weak and stiff, but only a few of the patients are aware of any deformity.

In the fully developed case the trunk is shortened and transverse skin creases are seen encircling the body between the ribs and the iliac crest. The lumbar curve is increased and the sacrum is prominent, while there is a still greater prominence of the fifth lumbar spinous process with a depression above it. Occasionally a slight degree of scoliosis is present owing to the unequal slipping forward of the vertebræ and the consequent rotation. Flexion of the spine is restricted in the immediate area, but it is a notable feature that such patients are usually able to touch their toes with their hands in the straight-leg bending exercise.

The diagnosis is confirmed by radiographic demonstration of the displacement. This can be seen even in the anteroposterior picture, which shows a characteristic arc or bow of the anterior border of the slipping body, while in the lateral view the vertebra is seen displaced forward on the sacrum or lower vertebra. A further characteristic feature is a break in the lamina which appears as a gap continuing the clear space of the lumbosacral joint backwards above the spinous process of the fifth lumbar. Often there is some bony buttress formation attached to the anterior surface of the sacrum under the projecting portion of the slipping vertebra.



Many cases have few symptoms and accordingly no treatment is indicated. In slight cases a conservative method of treatment is adopted by fitting the patient with a strong lumbosacral corset. Where the symptoms are extreme, operation is suggested—either a posterior arthrodesis, after the manner of Albee or Hibbs, or an anterior arthrodesis as recommended by Burns.

#### **Sacralisation of the Transverse Process of the Fifth Lumbar Vertebra.**

—This is a developmental anomaly where one or both transverse processes are abnormally large and strong. Occasionally they are so large that they form an intimate connection with either the upper part of the sacrum or even with the iliac crest. The condition is due to an overgrowth of the costal element, a centre of ossification additional to the normal one for each lateral process.

Symptoms usually start about the age of 18 to 20, when pain is complained of on the affected side low down in the back. Occasionally there is a history of trauma, after which the pain becomes almost continuous and is accompanied by a feeling of tiredness. Acute exacerbations occur. The patient is unable to sit comfortably on the buttock of the affected side and, as is usual with such pain, it is worse after exercise or when tired, but is improved by lying in bed or by the wearing of tight corsets.

Usually a certain amount of flattening of the lumbar curve is seen, and occasionally there is a lumbar scoliosis with the convexity to the affected side in unilateral cases. This remains even in the sitting posture. The sacrospinalis is tense. There is tenderness over the sacro-iliac joint on the affected side, and occasionally down the sciatic nerve.

An X-ray will confirm the enlargement of the transverse process, and a stereoscopic examination will show whether this abnormal process is articulating with the first sacral segment or with the ilium. The probable cause of the pain is a distraction of the sacro-iliac joint produced by the leverage of the abnormal process in lateral movement of the spine.

Many cases are helped by the fitting of a tight sacro-iliac corset, but in some cases it is necessary to operate and remove the affected process, or at any rate the terminal part of it, which is forming the abnormal articulation.

### **LOW BACK PAIN ASSOCIATED WITH TRAUMA**

#### **SACRO-ILIAC STRAIN**

The condition to which the term *sacro-iliac strain* is applied occurs when a mechanical force or injury forces the joint to one or other of the extremes of range of movement, and the joint then becomes locked. The symptoms vary considerably, but pain is usually experienced over the posterior aspect of the joint and may be elicited by digital pressure in that area. It is increased by movements which put added strain on the joint, and is more severe at night because the normal lumbar lordosis is obliterated and more strain is then thrown on the joint.



The pain is increased by the menstrual periods and by standing for long periods. Usually the body is inclined away from the affected joint. In stooping flexion of the trunk is avoided and in walking short steps are taken. Movements of the body on the thighs, or of the thighs on the body, are limited, while straight-leg bending is much restricted..

Among the symptoms stated by Albee to be pathognomonic of sacro-iliac disturbance are: pain in the joint on turning over from the recumbent position; pain while sitting on a hard chair, relieved by sitting on the opposite buttock; pain in the affected joint on forward bending; tenderness on deep pressure over the joint; and listing of the whole spine to the opposite side.

There are no characteristic X-ray changes in sacro-iliac strain until the condition has become so chronic that arthritis has set in, when the usual signs of an osteo-arthritis may be seen.

The usual treatment for such a condition is manipulation, but care should be taken to exclude the possibility of tuberculosis or other abnormality before this is undertaken. Usually a single manipulation produces a dramatic result in the acute case, but in chronic lesions other etiological factors, such as postural defects, gross overweight and so forth have also to be considered. In cases where manipulation fails to improve the condition, it may be necessary to arthrodesse the joint after the manner of Smith-Petersen.

#### LUMBOSACRAL STRAIN

Lumbosacral strain occurs in both acute and chronic forms. The acute form may be caused by a sudden blow forcing the joint into positions beyond the normal range of movement, or by any sudden movement whereby the muscles are caught off their guard and the ligaments thus sustain the full force of the injury.

The chronic form is usually slow in onset, but may follow an acute strain which has been unrecognised and untreated.

In acute cases with a history of recent trauma the pain and tenderness are situated at the lumbosacral junction, and the movements of the spine are restricted in all directions. A lumbosacral case will bend forward freely, whether sitting or standing, because he holds the lumbosacral region rigid and flexes chiefly at the hip joints.

In the chronic case the symptoms vary, often the patient complaining only of a "weak back." Frequently there is a history of intervening periods of comfort lasting several years between attacks of pain, but gradually the attacks become more and more frequent and constant as age advances.

In the acute stage rest in bed for a few weeks is essential. The patient lies in a bed fitted with fracture boards with pillows placed beneath the knees and lumbar spine. When the acute symptoms have subsided, massage, radiant heat and diathermy are of assistance. The chronic cases are more difficult to treat, and in them it is necessary to seek the underlying cause, such as a postural defect, increase of weight or active toxic foci. Thereafter exercises to increase muscle tone and

improve the posture form the essential local treatment. Where the abdomen is pendulous it is helpful to fit a support of the lumbosacral belt or strong corset type.

In severe cases of long-standing, which may have failed to react to conservative treatment, the joint may be arthrodesed in a manner similar to that described for tuberculous disease of the spine.

## LOW BACK PAIN ASSOCIATED WITH PATHOLOGICAL CHANGES

### SPONDYLOSIS DEFORMANS

In such cases the spine as a whole undergoes progressive deformation, and widespread osteophytic formation occurs. There is considerable interference with the mobility of the spinal column, and pressure is likely to be enforced on the nerves in relation to it. It occurs principally in men and especially those who have had a strenuous occupation, such as outdoor labourers, miners, etc. In the early stages the back is uncomfortable and the patient has difficulty in carrying out certain movements. There is a history of frequent attacks of lumbago, the pain never completely disappearing and one attack merging into the next. With each successive attack the spinal symptoms become more marked and the movements more limited. Pain is worse in the morning when the patient gets out of bed.

If the patient is seen for the first time only after deformity has developed, attempts should be made to correct this gradually, by rest in bed on appropriate pillows. Frequently manipulation of the spine is helpful in the early stages when there are more adhesions than osteophytes. This is followed by hot baking, either by fomentations or radiant heat, and exercises. The more the patient exercises the more likely is he to retain movement in the spine.

### SPONDYLITIS ANKYLOPOIETICA

This is probably primarily an infective condition of the small spinal joints which is later characterised by deposition of lime in the ligaments. The condition commonly affects young males, but its exact etiology is unknown, although apparently active toxic foci in this part of the body may have some relationship.

In the early stages the patient complains of muscle and joint pains. Later his general condition deteriorates and he loses weight and gets increasing stiffness and deformity of the spine. The sacro-iliacs are usually the first joints affected, there being osteoporosis in the early stages and, later, sclerosis and ankylosis. The whole spine becomes fused into a solid bony column, and in the late stages the costovertebral joints are equally affected, with the result that only abdominal respiration is possible.

Treatment is directed in the first place to obvious toxic foci which are eradicated. The pain is controlled by means of deep X-ray therapy. This appears to have a very beneficial effect on the disease. At the same time the optimum position for ankylosis is obtained by gradual

reduction of the deformity by means of pillows under the dorsal region. When the X-ray treatment is finished and the deformity of the spine reduced as much as possible, a light plaster jacket is applied—of the same type as is used for a recent spinal fracture—that is to say, it extends from the suprasternal notch to the pubis in front but a much shorter distance behind, so preventing any forward flexion of the spine. This reduction in the deformity can only be achieved in the comparatively early stages of the disease.

### FIBROSITIS

Fibrositis, or lumbago as it is commonly called, occurs in both acute and chronic forms. It is a non-suppurative inflammatory reaction of the fibrous supporting tissues of this part of the body. Little is known of the etiology, but it may be due to focal sepsis, an error in diet, some defect in metabolism, or to climatic conditions. Acutely tender areas in the erector spinae or its attachments are found, and at these parts it is usual to find enlarged fibrositic nodules. It is important, however, to exclude all other causes of low back pain before a diagnosis of fibrositis is made. There is often an associated referred sciatica—a further manifestation of the presence of rheumatic disease.

In regard to treatment, the first thing obviously is to eliminate any of the possible etiological factors. In the acute case the patient must be put to bed to ensure complete rest. The bowels are well opened, a light diet is prescribed and the patient is instructed to drink large quantities of bland fluid, this amounting to at least 4 or 5 pints a day. The pain may usually be controlled by 10 gr. each of sodium salicylate and sodium bicarbonate four-hourly for a few days.

Local treatment of the acute type consists of local heat from an infra-red lamp, hot bottles, or even from a hot iron. In some cases, where the condition is limited to a few localised tender areas, infiltration with  $\frac{1}{2}$  per cent. novocain is of value. Twenty to 50 c.c. may be used and the treatment followed by vigorous massage and active back exercises.

### LOW BACK PAIN ASSOCIATED WITH STATIC OR POSTURAL ERRORS

Postural errors, either habitual from occupation or from the presence of such abnormalities as weak feet or excessive weight, form a large proportion of the cases of low back pain, and as well as being probably the most frequent cause they are the most difficult to treat. Postural strain is precipitated by certain occupations. Surgeons and dentists who have to bend over their work for long periods are specially liable. Another of the common causes is a sagging or protuberant abdomen, which by its weight and its downward and forward pull tires out the muscles and leads to increased tension on the ligaments supporting the lumbar spine. The obvious treatment for such a condition is reduction of weight, and therefore of the size of the abdomen, by dietetic methods combined with active exercises designed to increase the tone and control of the abdominal muscles. Failing this the strain

may to some extent be relieved by supporting the abdomen. In fitting abdominal supports it should be borne in mind that the strain is not diminished if the belt is of equal width at the back and at the front. To be of real benefit the support should extend well above and well below the lumbar spine, while its abdominal width must be greater than that at the back.

Many cases of chronic back strain are caused by deformities of the feet or the knee. The feet, therefore, should be carefully examined and abnormalities such as flat foot or valgoid deformity corrected.

### **LOW BACK PAIN REFERRED FROM OTHER REGIONS**

Besides local causes, backache may be due to errors in different regions. Osgood discusses four types :—

- (a) General debility, with mental or physical fatigue.
- (b) Gynæcological and genito-urinary lesions.
- (c) Neurological lesions, such as spinal cord tumours.
- (d) Imperfect mechanical conditions in the lower limbs, especially faulty posture of the feet as referred to above.

In all cases where the etiology appears to be obscure, these conditions have to be considered and eliminated.

### **LOW BACK PAIN FROM A COMBINATION OF CAUSES**

This is probably the most important feature in difficult and persistent cases. Numerous combinations occur, as, for example, postural strains with susceptible bodily form or with anatomical variations ; or postural and traumatic strains superimposed on a pre-existing hypertrophic arthritis. Such a combination of errors demands a combination of methods of treatment.

WALTER MERCER.

## CHAPTER L

### DISEASES OF THE MUSCLES, TENDON SHEATHS AND BURSÆ

#### MUSCLES AND TENDONS

##### INJURIES

**SUBCUTANEOUS INJURY.** Contusions and sprains are due to falls, blows or violent muscular effort. They are commonly seen in men, who are taking strenuous exercise after a period of inactivity without graduated training, and are therefore common among athletes at the beginning of a season or in workmen beginning heavy work after a period of idleness. The muscles chiefly affected are those of the back or lower limb and at the moment of occurrence the patient experiences a sharp, stabbing pain. The muscle is bruised or a few of its fibres are torn across and a hæmorrhagic effusion of varying amount takes place among the muscle bundles. Active movement brings back the pain, but gentle passive movement is tolerated until the muscle is stretched; there is tenderness over the site of the lesion.

*Treatment* is directed towards relaxation of tension and immobilisation. The limb is placed in that position which will best relax the injured muscle, the whole area is firmly strapped with adhesive plaster and the patient kept at rest for five days. The strapping is then removed, the limb bandaged firmly and massage and electrical treatment given. Sound healing must be obtained before any further strenuous exercise is allowed, as otherwise a weak scar forms, which predisposes to a condition of recurrent sprain.

**Hernia of Muscle** results from similar injuries, the sheath being ruptured and the muscle fibres projecting through the gap thus formed. It is occasionally seen in the biceps muscle of the arm and in the adductor muscles of the thigh. The opening is small, causes little interference with function and treatment is rarely needed. If there is any incapacity the gap should be closed with a living suture of fascia lata or by a fascial graft.

**Dislocation of Tendons.** Tendons, which alter their direction on passing over a joint, are held in position by bands of fibrous tissue. Dislocation of the tendon from its bony groove will occur when this band is ruptured, as the result of a severe strain. The tendons commonly affected are the peroneus longus, the long head of the biceps and the extensors of the thumb. The accident is accompanied by a sharp pain and a sense of weakness in the limb. The tendon can be felt to

slip from its groove when the joint is moved and there is local tenderness over the ruptured band. In long-standing recurrent cases the tendon can be felt, or even heard, to slip in and out of its groove.

*Treatment* consists in reduction of the dislocation and immobilisation of the joint in the position of relaxation with a light plaster-of-Paris bandage for four weeks. This is followed by massage and graduated exercises and leads to a permanent cure. The recurrent cases require an exposure of the tendon, which is retained in its groove by a fascial or a periosteal graft.

**Rupture of a Muscle or Tendon** is produced by a sudden violent contraction of the muscle. The rupture occurs in one of four situations, either in the muscle belly, at its junction with the tendon, in the tendon or at the attachment of the tendon to the bone.

Rupture of a muscle is seen in men engaged in hard manual labour or during some form of athletic exercise. It usually takes place at the junction of tendon and muscle and is due to violent ill-balanced action applied suddenly. The muscle belly itself may be torn, but in such cases it has previously been weakened by disuse or localised disease, *e.g.*, a gumma. The muscle fibres contract and the gap is filled with blood clot. If no operation is performed a broad scar forms, which leads to loss of function and subsequently to contracture, unless great care is taken to prevent it. On examination the gap between the torn ends can easily be felt and it is increased in width when the muscle is made to contract, while the muscle itself becomes rounded and more prominent. Fluctuation may be detected in the gap from the presence of blood.

*Treatment* consists in early operation, when the blood is removed and the tear sutured. The limb is put up in a position allowing maximum relaxation of the affected muscle for seven days, after which gentle massage and movements are begun. If operation is refused the results are unsatisfactory, as adhesions may form to surrounding structures whereby function is further restricted.

Tendons are either torn from their insertions or ruptured. When the injury occurs the patient often imagines that he has been hit, so sharp and so localised is the pain, and if running he may fall down. The condition is recognised by the gap in the normal position of the tendon produced by the retraction of the muscle and by the absence of the movement which it normally produces, although the muscle itself can be felt to contract.

*Treatment* is immediate suture, the results of which are excellent. The limb must be immobilised for five days, after which massage and graduated movements are continued for two weeks.

The following muscles and tendons are those most frequently ruptured :—

1. The **biceps muscle** is ruptured at the junction with its tendon above the elbow. The tendon itself may be torn from the radial tuberosity and the long head may rupture inside the shoulder joint, especially if it is weakened by acting in an osteo-arthritic joint.

2. The **extensor tendons of the fingers** are injured close to their

attachment to the base of the distal phalanges. This condition is essentially associated with ball games and is very common in cricket, when a fast moving ball hits the tip of the finger. The base of the phalanx may be fractured at the same time, but rupture of the tendon does occur apart from fracture. Unless sound healing takes place the terminal interphalangeal joint is permanently flexed, a condition known as "mallet finger" (p. 971). Treatment consists in immobilising the finger so that the metacarpo-phalangeal and proximal interphalangeal joints are flexed and the terminal interphalangeal joint fully extended. This position is maintained for three weeks and care taken to protect the finger from further injury for an additional three weeks.

3. The **sternomastoid muscle** may be torn at childbirth, leading to torticollis (see p. 356).

4. The **rectus abdominis muscle** is occasionally ruptured during the spasms of contraction in tetanus and rarely by severe coughing.

5. The **erector spinæ muscles** are torn as the result of a patient attempting to lift heavy weights. The tear usually occurs in the lower dorsal and lumbar segments of the muscle. Pain and stiffness in the back often persist for many months and adhesions may form, necessitating manipulation under an anæsthetic before a return to full movement is obtained. This class of injury is a common cause of litigation under the Workmen's Compensation Act.

6. The **adductor longus** and **adductor magnus** are occasionally injured during riding and ski-ing. The former muscle is partially detached from its pubic origin and the latter is torn from its insertion into the femur. The injury to the adductor longus is apt to become a chronic recurrent one, especially in middle-aged people who take short periods of active exercise in the midst of a sedentary life.

7. The **extensor quadriceps muscle** of the thigh is ruptured by a sudden violent contraction of the muscle in an attempt on the part of the patient to regain his balance after a slip or fall. This type of injury may result in a fracture of the patella, a rupture of the muscle just above the patella or a detachment of the patellar ligament from the tibia. The fractured patella is recognised by the presence of two or more fragments. When the muscle is ruptured, the patella is intact and is separated from the muscle by a wide gap through which the anterior surface of the femur can be felt. If the ligament is detached from the tibia, the patella is drawn up into the thigh and the contours of the bones of the knee joint can be easily identified beneath the skin. Treatment of all these conditions is immediate operation, at which the joint is cleansed of blood clot and the tear repaired by careful suturing. The leg is placed in a divided plaster case or on a back splint and massage and faradism started on the third day. Patients should not be allowed to walk for three weeks.

8. The **plantaris tendon** is snapped by a sudden movement, and from its close association with games this injury has been termed a "tennis leg." The patient often thinks he has been struck by a stone in the back of the calf. The leg becomes painful and swollen, active plantar flexion of the ankle increases the pain and within twenty-four hours bruising appears in the popliteal space and in the back of the leg.

Firm strapping of the leg for seven days, followed by massage, rapidly relieves the symptoms. The patient need not be kept in bed.

9. The **tendo achillis** is sometimes ruptured in athletes and dancers at the beginning of training by sudden sharp movements. Rapid return to function can be obtained only by suture of the tendon.

**Division of Muscles and Tendons in Open Wounds.**—A muscle may be injured in a penetrating wound and, if the damage is extensive, a cavity is formed by retraction of the muscle fibres. This will be filled by blood clot which is likely to become infected. Such wounds should be carefully explored before the skin is sutured, and if the muscle is damaged it should be sutured and the wound closed with drainage.

The division of tendons is usually seen in the front of the wrist and in the fingers, being the result of a clean cut with a knife or of falls on pieces of broken glass or china. The condition is diagnosed by the loss of movement normally produced by the tendon in spite of the active contraction of its muscle and by the position in which the parts are held. When one tendon is severed, its opposing muscle contracts and pulls the joints into the position of its full action; for example, if the flexor tendons of a finger are divided the extensors hold the finger in full extension. After the injury, the proximal end of the tendon is retracted for a considerable distance by spasm of the muscle, whereas the distal end is lying in the lower surface of the wound or just inside its sheath.

*Treatment* follows the general lines of wound technique. It must always be borne in mind that other important structures, such as nerves, may be injured and the wound must therefore be explored to discover the exact extent of the injury. If more than one tendon is severed, the corresponding ends of each must be identified and care taken to distinguish the end of tendon from that of a nerve. Apposition is obtained by mattress sutures of silk or catgut introduced some distance from the cut end to prevent them cutting out. The limb is then put up in a position allowing full relaxation of the affected tendon for ten days.

When tendons are divided *inside* a synovial sheath, it is considered unwise to attempt primary suture, as the necessary exposure predisposes to infection in the sheath with resultant adhesions and fixation of the tendon. Some weeks later secondary suture is performed and the tendon surrounded with "amnioplastin." The results are extraordinarily bad.

Tendons are sometimes torn away from their attachment to muscles as the result of an injury in which a part of the body is dismembered. This is exemplified by "avulsion of a finger," which is produced by the digit being caught in the moving parts of a machine and wrenched from the hand. The flexor tendons are torn away from their muscle attachment and remain attached to the finger. Special attention to the danger of infection is needed, for the tunnels occupied by the tendons in the hand and forearm fill with blood clot and infection may ascend from the wound.



## INFLAMMATORY DISEASES OF MUSCLE

**Simple Myositis** follows minor injuries which bruise the muscle fibres. Pain on movement and localised tenderness persist for a few days.

**Acute Suppurative Myositis.**—Infection may reach the muscle by direct implantation in penetrating wounds, by extension from a neighbouring focus and by metastasis in pyæmia. The muscle becomes painful, swollen and tender, and all movements increase the pain. If drainage is not established, pus will track rapidly throughout the limb.

*Treatment* is by incision and drainage.

**Chronic Myositis.**—**Tuberculous Myositis** occurs only as a complication of tuberculosis of neighbouring structures. The psoas muscle is involved in spinal caries and the sternomastoid is invaded by the spread of infection from cervical glands. In the former case the pus tracks down the whole length of the muscle inside its sheath, but in the latter a localised induration appears and this may break down into a small abscess.

The *treatment* is directed primarily to the cause.

**Syphilitic Myositis** is of three types. Firstly, in the secondary stage transient pain and tenderness may affect one or more muscles; secondly, early in the tertiary stage a diffuse fibrosis may arise insidiously in several members of a group of muscles, leading to stiffness and finally to contractures; thirdly, later in the tertiary period localised gummata may occur in any individual muscle, those of the tongue and the sternomastoid being common examples. A gumma forms an indurated rounded swelling which is neither painful nor tender, and which eventually involves the surface epithelium and leads to the typical ulcer. The history of the original infection, the Wassermann reaction and the response to treatment establish the diagnosis.

**Actinomycosis** attacks muscles only by invasion from the primary lesion. The masseter, pterygoid muscles and those of the tongue, chest and abdominal walls are chiefly affected.

**Toxic Myositis** usually called "muscular rheumatism" is a common complaint among men and women after thirty years of age, and is due to a toxin derived from a focus of infection in the teeth, tonsils, air passages or intestinal canal. It also affects the muscle sheaths, fasciæ, ligaments and nerves and is probably a neurofibrositis rather than a myositis. The best-known example is "**Lumbago**," in which the lumbar aponeurosis and the erector spinae muscles are involved. In severe cases the pain is of sudden onset and movement is a painful and laborious process. Exposure to cold draughts gives rise to a "stiff neck," which is another common example of this condition.

*Treatment* consists of hot applications followed by radiant heat, short-wave diathermy and massage. Every effort should be made to discover and eradicate any focal sepsis that exists.

**Parasitic Myositis** is a painful oedematous swelling of muscles, particularly those of the upper arm, due to the presence of the embryo of the *Trichina spiralis* worm.

**Myositis Ossificans** is a rare disease with a familial tendency affecting young males. The pathology is obscure, but it is possibly a primary fibrositis leading to atrophy and replacement of the muscle fibres. The condition starts in the muscles of the back, and flat plaques or rods of bone are laid down irregularly and without attachment to the bones of the part. It is a slowly progressive disease and spreads to other groups of muscles. The movements of the body become increasingly difficult and death from asphyxia follows the immobilisation of the respiratory muscles. Pain is not a prominent symptom. No treatment is of any avail.

**Myositis Ossificans Traumatica** is the result of an injury in the region of joints. A fracture or a tear in the periosteum may be in close proximity to the origin or insertion of a muscle. Under certain conditions the bone-forming process spreads up the tendon or aponeurosis into the muscle belly, and a bony mass of considerable size may form (Fig. 615). The process may be initiated or aggravated by too early attempts at movement. It is met with most frequently in connection with fractures near the elbow joint in children, the brachialis anticus being chiefly affected.



FIG. 615

X-ray showing myositis ossificans in the brachialis anticus muscle.

The *treatment* entails complete immobilisation in a plaster-of-Paris case for many weeks until the greater part of the newly formed bone is reabsorbed. If any serious interference with movement remains after one year the bony mass should be removed.

A more chronic type is due to repeated slight trauma and is exemplified by the ossification of the tendon of the adductor longus muscle above the knee—the so-called “Rider’s bone.” No treatment is needed unless symptoms are present, which is unusual.

**Myositis Fibrosa**, also known as ischæmic paralysis or **Volkman’s contracture**, is the result of too tight bandaging, of pressure from ill-applied splints or of too prolonged use of a tourniquet; it may be due to injury to the brachial artery directly at the time of injury. It is usually associated with fractures near the elbow and affects the muscles in the forearm. As the result of pressure the brachial artery is compressed and the muscles are starved of blood, undergoing a process of autodigestion termed necrobiosis. The muscle fibres are replaced by fibrous tissue, the contraction of which leads to the deformity of the hand. The severity of the damage to the muscles varies considerably in different patients. The symptoms usually pass unnoticed until the splint is removed, the complaints of pain and a feeling of tightness in the limb being ascribed to the fracture. When the splint is removed, the patient notices that he is unable to use his fingers and it will be seen that they are flexed at the phalangeal and metacarpo-phalangeal

joints. If the wrist is fully palmar-flexed the fingers can be straightened, but when the wrist is moved into full dorsiflexion the fingers become progressively more flexed. There is limitation of supination of the forearm owing to contracture of the pronator radii teres. The differential diagnosis rests between Volkmann's and Dupuytren's contractures, lesions of the ulnar, median and musculospiral nerves, and deformities of the hand due to sepsis. The extension of the fingers when the wrist is flexed, the absence of anaesthesia and of the reaction of degeneration should serve to settle the diagnosis.

*Treatment* is essentially prophylactic. Careful attention to detail in the management of fractures should eliminate this condition and, if during the first few days the patient complains of pain and tightness of the bandages, the splints must be thoroughly investigated and removed entirely if any doubt exists. The radial pulse beat at the wrist is an excellent indicator of arterial obstruction, and if it is of much smaller volume on the injured side the splints should be removed. Established cases are treated by graduated movement on an adjustable splint, which has a hinge at the level of the wrist and a ratchet for varying the angle. The forearm, hand and fingers are fixed in the splint in a position of full flexion of the wrist, thus allowing the fingers to straighten. The angle of flexion at the wrist is decreased daily by the screw, the fingers being firmly fixed in extension. In this way the muscles are slowly stretched and finally the fingers can be kept extended even when the wrist is dorsiflexed. The more advanced cases will not respond to this method and Max Page's operation should be performed. This consists of stripping the common origin of the flexors and allowing them to gain fresh attachments lower in the forearm. The results of this condition are deplorable and it must be emphasised that it is preventable.



FIG. 616

A large sarcoma arising in the muscles of the right thigh in a young woman.

### TUMOURS OF MUSCLES

**Benign Tumours.** The only benign tumour which arises from muscle is the rare rhabdomyoma, which has been recorded in the tongue. Leiomyoma is common in the uterus but always contains fibrous tissue. Other benign tumours in muscle are lipoma, angioma, fibroma and fibromyoma.

**Malignant Tumours.** A pure muscle sarcoma does not exist, though it may be a constituent of a teratoma, but a spindle-celled fibrosarcoma arises in the connective tissue of muscle. It shows a marked tendency to spread throughout the muscle and to refrain from infiltrating its sheath and invading neighbouring structures. The muscle becomes enlarged and hard, and stands out prominently from its neighbours (Fig. 616). This type of growth is met with not infrequently in

the hamstring muscles. The rate of growth and the consistency of the tumour vary, but they are relatively slow-growing. The swelling cannot be moved in the long axis of the muscle fibres but can be from side to side. There is usually little doubt as to the diagnosis and a sarcoma is distinguished from diffuse gummatous infiltration by remaining localised to one muscle instead of affecting several members of the same group.

*Treatment* in the early stages consists of complete excision of the whole muscle, but in the later stages amputation will be necessary, and it must be so planned as to remove entirely all the affected muscles.

The electrical reactions of muscle and the methods in use for eliciting them are described on p. 890.

## DISEASES OF FASCIA

### DUPUYTREN'S CONTRACTURE OF THE PALMAR FASCIA

The middle division of the palmar fascia extends from the distal margin of the anterior annular ligament and, spreading out in a fan-shaped manner, affords a protective covering to the flexor tendons, nerves and vessels in the palm. At the base of each finger a digital prolongation is formed, which fuses with the sheath of the flexor tendons and sends a process on each side of the finger to blend with the deep fascia in its lateral aspects.

*Etiology.*—Dupuytren's contracture affects this middle division of the palmar fascia, and occurs in men in later life as a result of long-continued pressure in the palm. It is traditionally reputed to be due to gout, but the association lies in the use of a stick as an aid to walking when the big toe is affected by this complaint. It is not uncommon in the hands of skilled craftsmen, carpenters and mechanics, *e.g.*, the line repairer in the telephone and telegraph service is apt to suffer from contracture in his left palm, by which he supports himself while working mainly with his right.

*Pathology.*—A thickened indurated nodule first makes its appearance in the palm at the level of the distal flexion crease at the base of the ring finger. The skin is also thickened and firmly attached to the fibrous nodule beneath. Very slowly the induration spreads distally in the fascia towards the ring and little fingers and, still more gradually, proximally up the palm. In many patients the process does not extend across the palm, but in others the middle, and rarely the index, fingers may be affected.

*The Clinical Picture* is unmistakable. The contraction of the fibrous processes leads to flexion of the metacarpo-phalangeal and proximal interphalangeal joints, so that slowly the ring and little fingers are flexed into the palm. The terminal phalanx usually remains extended. After many years the flexion may become so advanced that the fingers can hardly be separated from contact with the palm.

If treatment is not adopted in the early stages, the skin also becomes contracted and hypertrophied across the front of the flexed joints.

*Treatment.*—Although a number of operations have been advised, nothing short of radical removal of the fascia should be contemplated. So closely adherent is the skin to the underlying fascia, that its nutrition may be badly damaged during operation, and in many patients a plastic pedicle skin graft will be needed to re-form the covering of the palm. The dissection must be carried into the fingers, as it is essential that the digital prolongation should be completely removed as well as the parent fascia in the palm.

It is important that treatment should be undertaken before the contracture has been allowed to advance too far.

Other diseases of fascia are dealt with elsewhere; toxic or rheumatic conditions under toxic myositis (p. 1165); and gonococcal fibrositis and fasciitis in the chapter on Venereal Disease (Chap. V.).

## THE TENDON SHEATHS

### TENOSYNOVITIS

**Acute Non-suppurative Tenosynovitis** commonly occurs in the sheaths of the extensor tendons of the thumb and of the peroneal tendons at the ankle. It is usually the result of over-use following a period of inactivity, and is therefore met with in men resuming heavy manual work after enforced idleness, in seamstresses and in both sexes at the beginning of a season of golf or tennis. The lining membrane becomes swollen and the sheath is filled with a serous effusion. The patient complains of pain during certain movements and a narrow elongated swelling is present in the position of the sheath, which is slightly tender. Movement produces a characteristic fine creaking, which disappears if the sheath becomes very distended and reappears as the effusion is absorbed.

*Treatment* consists of firm strapping over the lower part of the forearm and wrist and resting the arm in a sling for one week, after which massage and diathermy quickly complete the cure.

**Chronic Tenosynovitis** follows repeated attacks of the acute condition. The part should be immobilised for fourteen days and then counter-irritation, passive congestion, ionisation and massage should be tried. The condition is apt to be resistant to treatment.

**Acute Suppurative Tenosynovitis** may affect any tendon sheath, but is most frequently seen in the hand as a complication of septic fingers. Its results are so crippling, if imperfectly treated, that the condition is described in full in the chapter devoted to infections of the hand (p. 250).

### CHRONIC TUBERCULOUS TENOSYNOVITIS

The sheath may be the primary seat of the condition or it may be involved from a neighbouring joint. It affects either sex, occurs

at any age after puberty and is usually seen in the sheaths around the wrist and ankle. Two distinct varieties occur, the fluid and the dry.

**The fluid type** is the commoner and is characterised by a fluid effusion into the sheath, the endothelial lining of which becomes moderately thickened. Fibrin is deposited on its surface and flakes of it are detached by movement of the tendon. These are moulded into small flat elliptical bodies named "**melon-seed bodies**." At operation the sheath is tightly packed with them and in the fresh state they are pearly white, semi-translucent flakes. This type of

affection remains confined to the sheath and does not spread into the surrounding skin, bones or joints. Clinically there is a narrow elongated swelling in the whole length of the sheath, in which fluctuation can be obtained. The melon-seed bodies give a granular feeling and a fine creaking on movement.

This condition affects the sheaths of the flexor tendons at the wrist and spreads throughout the ulnar and radial bursæ and into the sheaths of the finger and thumb. It is known as the "**compound palmar ganglion**," in which the swelling is bi-lobed, the anterior annular ligament dividing it into a pouch above the wrist and another in the palm, between which cross fluctuation is obtained (Fig. 617).

*Treatment* may be conservative or radical. The conservative method entails immobilisation of the limb in a

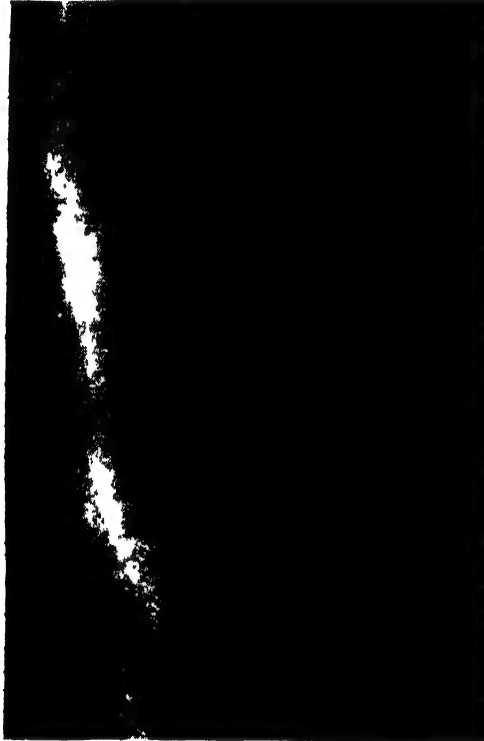


FIG. 617

A compound palmar ganglion.

plaster-of-Paris bandage for three months. If marked improvement is shown at the end of this time, general treatment can be continued with confidence in the eventual result; but if little or no improvement is achieved, then operation should be performed. Radical operative treatment is advocated by many surgeons at the outset, and Kanavel is of the opinion that open operation in compound palmar ganglion yields much superior results. The sheath is opened in its whole length, the melon-seed bodies removed, the tendons freed from their fibrinous covering and the thickened lining of the sheath dissected away. The wound is closed without drainage and placed in a plaster splint with the wrist in the dorsiflexed position.

**In the dry type** the synovial membrane is thickened, œdematous and infiltrated with tuberculous granulation tissue which spreads

on to the tendons and involves the adjacent bones, joints and subcutaneous tissues. Caseous foci develop and coalesce to form abscesses, and, if the skin becomes involved the pus is evacuated, a chronic sinus remaining. In the early stage there is a doughy swelling of the sheath, which later becomes more diffuse and fluctuant. Movements are painful and limited.

*Treatment* consists in prolonged immobilisation, but the results are less satisfactory than in the fluid type.

**Syphilitic Tenosynovitis** is rare. In the secondary stage there may be a transient serous effusion and in the tertiary stage a painless gummatous thickening. The history and the Wassermann reaction lead to a correct diagnosis and the condition yields readily to antisyphilitic treatment.

**Gouty Tenosynovitis** is occasionally seen in the fingers and hand and leads to the deposition of large masses of sodium biurate (tophi).

### TUMOURS OF TENDON SHEATHS

**A Ganglion** is an encapsuled cystic swelling arising from the synovial membranes of joints and tendon sheaths. Its true pathology is not understood. In the past it was described as a hernial protrusion of the synovial membrane through the fibrous sheath or capsule. This is certainly incorrect and it is probably a myxomatous degeneration of the subsynovial tissues or a pure myxoma. It consists of a fibrous cyst filled with a glairy, jelly-like fluid. The commonest situations are the dorsum of the hand and wrist (Fig. 618), the peroneal region of the ankle, the anterior surface of the wrist and the bases of the fingers on their flexor aspect. Recurrence after removal is admittedly frequent, because it is not recognised that the majority of ganglia arise from the subsynovial tissue of joints and not from the tendon sheaths; this is especially true of those on the dorsum of the wrist. The chief symptom is the appearance of the swelling, but for several days before this is apparent some patients complain of dull aching pain and the wrist feels weak. When a ganglion is small it is so tense that it may be mistaken for a solid, even bony tumour, but later it is fluctuant and translucent. It is fixed to the deeper structures but the skin moves freely over it.

*Treatment.*—It has been observed that accidental rupture has sometimes led to a lasting cure. The ganglion may be ruptured deliberately by pressure, a blow or by slitting its capsule with a fine tenotome, the fluid being then expressed into the subcutaneous tissues and the part firmly bandaged. A certain number appear to be cured by this method. Aspiration and injection with sodium morrhuate have been given a trial, but yield unsatisfactory results and are not devoid of risk. The method of choice is excision and the ganglion must be removed completely or recurrence will follow. The



FIG. 618

A simple ganglion on the back of the hand.

dissection should be carried down to the joint capsule and not stopped short at the tendon sheaths.

The compound palmar ganglion has been described above.

**New Growths** are rare. Subsynovial lipoma, hæmangioma, fibroma, endothelioma and sarcoma are described. The so-called myeloma of tendon sheaths (Fig. 619) is not infrequently met with in connection with the fingers. It is a soft spherical tumour with a definite capsule, and in naked-eye appearance and histological structure it corresponds closely to the xanthoma, being of golden-yellow



FIG. 619

A myelo-xanthoma of the foot of such long standing that calcification is present.

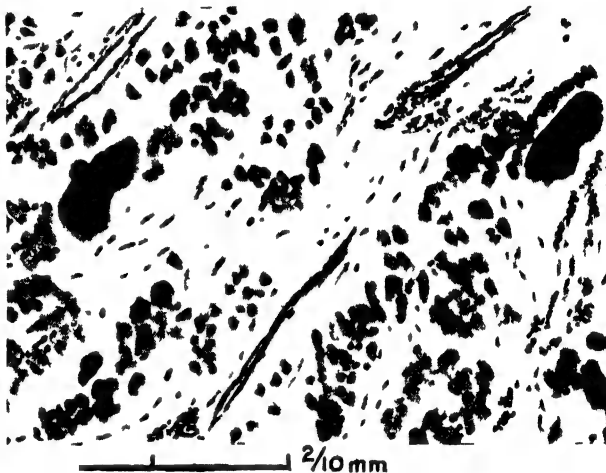


FIG. 620

Section of a myelo-xanthoma of a tendon sheath. (Kettle)

colour and containing lipoid material (Fig. 620). It is benign and local removal suffices.

## DISEASES OF BURSÆ

Bursæ are of three varieties. True bursæ are interposed between two moving surfaces to reduce friction or are placed over prominent bony points to act as protective cushions (*e.g.*, the ischial and olecranon bursæ). Bursal extensions of joints fulfil a similar function but are continuous with the synovial membrane of the joint. Adventitious bursæ are developed in situations in which bony prominences are subjected to pressure (*e.g.*, the bursa over the head of the metatarsal in hallux valgus, that over an exostosis, or those in connection with the carrying of weights on the shoulder). In structure all bursæ consist of a fibrous capsule lined with endothelium analogous to the synovial membrane of joints.

## INJURIES TO BURSÆ

**Wounds.**—Bursæ are opened by incised or punctured wounds or by falls in which the overlying skin is split. The injury is recognised



by the escape of synovial fluid, and if this persists—as it sometimes does—the bursa must be removed.

**Acute Traumatic Bursitis** follows a contusion. The bursa becomes distended by a blood-stained serous effusion and is prominently enlarged and tender. The overlying skin may be bruised, but the signs of inflammation are absent. The parts should be kept at rest by firm strapping, the fluid having been first aspirated if the collection is of any size.

**Chronic Traumatic or Serous Bursitis** is the commonest disease of bursæ and is the result of repeated slight trauma. A chronic serous effusion occurs, as a result of which the walls become thickened by the deposition of layers of fibrin and by fibrosis. At first there is a considerable amount of fluid present, but later the progressive thickening of the walls leads to a reduction in the size of the cavity to a mere cleft. The condition is then termed a chronic fibroid bursitis. Excision of the bursa is the only satisfactory treatment.

**Hæmorrhagic Bursitis** is an occasional result of injury, the bursa being distended with blood. It may be met with in certain blood diseases such as hæmophilia and leukæmia. Treatment consists in firm strapping and rest.



FIG. 621

An acute suppurative bursitis of the prepatellar bursa, which has been allowed to progress without treatment.

### INFLAMMATORY DISEASES OF BURSÆ

**Acute Suppurative Bursitis** follows incised or punctured wounds and is staphylococcal in origin. In some cases the infection is carried to the bursa by the lymphatics from a septic focus in the vicinity, when it may be either staphylococcal or streptococcal. Although the infection is primarily limited to the bursa, pus readily spreads through the capsule and an acute cellulitis results (Fig. 621). In neglected cases the underlying bone or joint may be involved. Treatment consists in free incision and drainage, and if later a sinus persists the bursa must be excised.

**TUBERCULOUS BURSTITIS** is similar to tuberculous tenosynovitis in being of two types, the fluid and the dry. The infection may be primary in the bursa or it may spread from a joint with which the bursa communicates. The fluid type is characterised by an effusion and melon-seed bodies, while the dry tends to caseate and form an abscess. Treatment depends on the anatomy of the affected bursa; those which are unconnected with a joint should be excised, whereas the others share in the appropriate treatment for the tuberculous joint.

**Syphilitic Bursitis** is sometimes seen in the secondary stage in the form of a transient effusion. In the tertiary stage it usually affects

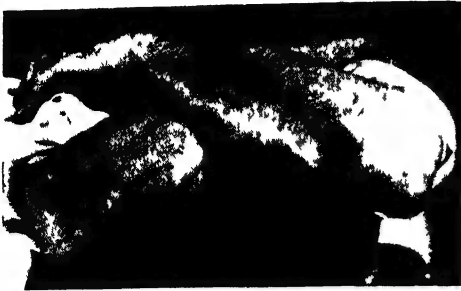


FIG. 622

Bilateral olecranon bursitis.

the ischial and prepatellar bursæ, is frequently bilateral and occurs as a diffuse gummatous infiltration. Ulceration through the overlying skin is liable to follow. Chronic bilateral bursitis should always suggest the possibility of syphilis and the history, the Wassermann reaction and the rapid response to specific treatment confirm the diagnosis. Treatment follows the usual antisyphilitic routine.

**Gonococcal Bursitis** is one of the complications of gonorrhœa (see p. 60).

**Gouty Bursitis** is similar to gouty tenosynovitis, sodium biurate being deposited in the wall of the bursa. Large swellings result and the skin may give way, leading to a gouty ulcer. The olecranon bursa is commonly affected. The mass should be excised.

### INDIVIDUAL BURSAE

#### Group I.—The True Bursæ.

1. The subdeltoid bursa lies between the deltoid muscle and the underlying great tuberosity of the humerus. It may be the seat of an acute gonococcal infection, particularly in women, and of chronic tuberculous disease. It forms a fluctuant swelling beneath the muscle and active abduction is painful and limited.

2. The olecranon bursa may be acutely infected by wounds or by lymphatic infection from sepsis in the forearm or hand. Chronic serous bursitis (Fig. 622) is common in certain types of people and is termed the "miner's or student's elbow." It is also the seat of gouty deposits.

3. The ischial bursa is met with in people whose work entails prolonged sitting and has long been known as the "weaver's bottom." It may be affected in tertiary syphilis.

4. The gluteal bursa lies between the tendon of the gluteus maximus and the great trochanter of the femur and is occasionally affected by tuberculosis. It appears as a swelling behind the trochanter and causes abduction and eversion at the hip joint.

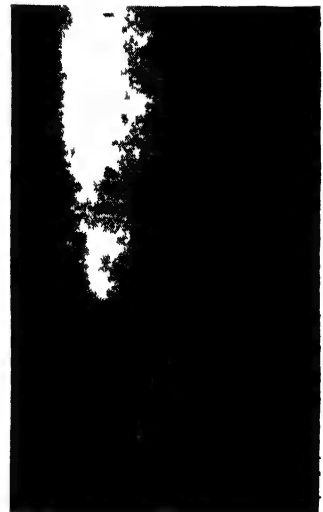


FIG. 623

A typical example of "housemaid's knee."

5. The prepatellar bursa is the most commonly affected by all types of disease, particularly as the chronic serous bursitis or "housemaid's knee" (Fig. 623).

6. The bursa between the ligamentum patellæ and the anterior surface of the upper end of the tibia is occasionally the seat of a chronic serous effusion and more rarely of an acute infection. It gives rise to a fluctuant swelling on either side of the ligament and to discomfort when the knee is straightened. If it becomes acutely infected it must be drained immediately, lest the knee joint be secondarily infected.

7. The tendo achillis bursa is chronically enlarged as the result of pressure from ill-fitting shoes. It presents as a fluctuant swelling on either side of the tendon.

### Group II.—Bursæ Communicating with Joints.

8. The semi-membranosus bursa lies between the semi-membranosus tendon and the inner head of the gastrocnemius muscle, and has a narrow opening into the postero-internal aspect of the knee joint. Its enlargement therefore may be secondary to disease of that joint and this must be carefully considered and excluded before the removal of the bursa is advised. Chronic serous bursitis is frequent among children

of both sexes and may be bilateral. The swelling is more prominent and more tense in full extension of the joint. Treatment consists in dissection of the sac and ligation of its neck at its entrance to the joint.

9. The popliteus bursa communicates freely with the knee joint and is rarely enlarged except as a result of disease of the knee.

10. The psoas bursa is frequently in communication with the hip joint, but in some people is a separate sac. It lies between the psoas tendon and the hip joint. When it opens into the joint it participates in its diseases; in other cases it is occasionally the seat of a chronic serous bursitis, which is often bilateral. It forms a swelling in Scarpa's triangle, and is tense in extension and flaccid in flexion of the hip.

### Group III.—Adventitious Bursæ.

These are of new formation over bony prominences subjected to constant pressure. for example :—

- (a) The "deal runner's shoulder" (Fig. 624).
- (b) The "Covent Garden hummy," over the 7th cervical vertebra.
- (c) The "basket carrier's bursa," in the scalp.
- (d) The "tailor's ankle," a bursa over the external malleolus.
- (e) Those over exostoses, hallux valgus and deformed feet.

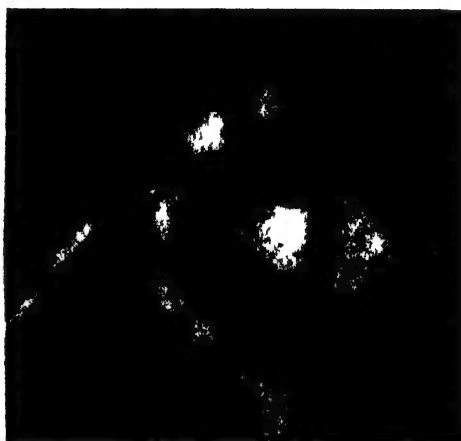


FIG. 624

A large adventitious bursa over the left shoulder.



# INDEX

## A

**A.B.A., 976**

**Abbott's jacket for scoliosis, 1121**

**Abdominal injuries, 528**

- non-penetrating, 529
  - of abdominal wall, 529
  - of intestine, 529
  - of liver, 531
  - of pancreas, 532
- penetrating, 532
  - of large intestine, 533
  - of liver, 534
  - of small intestine, 533
  - of spleen, 534
  - of stomach, 534

**Abdominal movements, mechanism of, 560**

**Abdominal rigidity, 548, 673**

**Abdominal wall,**  
 dilated veins in, 538  
 growths of, 537  
 hematoma of, 537  
 inflammation of, 536  
 injuries to, 529

**Abducent nerve, 907**

**Aberrant renal artery, 719, 723**

**Abrasion, 119**

**Abscess, acute, 2, 22**

- amœbic of liver, 689
- appendicular, 675, 682
- Bezold's, 380
- clinical picture of, 22
- cross-infection of, 23
- extradural, 850
- intrapertitoneal, 553
- ischio-rectal, 653
- of alveolus of jaws, 312
- of axilla, 23
- of Bartholin's gland, 63, 811
- of brain, 394, 476, 487, 850
- of breast, 509
- of Cowper's gland, 58
- of middle palmar space, 253
- of nasal septum, 405
- of palm (collar stud), 252
- of prostate, 58
- of scalp, 827
- of spleen, 712
- of thenar space, 253
- pelvic, 555
- pelvirectal, 652
- perinephric, 730
- peritonsillar, 429
- periurethral, 58, 788
- retropharyngeal, 429

**Abscess, acute—continued**

- solitary, 689
- subphrenic, 553
- treatment of, 23
- tropical, 689

**Abscess, chronic, 24**

- amyloid disease in, 25
- in breast (non-tuberculous), 513
- in tuberculous arthritis, 1074
- ankle joint, 1084
- elbow joint, 1086
- hip joint, 1081
- sacro-iliac joint, 1087
- shoulder joint, 1085
- spine, 1089

**Abscess, formation of, 2, 24**

pathology of, 2, 24

**Absence of anal canal, 647**

- of radius, congenital, 1115
- of rectum, 647

**Acanthosis nigricans, 539**

**A.C.C., 890**

**Accessory nasal sinuses. See Nasal Sinuses**

**Accessory spleen, 711**

**Acetabulum, fracture of, 974**

**Achalasia of bladder, 759**

of œsophagus, 439

**Acholic jaundice, 713**

**Achondroplasia, 1037**

"trident-hand" in, 1037

**Acid-fast bacilli, 38**

**Aclasia, diaphyseal, 1048**

**Acoustic neuroma, 859**

**Acquired hernia, 564**

**Acrocyanosis, 911**

**Acromegaly, 1038**

**Actinomyces, 48**

- of appendix, 684
- of breast, 514
- of jaw, 313
- of kidney, 732
- of liver, 689

**Actual cautery, 217**

**Acusection, 218**

**Acute ulcer, 165**

**Adamantinoma, 94, 323**

**Adductor muscles of thigh,**  
 rupture of, 1163

**Adenoids, 421**

**Adenoma, 104**

- of bronchus, 490
- of kidney, 745
- of large intestine, 626
- of liver, 691
- of pancreas, 710

**Adenoma—continued**

- of pituitary, 857
- of rectum, 661
- of salivary glands, 350
- of sebaceous glands, 242, 827
- of small intestine, 626
- of sweat glands, 242, 828
- of thyroid, simple, 375
- toxic, 376
- of umbilicus, mucous, 540

**Adenomyoma of uterus, 821**

**Adiposis dolorosa, 91**

**Aditus ad antrum, 379**

**Adolescent kyphosis, 1123**

**Adrenalin, 211**

**Adult kyphosis, 1124**

**Adventitious bursa, 1175**

- on ankle, 1175
- on scalp, 1175
- on shoulder, 1175
- over cervical vertebra, 1175
- over exostosis, 1175

**Aerocœles, 362**

**Agordian colostomy belt, 665**

**Air embolism, 154, 281**

**Air hunger, 144**

**Airway, Hewitt's, 196**

**Albee's operation, 1096**

**Albuminuria, 716**

**Alcohol,**

- industrial, 181
- surgical spirit, 181

**Allergic coryza, 407**

**Allergy, 20**

nasal manifestations of, 407

**Alopecia, 68**

**Alveolar abscess, 312**

**Amasia, 505**

**Amœbic abscess of liver, 689**  
 infections, 53

**Amnioplastin, 1164**

**Amputation, neuroma, 98**

**Amyloid disease, 10, 25**

in tuberculous arthritis, 1075

**Anæsthesia, 188**

administration of, 193

apnoea in, 196

care of patient after, 208

choice of anæsthetic, 198

contraindications, 188

dolorosa, 877

examination before, 188

pre-operative medication, 190

signs of, 194

stages of, 194

vomiting after, 209

- Anal canal, 645**  
 anatomy of, 645  
 carcinoma of, 665  
 fistula-in-ano, 655  
 imperforate anus, 647  
 perianal abscess, 653  
 pruritus ani, 660
- Analgesia, 210**  
 extrathecal, 211  
 infiltration, 211  
 intrathecal, 211  
 regional methods, 211  
 surface application in, 210
- Anaphylactic shock, 20**
- Anaphylaxis, 19**
- Anastomotic ulcer.** See Gastro-jejunal Ulcer
- Anel's ligature for aneurysm, 270**
- Aneurysm, 71, 268**  
 Anel's ligature for, 270  
 Brasdor's ligature for, 270  
 Hunter's ligature for, 270  
 of aorta, 272, 274  
 of axillary artery, 274  
 of common carotid artery, 273  
 of external carotid artery, 273  
 of femoral artery, 274  
 of gluteal artery, 275  
 of iliac arteries, 274  
 of innominate artery, 272  
 of intracranial arteries, 273, 275  
 of popliteal artery, 275  
 of sciatic artery, 275  
 of subclavian artery, 274  
 of superficial femoral artery, 275  
 varieties of, 266  
 Wardrop's ligature for, 270
- Aneurysm by anastomosis, 268**
- Aneurysmal varix, 271**
- Angina pectoris, 913**
- Angioma,**  
 capillary, 111  
 cavernous, 111  
 of bones of skull, 837  
 of brain, 863  
 of larynx, 455  
 of liver, 691  
 of renal pelvis, 749
- Angular vein, ligature of, 852**
- Ankle joint,**  
 deformities in infantile paralysis, 1150  
 dislocation, 1004  
 effusion into, 1004  
 fractures involving, 1000  
 sprains, 1004  
 tuberculosis of, 1084  
 in adults, 1085
- Ankyloglossia, 336**
- Ankylosis, 1062**  
 position of election for, 213, 1063
- Anorchism, 793**
- Anterior crural nerve, 903**
- Anterior poliomyelitis, 1147**
- Anterior tibial nerve, 905**
- Anthrax, 42**
- Antibodies, 18**
- Antimony tartrate, 51, 52**
- Antiphlogistine, 216**
- Antiseptic surgery, 180**
- Antitoxins, 17**
- Antivenin, 17, 121**
- Antrum, maxillary.** See Maxillary Antrum
- Anuria, 716**  
 calculus, 752
- Aphonia, functional, 453**
- Aphthous stomatitis, 329**
- Aplasia cranii, 835**
- Apnoea, 196**
- Apoplexy, 143**
- Appendicitis, 668**  
 effects on peritoneum, 670  
 etiology, 668  
 pathology, 669
- Appendicitis, acute, 672**  
 clinical varieties of, 677  
 complications of, 682  
 diagnosis of, 678  
 disappearance of pain in, 675  
 expectant treatment *v.* operation, 680  
 general peritonitis in, 679  
 ileus duplex in, 682  
 localised abscess in, 675, 682  
 pelvic, 677  
 retro-cæcal, 677  
 signs of, 673  
 spreading gangrene in, 682  
 symptoms of, 672  
 treatment of, 679
- Appendicitis, chronic, 672, 683**
- Appendix,**  
 actinomycosis of, 684  
 anatomy, 666  
 carcinoid tumour of, 684  
 foreign bodies in, 667  
 growths of, 684  
 inflammation, acute, 672  
     chronic, 682  
 mucocele of, 683  
 pseudo-myxoma peritonei in, 683  
 stercoliths, 667
- Aqueduct of Fallopius, 379**
- Arachnoid cyst, 119**
- Aran's theory, 831**
- Arch supports in flat foot, 1141**
- Argentiform tumour, 684**
- Argyll-Robertson pupil, 1111**
- Arrest of hæmorrhage, 144, 152**
- Arsenic poisoning, 79**  
 albuminuria in, 79  
 dermatitis in, 79  
 jaundice in, 79
- Artefact, 166**
- Arterial hæmorrhage, 153**
- Arteries,**  
 affections of, 263  
 contusions of, 259  
 injury to, 259  
 penetrating wounds of, 261  
 rupture of, 260
- Arteriography, 229, 855**
- Arteriosclerosis, 263**
- Arteriovenous aneurysm, 271**  
 wounds, 262
- Arteritis, acute,**  
 embolic, 263  
 infective, 263  
 traumatic, 263
- Arteritis, chronic, 263**  
 toxic, 265
- Artery forceps, 152**
- Arthritis, acute, 1065**  
 dysenteric, 1069  
 gonococcal, 1070  
 of hip, 1068  
 of knee, 1067  
 pneumococcal, 1069  
 pyæmic, 1068  
 typhoid, 1069
- Arthritis deformans, 1069**
- Arthritis, rheumatoid, 1060**
- Arthrodesis, 1065**  
 for infantile paralysis of ankle, 1150  
 for osteo-arthritis of ankle, 1107  
 of hip, 1106  
 of knee, 1107  
 for tuberculous arthritis, 1075  
 of hip, 1080  
 of knee, 1083
- Arthrotomy, 1063**  
 for diagnosis, 1082
- Artificial pneumothorax.** See Pneumothorax, Artificial
- Artificial respiration, 197**
- Aschheim-Zondek test, 802, 823**
- Ascites, cirrhotic, 693**
- Asepsis, 181**
- Asthma, 408**
- Astragalus, fractures of, 1003**
- Astrocytoma, 98, 863**
- Athelia, 505**
- Atheroma, 263**
- Atlas, fracture of, 1011**
- Atrophic scirrhus, 519, 524**
- Atrophy from disuse, 11**
- Atropine, 190**
- Auditory nerve, 909**  
 Ménière's disease, 869  
 tumour, 859
- Aural speculum, 380**  
 Siegel's, 380
- Aural syringe, 384**
- Auricular fibrillation, 372**
- Automatic bladder, 759, 877**
- Autonomic nervous system, 910**  
 anatomy, 910  
 parasympathetic, 911  
 sympathetic, 910  
 indications for surgery, 911  
 cardiovascular, 912  
 hyperidrosis, 914  
 intractable dysmenorrhœa, 914  
 pain, 914  
 visceral, 913

**Autonomic Nervous System—**  
*continued*  
 sympathectomy, cervical, 912  
 lumbar, 912  
 pre-sacral, 914  
**Avertin, 191**  
**Avulsion of finger, 1164**  
 of scalp, 826  
**Axenhausen's operation, 332**  
**Axillary abscess in Pott's**  
**disease, 1092**  
**Axis, fracture of, 1011**

## B

**Bacilluria, 717**  
**Bacillus coli communis, 45**  
 mallei, 43  
 of malignant cedema, 35  
 pestis, 44  
 proteus, 46, 761  
 pyocyaneus, 46  
 sporogenes, 35  
 typhosus, 46  
 welchii, 35  
**Bacteria,**  
 definition of, 15  
 distribution of, 15  
 pathogenicity of, 16  
 types of, 15  
 virulence, 16  
**Baker's cyst, 1103**  
**Balanoposthitis, 782**  
**Balkan frame, 923, 985**  
**Bands, strangulation by, 635**  
**Banti's disease, 712**  
**Barbiturate drugs, 192**  
**Barium enema in radiography,**  
**226, 629**  
 meal in radiography, 224, 586  
**Bartholin's abscess, 811**  
 cyst, 811  
**Basal-celled carcinoma, 106, 239**  
**Basal metabolic rate, 364**  
**"Basket carriers' bursa" of**  
**scalp, 1175**  
**Basophil adenoma, 857**  
**Bath, spa treatment at, 1101**  
**Baths, as a form of treatment,**  
**216**  
**Bazin's disease, 234**  
**Bed-sores, 243, 877, 881**  
**Belfield's operation, 58**  
**Bell, nerve of, 898**  
**Bell's palsy, 908**  
**Bence-Jones's albumose, 97,**  
**1056**  
**Benign giant-celled tumour of**  
**bone, 95, 1049**  
**Bennett's fracture, 968**  
**Bergner waves, 849, 855**  
**Bezold's abscess, 380**  
**Biceps, muscle and tendon,**  
 rupture of, 1162  
**Jer's bandage, 256**  
 cupping glass, 9, 232  
**Excursion osteotomy of**  
**Lorenz, 1130**  
**Bigelow's evacuator, 766, 790**  
**Bilharzia, 52**  
 of bladder, 52  
 of kidney, 732  
 of rectum, 661  
**Bilirubin calcium, 700**  
**Biniodide of mercury, 180**  
**Bismuth metal in syphilis, 77**  
**Black eye, "anterior fossa," 832**  
 tongue, 339  
**B.L.B. mask, 185, 161, 463**  
**Bladder, urinary,**  
 absence of, 755  
 anatomy of, 754  
 anomalies of, 754  
 automatic, 759, 877  
 calculi, 765  
 carcinoma, 769  
 cystitis, 761  
 acute, 762  
 chronic, 763  
 diverticulum, 756  
 ectopia vesicæ, 755  
 examination of, 754  
 extraperitoneal rupture, 760  
 fistula, 764  
 vesico-intestinal, 765  
 vesico-vaginal, 765  
 foreign bodies in, 767  
 growths, 767  
 incontinence of urine, 757  
 injuries, 760  
 complicating fractured  
 pelvis, 760, 974  
 intraperitoneal rupture, 760  
 nocturnal enuresis, 757  
 neurogenic, 758  
 papilloma, 767  
 patent urachus, 754  
 retention of urine, 758  
 sarcoma, 770  
 spasm, 757  
 tuberculosis, 764  
**Blast injury**  
 to lungs, 123, 464  
 to soft tissues, 123  
**Blastomycosis, 50**  
**"Blind caecostomy," 630**  
**Blood clot,**  
 organisation of, 7  
 red, 144, 277  
 white, 144, 277  
**Blood groups, 148**  
 rheus factor, 149  
 sub-groups, 149  
 tests for, 148  
**Blood transfusion, 146**  
 blood plasma in, 151  
 citrated blood for, 150  
 continuous-drip method, 151  
 defibrinated blood, 150  
 for duodenal hæmorrhage,  
 610  
 for gastric hæmorrhage, 605  
 grouping, 148  
 tests, 148  
 indications for, 151  
 methods of, 149  
 stored blood in, 151  
**Blood urea, estimation of, 718**  
**Blood vessels,**  
 See Arteries  
 See Veins  
 surgery of, 259  
 tumours of, 286  
**Blue-domed cysts, 510**  
**Bohler's traction frame, 999**  
 walking iron, 998, 1002  
**Boils, 231**  
**Bollinger, Spät-apoplexie of,**  
**842, 848**  
**Bone, diseases of, 1014**  
 achondroplasia, 1037  
 acromegaly, 1038  
 acute epiphysitis, 1022  
 osteomyelitis, 1018  
 traumatic osteomyelitis,  
 1022  
 anatomy, 915, 1014  
 cancellous, structure of, 1014  
 chronic osteomyelitis, 1022  
 circulus vasculosus articuli,  
 1015  
 cleido-cranial dysostosis, 1046  
 compact, structure of, 1014  
 contusions, 916  
 cyst, 1040, 1058  
 development of, 1015  
 diaphysis of, 1014  
 epiphyseal cartilage, 1014  
 epiphysis of, 1014  
 fractures of, 917  
 classification of, 917  
 fragilitas ossium, 1041  
 Haversian canals, 1014  
 hypertrophic pulmonary  
 osteoarthropathy, 1045  
 inflammation of, 1015  
 caries, 1018  
 necrotic, 1018  
 sicca, 1018  
 suppurativa, 1018  
 cloaca, 1017  
 involucrum, 1017  
 necrosis, 1016  
 pathology, 1016  
 rarefaction, 1018  
 sclerosis, 1018  
 sequestrum formation,  
 1016  
 metaphysis, 1015  
 nutrient artery of, 1015  
 osteitis deformans, 1038  
 fibrosa, 1040  
 osteochondritis, 1042  
 Keimboch's, 1045  
 Kohler's, 1044  
 Perthes', 1042  
 Scheuermann's, 887, 1045  
 Schlatter's, 1044  
 osteogenesis imperfecta, 1041  
 osteomalacia, 1035  
 Paget's disease, 1038  
 periosteal blood vessels,  
 1015  
 renal rickets, 1035  
 rickets, 1031  
 scurvy, 1036

- Bone, diseases of—continued**  
 syphilis of, 1028  
 congenital manifestations, 1030  
 gumma, 1029  
 osteitis, 1028  
 periostitis, 1028  
 tuberculosis of, 1025  
 dactylitis, 1027  
 osteitis, 1026  
 periostitis, 1025  
 tumours of, 1046  
 chondroma, 1046  
 Ewing's tumour, 1055  
 multiple myeloma, 1056  
 osteoclastoma, 1049  
 osteoma, 1048  
 sarcoma, 1052  
 secondary tumours of, 1056  
 typhoid osteitis, 1025
- Bouchacourt's sign, 538**
- Bougies, 780**
- Bow leg, 1134**
- Brachial plexus,**  
 anatomy of, 895  
 injury, lower-arm type, 897  
 to cords, 898  
 upper-arm type, 896  
 whole plexus type, 896
- Bradford's spinal frame, 1095**
- Brain,**  
 abscess of, 394, 476, 487, 850  
 compression of, 844  
 concussion, 839  
 contusion of, 841  
 delayed softening of, 842  
 hæmorrhage in, 842  
 hydrocephalus, 852  
 increased intracranial pressure, 839  
 injury, by "coup," 842  
 by "contrecoup," 842  
 irritation of, 843  
 laceration, 841  
 œdema, 842  
 physiology of intracranial circulation, 838  
 tumours of, 854
- Branchial carcinoma, 362**  
 cyst, 114, 353  
 fistula, 353
- Branchiogenetic carcinoma, 362**
- Brasor's ligature for aneurysm, 270**
- Braun's splint, 984, 998, 1006**
- Brawny arm, 291, 521**
- Breast,**  
 abscess, 509, 513  
 acute inflammatory carcinoma, 525  
 anatomy, 504  
 anomalies, 505  
 atrophic scirrhus, 519, 524  
 carcinoma, 518  
 chronic mastitis, 510  
 columnar-celled carcinoma, 526  
 cysts, 514  
 duct papilloma, 515
- Breast—continued**  
 encephaloid carcinoma, 519, 525  
 examination of, 505  
 fat necrosis, 514  
 fibro-adenoma, 516  
 inflammation of, 508  
 male, diseases of, 526  
 nipple and areola, 506  
 peripheral carcinoma, 525  
 pump, 510  
 sarcoma, 526
- Breathing exercises, 187**
- Broad ligament, cysts of, 819**
- Brook's pin, 969**
- Brodie's abscess, 1023**
- Bronchial fistula, 471, 476**
- Bronchiectasis, 483**  
 cerebral abscess in, 487  
 empyema in, 487  
 examination in, 485  
 lobectomy for, 488  
 postural drainage in, 487  
 varieties of, 484
- Bronchography, 224, 486, 490**
- Broncho-pneumonia, 209**
- Bronchoscope, 447**
- Bronchoscopy, 482, 493**
- Brood capsules, 116**
- Brown-Séquard syndrome, 883**
- Bryant's "gallows" method, 986**  
 triangle, 974, 1061
- "Bucket-handle" tear of semilunar cartilage, 992**
- Buerger's disease, 265**
- Bulbar paralysis, progressive, 434**
- Bulla ethmoidalis, 399, 402**
- Buller's shield, 60**
- Bullous œdema, 762**
- Bunion, 1145**
- Burns, 129**  
 classification, 130  
 clinical picture, 133  
 due to chemicals, 141  
 electricity, 140  
 frost, 139  
 X-rays, 141  
 in special sites, 139  
 of eyeball, 139  
 of face, 139  
 of fingers, 139  
 of scalp, 826  
 pathology, 131  
 treatment, 135
- Bursæ, diseases of, 1172**  
 individual bursæ, 1174  
 inflammation of, 1173  
 injury to, 1172  
 os calcis, 1175  
 subhyoid, 362  
 tendo achillis, 1175
- Bursitis,**  
 gonococcal, 60, 1174  
 gouty, 1174  
 hæmorrhagic, 1173  
 serous, 1173  
 suppurative, 1173
- Burnitis—continued**  
 syphilitic, 1174  
 traumatic, 1173  
 tuberculous, 1173
- Butcher's wart, 235**
- Butterfly erythema, 236**

## C

**Cæcostomy, blind, 630**

**Calcaneal spur, 1144**

**Calculus in common bile duct, 703**

in gall-bladder, 701

in kidney, 736

in pancreas, 710

in prostate, 771

in salivary glands, 349

in ureter, 750

in urethra, 790

in urinary bladder, 765

**Calculus anuria, 752**

**Caldwell-Luc operation, 418**

**Caliper walking splint, 1083**

**Callosity, 237**

**Callus, 919**

**Calmette's antivenin, 17, 121**

**Cancellous exostosis, 93, 1048**

fractures, 917

hæmatoma, 916

osteoma, 93, 1048

subungual, 1049

**"Cancer en cuirasse," 521**

**Cancrum oris, 176**

**Capillaries, increased permeability of, 2**

**Capillary angioma, 111, 286**

hæmangioma, 286

hæmorrhage, 154

lymphangioma, 290

**"Caput medusæ," 539**

**Carbolic acid, 180**

gangrene from, 176

**Carbuncle of kidney, 731**

**Carbuncles, 232**

**Carcinoid tumour, 684**

**Carcinoma, 105**

basal-celled, 106, 239

glandular, 106

of anal margin, 665

of bladder, 769

of branchial cleft, 362

of breast, female, 518

male, 527

of bronchus, 491

of cervix uteri, 822

of colon, 627

of common bile duct, 705

of duodenum, 612

of face, 304

of floor of mouth, 346

of gall-bladder, 705

of gums, 322

of jaws, 315

of kidney, 737

of larynx, 457

of lips, 307

of liver, 692



- Carcinoma**—*continued*  
 of lung, 491  
 of oesophagus, 442  
 of ovary, 819  
 of pancreas, 710  
 of penis, 783  
 of pharynx, 436  
 of prostate, 776  
 of rectum, 662  
 of renal pelvis, 749  
 of salivary glands, 351  
 of scrotum, 808  
 of skin, 237  
 of stomach, 594  
 of testis, 801  
 of thyroid, 376  
 of tongue, 342  
 of urethra, 790  
 of uterine body, 821  
 of vulva, 811  
 simplex, 106, 518  
 spheroidal-celled, 106, 518  
 squamous-celled, 105  
 transitional-celled, 106
- Cardiac massage**, 197  
 "tamponade," 468
- Cardiospasm**, 439, 913
- Caries of bone**, 1018  
 necrotica, 1018  
 sicca, 1018, 1072, 1085  
 suppurativa, 1018
- Carotid body tumour**, 362
- Carpal bones**,  
 dislocations, 967  
 fractures, 966
- Carpo-pedal spasm**, 448
- Carrel-Dakin treatment**, 128
- Carriers of infection**, 16
- Carrying angle of arm**, 1117
- Caruncle**, 812
- Caseation**, 39
- Casoni reaction**, 117
- Catalepsy**, 34
- Catarrhal inflammation**, 5  
 jaundice, 688  
 stomatitis, 329
- Catgut**, 184
- Catheter fever**, 729
- Cauda equina**, injuries to, 882
- Causalgia**, 884, 901, 905
- Cautery**, actual, 217  
 electric, 217
- Cavernositis**, 783
- Cavernous angioma**, 111, 287  
 lymphangioma, 111, 290
- Cavernous sinus thrombosis**,  
 275, 852
- Cell nests**, 106
- Cellulitis**, 26  
 clinical picture, 26  
 of neck, 27, 358  
 of orbit, 27  
 of pelvis, 27, 652, 815  
 of scalp, 27, 827  
 of scrotum, 27  
 mentoma, 95, 325  
 phalæmatocoele, 828  
 phalæmatoma, 825  
 phalhydrocele, traumatic, 831
- Cephalocele**, 835
- Cerebellar abscess**, 396, 851  
 tumours, removal of, 867
- Cerebral abscess**, 395, 850  
 aneurysm, 863  
 hernia, 870  
 irritation, 843  
 tumours, removal of, 863
- Cerebrospinal fluid**, escape of,  
 832  
 changes due to compression  
 of cord, 878
- Cerumen**, 384
- Cervical fascia**, 352  
 plexus, 895  
 rib, 354, 911  
 sinus, 353
- Chancere**,  
 extragenital, 65, 433  
 Hunterian, 66  
 in men, 66  
 in women, 67  
 intra-urethral, 66  
 meatal, 66
- Chancroid**, 80
- Charcot's disease**, 71, 1110  
 in syringomyelia, 1111  
 in tabes dorsalis, 71, 1110
- Chauffeur's fracture**, 965
- Chaulmoogra oil**, 42
- Chemical injuries**, 141
- Chemotherapy in gonorrhœa**,  
 55, 62  
 in wounds, 38
- Chest**. See Thorax
- Chevalier Jackson's broncho-  
 scope**, 447  
 laryngoscope, 424, 445
- Cheyne-Stokes breathing**, 158  
 phenomenon, 846
- Chigoe**, 53
- Chilblains**, 236
- Chilney-sweep's cancer**, 808
- Chloramine T**, 181
- Chloroform**, use of, 200  
 complications after, 202  
 indications for and against,  
 201  
 Junker's inhaler, 202  
 Shipway's apparatus for, 202
- Chloroma**, 837
- Choked disc**, 906
- Cholangitis**,  
 acute suppurative, 688  
 catarrhal, 688  
 subacute, 688
- Cholecystectomy**, 705
- Cholecystgastrostomy**, 705
- Cholecystitis**, acute, 696  
 catarrhal, 698  
 empyema of gall-bladder in,  
 698  
 gangrenous, 698  
 suppurative, 698
- Cholecystitis**, chronic, 700  
 strawberry gall-bladder in,  
 700
- Cholecystography**, 226, 694
- Cholecystostomy**, 705
- Choledochotomy**, 705
- Cholesteatoma**, 391, 863
- Cholesterol calculi**, 700
- Chondro-arthritis of Virchow**,  
 1099
- Chondroma**, 92, 1046  
 cystic, 93  
 enchondroma, 92, 1046  
 enchondroma, 92, 1046  
 solitary, 92, 1046  
 spinal, 888
- Chondro-sarcoma**, 101, 1054
- Chordee**, 54
- Chordoma**, 92, 875
- Chorionic carcinoma**, 113, 802,  
 822
- Choroiditis**, 73
- Choroid plexus**, tumours of,  
 863
- Chromophile adenoma**, 857
- Chromophobe adenoma**, 857
- Chronic ulcer**, 165
- Chylous ascites**, 289  
 hydrocele, 289
- Chyluria**, 289
- Circulation**, collateral, 259
- Circulus vasculosus articuli**,  
 1015
- Circumcision**, 781
- Circumflex nerve**, 899
- Cirrhosis of liver**, 663
- Cirroid aneurysm**, 287, 827
- Citrated blood**, 150
- Clavicle**,  
 dislocations of, 940  
 fractures of, 938
- Claw foot**, 1143
- Claw hand**, 902
- Cleft palate**, 330
- Cleido-cranial dysostosis**, 1046
- Clicking jaw**, 318
- Climatic bubo**, 81
- Cloacæ in bone**, 1017
- Cloacal membrane**, persistence  
 of, 647
- Clostridia**, 35
- Clostridium botulinum**, 17  
 tetani, 17, 32  
 welchii, 35
- Clover's inhaler**, 199
- Clubbing of fingers**, 476, 485,  
 1045
- Club foot**, 1143
- Club hand**, 1115
- Clutton's knees**, 73, 1098
- Coagulation therapy**, 136
- Cocaine**, 210
- Coccydynia**, 888, 976
- Coccyx**, fracture of, 976
- Cock's peculiar tumour**, 242,  
 827
- Cock-up splint**, 900
- "Coffee-ground" vomit**, 596
- Cohnheim's theory**, 84
- Cold abscess**, 24, 40
- Cold Agglutinins**, 149
- Coley's fluid**, 48
- Colitis**, 617
- Collapse of lung**, massive, 209

- Collar-stud abscess**,  
in neck, 296  
in palm, 252
- Collateral circulation**, 259
- Colles, fascia of**, 789
- Colles's fracture**, 963
- Colloid carcinoma**, 627, 662
- Colloid goitre**, 368
- Colloids in urine**, 736
- Colon**. See Intestine, Large
- Colostomy**, 664
- Colostomy horn**, 665  
belt, Agordian, 665
- Columna adiposa**, 232
- Comminuted fracture**, 918
- Common bile duct**,  
anatomy, 686  
growths of, 705  
operations on, 705  
stones in, 703
- Complete division of nerve**, 891
- Complete transverse lesion of spinal cord**, 879
- Complicated fractures**, 917
- Composite odontome**, 95, 324
- Compound follicular odontome**, 95, 324
- Compound fractures**, 918  
treatment of, 932
- Compound palmar ganglion**, 1170
- Compression fractures of spine**, 1007
- Compression of brain**, 844  
affect on cardiac centre, 845  
consciousness, 844  
motor cortex, 845  
pupils, 845  
respiratory centre, 845
- Cheyne-Stokes phenomenon**, 846  
diagnosis of, 846  
non-focal symptoms, 846  
treatment of, 847  
vaso-motor reaction, 845
- Compression of spinal cord**, 876  
automatic bladder in, 877  
cerebrospinal fluid in, 878  
motor effects, 877  
sensory changes, 877  
sphincter control, 877  
trophic changes, 877
- Concussion of brain**, 839  
of nerve trunks, 891  
of spinal cord, 875
- Condylomata**, 68, 72
- Confinement to bed**, 187
- Congenital aplasia cranii**, 835
- Congenital dislocation of hip**, 978
- Congenital hernia**, 563
- Congenital polycystic disease**, 744
- Congenital sacro-coccygeal tumour**, 112, 875
- Congenital syphilis of bone**, 71, 1030  
craniotabes, 73, 1030  
dactylitis, 72, 1030
- Congenital Syphilis of Bone—continued**  
epiphysitis, 72, 1031  
hot-cross-bun head, 73, 1031  
Parrot's nodes, 73, 1031  
periosteal, 72, 1028  
pseudo-paralysis, 1031  
symmetrical overgrowth of tibia, 1030
- Conjunctivitis**, post-operative, 210
- Consciousness**, alterations of, in head injury, 839
- Consolidated aneurysm**, 268
- Continuous-drip infusion apparatus**, 145
- Contraction of fingers**, congenital, 1116
- Contraction of hip**, 1149
- Contraction of knee**, 1150
- "Contrecoup" injury**, 842
- Contusion**, 118  
of abdominal wall, 529  
of arteries, 259  
of bladder, 760  
of bones, 916  
of brain, 841  
cerebral irritation in, 843  
delayed softening after, 842  
hemorrhage in, 842  
oedema in, 842  
treatment of, 843  
of intestinal tract, 529  
of kidney, 721  
of knee joint, 989  
of muscles, 1161  
of nerve, 891  
of shoulder joint, 946  
of stomach, 587  
of tendons, 1161
- Corn**,  
hard, 237  
soft, 237
- Coronoid process**, fracture of, 959
- Corpora amylacea**, 773
- Corpora quadrigemina**, tumours related to, 859
- Corynebacterium diphtheriæ**, 17
- Counter-irritants**, 11
- Counter-traction**, 928
- Courvoisier's law**, 704, 711
- "Covent Garden hummy,"** 1175
- Cowper's glands**, 58, 778  
ligament, 504
- Coxa valga**, 1133
- Coxa vara**, 1181  
adolescent, 978  
epiphyseal, 978, 1131
- Cracks of tongue**, 338
- Cramer's skeleton wire splint**, 924
- Cranial nerves**. See Nerves, Cranial
- Craniotabes**, 73, 952
- Crepitus**, 921
- Cretinism**, 364
- Crohn's disease**, 621
- Cross infection**  
of abscess, 23  
of wounds, 128
- Cruciate ligament**, rupture of, 994
- Crush syndrome**, 119
- Crutch palsy**, 894
- Cubitus valgus**, 1117  
varus, 1117
- Culex fatigans**, 291
- Cullen's sign**, 539
- Cupping**, 8
- Cushing's syndrome**, 857
- Cushing's vasomotor reaction**, 845
- Cut throat**, 358
- Cyclopropane**, 207
- Cyst-adenoma of breast**, 517
- Cysticercosis**, 51
- Cystic hygroma**, 361
- Cystin calculi**, 738
- Cystitis**, 761  
acute, 762  
bilharzial, 52  
chronic, 763
- Cystitis cystica**, 764
- Cystography**, 756
- Cystoscopy**, 717, 754  
for diseases of bladder, 754  
for ureteric calculus, 752  
for urinary tuberculosis, 735
- Cystostomy**, suprapubic, 788
- Cysts**, 114  
hydatids, 115  
mucous retention, 308  
of arachnoid, 119  
of Bartholin's gland, 811  
of bone, 1040, 1058  
hydatid, 1058  
spontaneous fracture in, 1058  
of breast, 514  
of broad ligament, 819  
of liver, 692  
of kidney, 742  
of mesentery, 543  
of neck, 353, 361  
of ovary, 817  
of pancreas, 709  
of scalp, 827  
of spleen, 712  
of urachus, 755  
of ureter, 750  
of vulva, 811
- D**
- Dactylitis**,  
syphilitic, 1030  
tuberculous, 1027
- Dakin's solution**, 181
- Dalrymple's sign**, 373
- Deafness**, 386  
due to fractured skull, 834
- "Deal-runner's shoulder,"** 1175
- "Débridement,"** 124
- Deep sensation**, 889  
sensitivity, 889  
therapy, 229

**Defibrinated blood, 150****Deformity, 1113**

- correction of, 219
- in coxa vara, 1133
- infantile paralytic, 1147
- in tuberculous hip, 1076, 1078
- knee, 1082
- of lower extremity, 1125
  - congenital dislocation of hip, 1125
  - coxa valga, 1133
  - coxa vara, 1131
  - dislocation of patella, 1136
  - genu recurvatum, 1135
  - valgum, 1135
  - varum, 1134
- hallux rigidus, 1146
- valgus, 1145
- hammer-toe, 1146
- metatarsalgia, 1147
- painful heel, 1144
- pes cavus, 1143
- planus, 1139
- snapping hip, 1134
- talipes equinovarus, 1137
- of neck and shoulder,
  - cervical rib, 354
  - spasmodic torticollis, 357
  - Sprengel's shoulder, 1114
  - torticollis, 356
- of spine, 1118
  - kyphosis, 1122
  - lordosis, 1124
  - scoliosis, 1118
  - spondylolisthesis, 1155
- of thorax in scoliosis, 1119
- of upper extremities, 1114
  - absence of radius, 1114
  - contraction of fingers, 1116
  - cubitus valgus and varus, 1117
  - Madelung's, 1114
  - "trigger finger," 1117
  - prevention of, 213
  - spastic paralytic, 1152

**Degeneration cysts, 115****Degeneration of nerve, 890****Delayed union of fractures, 920****Delhi boil, 50****Dental cyst, 322**

- infection, 322
- ulcer, 340

**Dentigerous cyst, 94, 324****Depressed fracture, 917**

- of skull, 829

**Dercurm's disease, 91****Dermoid cyst, 114, 238**

- of face, 305
- of neck, 361
- of ovary, 818
- of scalp, 827
- of skin, 237
- of testis, 801
- of tongue, 340

**Descent of testis, 570, 792****Desmoid tumour, 90, 537****Dettol, 181****Deviation of nasal septum, 402****Diabetic arteritis, 265****Diabetic gangrene, 178****Diaphragm, injury to, 467****Diaphragmatic hernia, 580****Diaphysial sclerosis, 1048****Diarrhoea, spurious, 629, 642, 652****Diastase index in urine, 708****Diathermy, 218****Di-chloramine T, 181****Dick test, 19****Dietl's crisis, 721****Differential diagnosis table,**

- of infections of hand, 256
- of swellings in breast, 523
- in testis, 803

**Digitalis, 374****Dilatation of stomach,**

- acute, 591
- chronic, 593

**Dilatation of urethral strictures, 787****Dinner-fork deformity, 964****Diphtheria, 43****Discharge from nipple, 508****Dislocation, 933**

- of ankle, 1004
- of clavicle, 940
- of elbow, 955
- of hip, 976
  - congenital, 1125
- of interphalangeal joints, 971
- of knee, 990
- of metacarpo-phalangeal joints, 971
- of patella, 991
  - habitual, 991
- of penis, 782
- of semilunar, 968
- of shoulder, 942
- of spine, 1010
  - with fracture, 1009
- of temporo-mandibular joint, 317
- of tendons, 1161
- of ulnar nerve, 903
- of wrist, 968
- pathological, 935
- recurrent, 935

**Disruption wounds, 123****Dissecting aneurysm, 267****Dissemination, 86****Distal pulp of fingers, 245**

- infection of, 248

**Distension with overflow, 757****Disuse atrophy, 11****Divarication of recti muscles, 579****Diverticulitis,**

- acute, 623
- chronic, 623
- fistula in, 624
- perforation in, 624
- pneumatocyst in, 624, 717

**Diverticulosis, 622****Diverticulum,**

- Meckel's, 614
- of bladder, 756
- of colon, 622
- of duodenum, 611

**Diverticulum—continued**

- of oesophagus, 440
- of pharynx, 435
- of small intestine, 622

**Division of a nerve,**

- complete, 890
- partial, 894

**Dmelcos, 81****Dorsal abscess, 1092****Doryl, 758****Double penis, 780****Dracunculus medinensis, 53****Dressing wounds, technique of, 128****Droitwich Spa, 1101****Dropped foot, 1150**

- wrist, 899

**Dry gangrene, 171****Duck-like gait, 1127****Ducrey's bacillus, 80****Duct papilloma of breast, 515****Dudgeon's wet film method, 492****Duodenal catheterisation, 685****Dumb-bell tumour, 486****Duodenal tube, 643, 695****Duodenal ulcer,**

- acute, 606
- chronic progressive, 607
- complications of, 609
- duodeno-pyloric stenosis in, 610
- hemorrhage in, 609
- hunger pain in, 608
- penetration of, 609
- perforation of, 609
- symptoms of, 607
- treatment of, 608

**Duodenum,**

- acute ulcer of, 606
- anatomy of, 585
- chronic ileus of, 611
- chronic ulcer, 607
  - complications of, 609
- diverticulum of, 611
- examination of, 225, 580
- fistula, 589
- foreign bodies in, 588
- growths of, 612
- injury to, 588
- uncomplicated ulcer of, 606

**Dupuytren's contracture, 1168**

- fracture, 1001

**Durham's tracheotomy tube, 460****Dysenteric arthritis, 1069****Dysentery, amoebic, 53, 618**

- bacillary, 46, 618

**Dysmenorrhoea, 812, 814****Dyspeptic ulcers, 339****E****Ear,**

- anatomy, 378
- examination of, 380
- external, diseases of, 383
- growths, 391
- middle, diseases of, 387
- operations on, 396

- Ear**—*continued*  
 syphilis, 391  
 tuberculosis, 391  
**Eburnation of bone**, 1102  
 of cartilage, 1102  
**Echondroma**, 92  
**Echymosis**, 142  
**Echinococcal cysts**. See Hydatid Cysts  
**Ectopia testis**, 783  
**Ectopia vesicæ**, 755  
   Stiles' operation in, 755  
**Ectopic gestation**, 816  
**Eczematous ulcer**, 166  
**Effleurage**, 214  
**Egg-shell cracking**, 321, 1050  
**Elbow joint**,  
   dislocations of, 955  
   of radius alone, 956  
   subluxation of head of  
     radius, 956  
   varieties of, 955  
   effusion into, 1062  
   injury to, 957  
   ischemic paralysis, 957  
   myositis ossificans, 958  
   ulnar nerve lesion, 957  
   position for ankylosis, 1063  
   tennis elbow, 956  
   tuberculosis of, 1086  
   abscess formation in, 1086  
**Electric cautery**, 217  
**Electrical burns**, 140  
   injuries, 140  
   methods of treatment, 218  
**Electro-coagulation**, 218  
**Electro-desiccation**, 218  
**Electro-encephalography**, 849,  
   855  
   Berger's waves in, 849, 855  
**Electro-therapy**, 218  
**Elephantiasis**, 291  
   neuromatosis, 90  
**Embolectomy**, 280  
**Embolus gangrene**, 175  
**Embolism**, 278  
   air, 281  
   fat, 162  
   gangrene due to, 175, 279  
   in central artery of retina, 280  
   in intracranial arteries, 280  
   in liver, 280  
   in mesenteric vessels, 280  
   infarction due to, 279  
   of malignant cells, 88  
   pulmonary, 187, 209, 279  
**Embryonic cysts**, 114  
**Emetine**, 53, 691  
**Empyema**,  
   mediastinal, 463  
   surgical, 463, 1012  
**Empyema, acute**, 468  
   aspiration of, 471  
   bilateral, 471  
   brain, abscess in, 471  
   bronchial fistula in, 471  
   intercostal drainage of, 473  
   irrigation of, 474  
   meningitis in, 471  
**Empyema, acute**—*continued*  
   necessitatis, 471  
   negative pressure drainage,  
     474  
   pericarditis in, 471  
   respiratory exercises, 474  
   rib resection, 473  
   treatment of, 472  
**Empyema, chronic**, 475  
   brain abscess in, 476  
   treatment of, 477  
   undrained, 475  
   with bronchial fistula, 476  
   with external sinus, 475  
   with fistula and sinus, 476  
**Empyema of gall-bladder**, 698  
   of joints, 1061, 1066  
**Encephalitis**, otogenic, 394  
**Encephalocele**, 835  
**Encephaloid carcinoma**, 108,  
   519  
**Enchondroma**, 92, 1046  
**"End-bulb"** of divided nerve,  
   891  
**Endaneurysmorrhaphy of Matas**,  
   270  
**Endarteritis obliterans**, 264  
**Endometrioma of ovary**, 818  
   of umbilicus, 540  
   of vulva, 811  
**Endometritis**, 63  
**Endothelioma**, 111, 885  
   of lung, 494  
**Enema tests in intestinal ob-**  
**struction**, 579  
**Eneuresis**, nocturnal, 757  
**Engel-May apparatus**, 980  
**Entamoeba histolytica**, 53, 618  
**Enteritis**, 617  
**Eosinophil leucocyte**, 4, 10  
**Epicritic sensation**, 889  
**Epidermoids**. See Sebaceous  
   Cysts  
**Epididymitis**, non-tuberculous,  
   800  
**Epididymitis**, tuberculous, 798  
   changes in cord, 798  
   in prostate, 798  
   in testis, 798  
   in vesicles, 798  
   chronic, 799  
   subacute, 798  
**Epididymo-orchitis**,  
   acute, 797  
   gonococcal, 54, 797  
   pyogenic, 797  
**Epiglottitis**, 444  
**Epilepsy**,  
   after head injury, 849  
   in intracranial tumours, 854  
**Epiphysis**, separation. See  
   Separation of Epiphyses  
**Epiphysitis**,  
   acute, 1022  
   syphilitic, 1031  
**Epispadias**, 780  
**Epistaxis**, 142, 406  
**Epithelial odontome**, 94, 323  
**"Epithelial pearls,"** 106  
**Epithelioma**. See Squamous-  
   celled Carcinoma  
**Epithelioma adenoides cysticum**,  
   242  
**Epulis**,  
   fibrous, 90, 321  
   myeloid, 96, 321  
**Erb-Duchenne's palsy**, 896  
**Erector spinæ muscle**, rupture  
   of, 1163  
**Ergotism**, 176  
**Erysipelas**, 47  
   of scalp, 827  
**Erythema ab igne**, 243  
**Erythema dose in X-ray therapy**,  
   141, 229  
**Erythema induratum**, 234  
   nodosum, 235  
   pernio, 236  
**Erythrocytæmia**, 911  
**Erythromyalgia**, 911  
**Eserine**, 636, 643  
**Essential renal hæmaturia**, 717,  
   749  
**Ether anaesthesia**, 199  
   by inhalation, 199  
   by insufflation, 200  
   Clover's inhaler for, 199  
   Schimmelbusch's mask for,  
     199  
**Ethmoidal air cells**,  
   acute infection of, 414  
   chronic infection of, 418  
   treatment of, 418  
**Ethyl chloride anaesthesia**, 203  
   Loosely's inhaler for, 203  
**Eusol**, 181  
**Evacuator**, Bigelow, 766  
**Evipan sodium**, 192  
**Ewing's sarcoma**, 1055  
**Excision of joints**, 1063  
   of wounds, 124  
**Exercises**, remedial, 215  
**Exomphalos**, 578, 614  
**Exophthalmic goitre**, 369  
**Exophthalmos**, 373  
   pulsating, 275  
**Exostosis**, 94, 1048  
   ivory, 1049  
   subungual, 1049  
**External auditory meatus**,  
   foreign bodies in, 385  
   furunculosis of, 385  
   injuries to, 384  
   osteoma of, 385  
   wax in, 384  
**External popliteal nerve**, 905  
**External sphincter muscle**, 645  
**External urethrotomy**, 788  
**Extra-articular arthrodesis**,  
   1063, 1075  
**Extracapsular fractures of neck**  
   of femur, 982  
**Extraction of teeth**, 326  
**Extradural abscess**, 389, 847  
   hæmorrhage, 847  
**Extra-uterine gestation**, 816  
   tubal hæmorrhage in, 816  
   rupture, 816

**Extravasation of urine, 760, 789**  
**Extrinsic carcinoma of larynx, 458**  
**Ewing's sarcoma, 1055**  
**Eyeball, burns of, 139**

## F

**Face**

burns, 139  
 cysts, 303  
 deformities, 300  
 development, 300  
 growths, 303

**Facial carbuncle, 233**

**Facial cleft, 303**

**Facial nerve, 907**

Bell's palsy of, 908  
 facial tic in, 908  
 injury to trunk, 908

**Facial palsy, due to fractured skull, 834**

**Facies Hippocritica, 549, 676**

**Facultative aerobes, 15**

parasites, 15

**Faecal fistula, 624**

vomit, 633

**Faecal impaction, 615**

**Fairbank's splint, 397**

**Fallopian aqueduct, 379**

**Faradic current, 218**

**Farabouef's amputation, 175**

**Farcy buds, 44**

pipes, 44

**Fascia,**

diseases of, 1168  
 Dupuytren's contracture, 1168  
 of Colles, 789  
 of Scarpa, 789

**Fascial spaces in palm, 246, 253**

abscesses of, 253

**Fat embolism, 162**

**Fat necrosis, 514**

**Female genital organs, 809**

**Femoral hernia,**

anatomy, 574  
 diagnosis, 575  
 treatment, 575

**Femur, fractures of, 978**

extracapsular, 982  
 in children, 982, 986  
 intracapsular, 979  
 lower end, 986  
 neck, 979  
 shaft, 983  
 trans-trochanteric, 982  
 trochanters, 983  
 upper end, 978

**Fibro-adenoma of breast, 516**

cyst-adenoma, 517  
 hard, 516  
 soft, 517

**Fibroblasts, 4**

**Fibrocystic disease,**

of bone, 1040  
 of jaw, 94, 323  
 of testis, 801

**Fibroid of uterus, 97, 820**

**Fibroma, 89**

of mediastinum, 495

**Fibrosarcoma, 101**

**Fibrositis, 1159**

**Fibrous epulis, 90, 321**

odontome, 95, 325

union of fractures, 920

**Fibula, fractures of, 997**

and tibia, 997

**Figure-of-eight bandage, 938**

**Filaria sanguinis hominis, 291**

**Filariasis, 52**

*F. diurna*, 52, 292

*F. nocturna*, 52, 292

**Fimbrial cysts, 819**

**Fingers,**

avulsion of, 1164  
 burns, 139  
 congenital contraction of, 1116  
 infections, 245  
 rupture of extensor tendon, 1163  
 trigger, 1117

**Finnsen lamp, 235**

**Fissured fractures, 917**

of skull, 828

**Fissure-in-ano, 649**

**Fissures of tongue, 338**

**Fistula, 24**

biliary, 704  
 complicating diverticulitis of colon, 624  
 fecal, 624  
 in-ano, 655  
 of duodenum, 590  
 of parotid gland, 348  
 of rectum, 656  
 of Stenson's duct, 348  
 of stomach, 589  
 recto-urogenital, 647  
 renal, 732  
 urethral, 789  
 vesico-intestinal, 765  
 vesico-vaginal, 765

**Flake sequestrum, 1006**

**Flat foot, 1139**

**Flavine, 181**

**Flexner's bacillus, 46, 618**

**Floating kidney, 720**

**Floor of mouth, carcinoma of, 346**

**Fluctuation, 23**

**Foetal lobulation of kidney, 719**

**Follicular odontome, 94, 324**

**Footwear for flat foot, 1141**

**Forearm space, 254**

**Foreign bodies,**

in appendix, 667  
 in bladder, 767  
 in duodenum, 588  
 in ear, 385  
 in larynx, 447  
 in oesophagus, 425  
 in pharynx, 424  
 in stomach, 588  
 in urethra, 790  
 localisation of, 229

**Formalin, 181**

**Fouadin, 82**

**Fowler's position, 545, 551**

**Fracture-dislocation of spine, 1009**

**Fractures,**

classification of, 917  
 cancellous, 917  
 closed or simple, 918  
 comminuted, 918  
 complete and incomplete, 917  
 complicated, 918  
 depressed, 917  
 fissured, 917  
 greenstick, 917  
 impacted, 918  
 open or compound, 918  
 delayed union, 920  
 treatment of, 930  
 fibrous union, 920  
 mal-union, 920  
 non-union, 920  
 treatment of, 930  
 pathological or spontaneous, 916  
 pseudarthrosis following, 920  
 repair of, 919  
 signs of, 921  
 traumatic, 917  
 treatment of, 922  
 fixation, 923  
 reduction, 923  
 splints, 923  
 X-ray examination in, 922

**Fractures of individual bones,**

acetabulum, 975  
 ankle,  
 astragalus, 1003  
 Dupuytren's, 1001  
 Pott's, 1000  
 Wagstaffe's, 1002  
 anterior spines of ilium, 975  
 base of skull, 831  
 anterior fossa, 832  
 middle fossa, 833  
 posterior fossa, 834  
 carpal scaphoid, 966  
 chauffeur's, of radius, 965  
 clavicle, 938  
 coccyx, 976  
 Colles's, of radius, 963  
 cranial vault, 828  
 crest of ilium, 975  
 femur,  
 great trochanter, 983  
 in children, 986  
 lesser trochanter, 983  
 lower end, 986  
 neck, 979  
 shaft, 983  
 upper end, 978  
 "bulb, 997  
 humerus,  
 lower end, 951  
 shaft, 949  
 upper end, 946  
 ilium, 973, 975  
 ischium, 975  
 mandible, 309

**Fractures of individual bones—***continued*

- maxilla, 309
- metacarpals, 968
- metatarsals, 1006
- neural arches of vertebræ, 1011
- olecranon, 958
- os calcis, 1004
- patella, 987
- pelvic girdle, 973
- phalanges of fingers, 969
- of toes, 1006
- radius, 959
  - and ulna, 961
- ribs, 1012
- sacrum, 975
- scaphoid, carpal, 966
- scapula, 941
- Smith's, 965
- sternum, 1011
- tibia, 995
  - and fibula, 997
- tibial spine, 995
- transverse processes, 1011
- ulna, 958
- vertebral bodies, 1007

**Fraenkel's sign, 416****Fragilitas ossium, 1041****Frenum**

rupture of, 782

**Freyer's prostatectomy, 775****Frey's test, 82****Frohlich's syndrome, 857****Froin's syndrome, 878****Frontal sinusitis,**

- acute, 414
- chronic, 418
- treatment of, 418

**Frost bite, 139****Fulguration, 768****Function,**

- position of, 213
- restoration of, 10

**Functional aphonia, 453****Fungus testis, 801****Furunculosis, 232, 384****Fusiform aneurysm, 266****G****Gait, duck-like, 1127****Galatocoele, 515****Gales aponeurotica, 824****Gall-bladder,**

- anatomy of, 685
- anomalies of, 695
- carcinoma of, 705
- cholecystectomy, 705
- cholecystitis, acute, 696
- chronic, 700
- cholecystography, 694
- empyema of, 698
- fistula, 704
- gall-stones in, 700
- growths of, 705
- injuries to, 696
- investigation of, 226, 693

**Gall-bladder—continued**

- mucocoele of, 698
- "strawberry gall-bladder," 700

**"Gallows method" of Bryant, 986****Gall-stones, 700**

- attempting to migrate, 702
- composition of, 700
- Courvoisier's law, 704
- hypercholesterinæmia in, 701
- in common bile duct, 703
- in gall-bladder, 701
- intestinal obstruction due to, 640

jaundice with, 703

**Galvanic current, 218****Ganglio-neuroma, 98, 496****Ganglion, 1171****Gangrene, 170**

- classification of, 173
- diabetic, 178
- dry, 171
- due to carbolic acid, 176
  - to embolism, 175
  - to thrombo-angiitis obliterans, 265
  - to thrombosis, 278
  - to trauma, 178

infective, 176

moist, 171

of lung, 482

results of, 172

signs of, 171

spreading, 177

threatened, 171

varieties of, 171

vascular, 174

**Gangrenous cystitis, 764****Gangrenous stomatitis, 329****Gas anæsthesia, 204**

and oxygen anæsthesia, 204

**Gas gangrene, 35**

clinical picture, 36

pathology, 35

prognosis, 37

treatment, 37

antitoxin, 38

chemotherapy, 38

**Gasserian ganglion, 869, 907****Gastric fistula, 589****Gastric pathway, 597, 599****Gastric tetany, 593****Gastric ulcer,**

- acute, 600
- chronic, progressive, 600
  - complications of, 602
  - partial gastrectomy for, 601
- symptoms of, 600
  - treatment of, 601
- erosions, 600
- hæmorrhage in, 604
- hour-glass stomach in, 605
- leaking, 603
- malignant change in, 606
- penetration of, 602
- perforation of, 602
- pyloric stenosis in, 605

**Gastro-colic fistula, 589****Gastrojejunal ulcers, 610****Gastroscopy, 587****Gaucher's splenomegaly, 713****Gelenkmaus, 1109****General paralysis of the insane, 74****Genu recurvatum, 1135**

valgum, 1135

varum, 1134

**Geographical tongue, 339****Gestation, extra-uterine, 816****Giant cells, 4**

- in lymphadenoma, 297
- in osteoclastoma, 95, 1049
- in tuberculosis, 39

**Giant-celled sarcoma, 101****Gigantism, 857****Gingivitis, 320****Giraldes, organ of, 804****Glanders, 43****Glandular carcinoma, 108****Glandular fever, 45****Glenard's disease, 616****Glioma, 98, 859, 861****Globus hystericus, 434****Glomangioma, 111, 268****Glossitis,**

- acute superficial, 337
- chronic superficial, 337
- parenchymatous, 337

**Glossodynia exfoliativa, 339****Glossopharyngeal nerve, 909****Glottis, œdema of, 358, 360, 449****Gluteal bursa, 1174****Glycerin-sulphapyridine paste, 128****Glycosuria, 716****Goitre. See Thyroid Gland****Gonococcal arthritis, 60, 1070**

bursitis, 60

**Gonococcus, 54****Gonorrhœa, female, 62**

complications of, 63

tests of cure, 63

treatment, 62

vulvo-vaginitis in children, 64

**Gonorrhœa, male, 54**

complications, 58

extra-urethral infections, 60

metastatic complications, 60

methods of diagnosis, 55

of infection, 54

tests of cure, 61

treatment, 55

**Gonorrhœal ophthalmia, 60**

proctitis, 60, 63

**Gooch's splinting, 923****Gouty bursitis, 1174**

tenosynovitis, 1171

tophi, 234

**Gradenigo, syndrome of, 389****Granular cystitis, 764****Granulation tissue, 4****Graves's disease, 369****Grawitz's tumour, 109, 746****Great sciatic nerve, 903**

sciatica, 904

**Greater tuberosity of humerus,**  
fractures of, 947  
**Greenstick fractures, 917, 1034**  
**Groups, blood, 148**  
tests for, 148  
**Guinea worm, 53**  
**Gumboil, 312**  
**Gumma, 70**  
of bone, 1029  
of breast, 513  
of kidney, 732  
of larynx, 451  
of liver, 689  
of pharynx, 433  
of testis, 800  
of tongue, 340  
**Gummatous infiltration diffuse,**  
of larynx, 451  
of testis, 800  
**Gums,**  
growths of, 321  
hypertrophy of, 319  
infections of, 320  
**Gutter fractures, 829**  
**Gutter splints, 923**  
**Gymnastic exercises for scoli-**  
**osis, 1121**  
**Gynaecomastia, 505**

## H

**"H" substances, 2**  
**Hæmangioma, 286, 303, 307,**  
**837**  
**Hæmarthrosis, 143, 1061, 1109**  
**Hæmatemesia, 142**  
**Hæmatocele, 143, 807**  
tubal, 816  
**Hæmatocolpos, 143**  
**Hæmatoma, 119, 142**  
auris, 384  
of abdominal wall, 529  
of nasal septum, 405  
pulsating, 260, 825  
**Hæmatometria, 143**  
**Hæmatomyelia, 143, 878**  
**Hæmatorrhachis, 143, 878**  
**Hæmatosalpinx, 143**  
**Hæmaturia, 142, 717**  
essential renal, 717  
**Hæmoglobinuria, 717**  
**Hæmoperitoneum, 143**  
**Hæmophilia, 154, 1109**  
**Hæmophilic joints, 1109**  
**Hæmoptysis, 142**  
**Hæmorrhage, 142**  
air hunger in, 144  
arrest of, 144  
arterial and its varieties,  
153  
capillary, 154  
external, 142  
from intra-cranial venous  
sinus, 848  
internal, 142  
intracranial, 847  
venous, 154

**Hæmorrhoids,**  
external, 660  
internal, 657  
complications of, 659  
in carcinoma of rectum,  
663  
of three degrees, 657  
treatment by injection, 658  
by operation, 659  
**Hæmothorax, 143, 467, 1012**  
**Haffkine's vaccine, 18**  
**"Hair-ball," 588**  
**Hairy tongue, 339**  
**Hallux flexus, 1146**  
rigidus, 1146  
valgus, 1145  
**Hammer toe, 1146**  
**Hand - Christian - Schüller dis-**  
**ease, 837**  
**Hare-lip, 300**  
**Harris's prostatectomy, 775**  
**Harrison's sulcus, 1033**  
**Hartmann's pouch, 685**  
**Hay fever, 407**  
**Headache in head injury, 843,**  
**846, 848, 849**  
after concussion, 841  
**Headache of nasal origin, 408**  
**Head injury,**  
See Fractures of Skull, 828  
See Injuries to Brain, 839  
sequelæ of, 848  
**Healing**  
beneath a crust, 5  
by first intention, 4  
by second intention, 5  
process of, 3  
tissue changes in, 3  
**Healing ulcer, 165**  
**Heart, injury to, 468**  
**Heat,**  
as a therapeutic agent, 215  
dry, 216  
moist, 215  
**Hectic fever, 25**  
**Heel, painful, 1144**  
**Hemisection of spinal cord,**  
**883**  
Brown-Séquard's syndrome  
in, 883  
**Heparin, 282**  
**Hepatoma, 691**  
**Hernia, 559**  
acquired, 564  
anatomy of, 559  
causation, 563  
complicated by intestinal  
obstruction, 565  
complications, 565  
congenital, 563  
contents of sac, 563  
inflammation of, 566  
medico-legal aspect of, 565  
reducibility, 566  
symptoms of, 566  
treatment of, 567  
by injection, 568  
by operation, 569  
by taxis, 568

**Hernia, treatment of—continued**  
by truss, 567  
**Hernia testis, 800**  
**Hernia, varieties of,**  
cerebri, 870  
diaphragmatic, 580  
en glissade, 572  
epigastric, 578  
femoral, 574  
incisional, 576  
inguinal, 569  
inguinal, direct, 572  
indirect, 570  
interstitial, 572  
Littre's, 566  
lumbar, 579  
obturator, 580  
perineal, 580  
retroperitoneal  
intersigmoid, 583  
paracæcal, 583  
paraduodenal, 583  
Richter's, 576  
sciatic, 579  
supra-umbilical, 578  
through linea semilunaris,  
579  
umbilical, 578  
**Hernial sac,**  
anatomy, 563  
contents, 563  
**Hernioplasty, 569**  
**Herniorrhaphy, 569**  
**Herniotomy, 569**  
**Hewitt's airway, 196**  
**Hibbs' operation, 1096, 1122**  
**Hilton's rest and pain, 8**  
**Hip, snapping, 1134**  
**Hip joint,**  
acute arthritis of, 1068  
adduction deformity of, in  
spastic paraplegia, 1153  
Bryant's triangle, 974, 1060  
contracture of, in infantile  
paralysis, 1149  
dislocations of (see below),  
977  
effusion into, 1061  
examination of, 1060  
Nelaton's line, 974, 1060  
osteo-arthritis of, 1104  
position for ankylosis, 1063  
Trendelenburg's sign, 1061  
tuberculosis of (see below),  
1076  
**Hip joint, dislocation of,**  
anterior, 977  
central, 975  
congenital, 1125  
changes in bone, 1125  
soft parts, 1126  
duck-like gait in, 1127  
telescopic movement in,  
1127  
treatment of, 1128  
Trendelenburg's sign in,  
1128  
irregular, 978  
posterior, 976

- Hip joint, tuberculosis of**, 1076  
 abscess formation in, 1081  
 diagnosis, 1078  
 first stage,  
   apparent lengthening, 1076  
   deformity, 1076  
   limitation of movement,  
     1076  
   muscular wasting, 1076  
   "night cries," 1078  
   X-ray appearances, 1077  
 prognosis, 1079  
 second stage,  
   deformity, 1078  
   shortening, 1078  
 treatment, 1079  
   of neglected cases, 1081  
   operative, 1080
- Hirschsprung's disease**, 615
- Histrionic spasm**, 908
- Hodgen's splint**, 981
- Hodgkin's disease**. See Lymph-adenoma
- Hollow viscera**, injury to, 529
- Horner's syndrome**, 492, 817, 879
- Horse-shoe abscess**, 654  
 fistula, 655  
 kidney, 719
- Hot-cross-bun head**, 73, 1031
- Hot fomentations**, 215
- Hour-glass stomach**, 605
- Housemaid's knee**, 1175
- Houston**, valves of, 646
- Humerus, fractures of**, 946  
 greater tuberosity, 947  
 head and anatomical neck, 946  
 lower end, 951  
   capitellum, 954  
   intercondylar, 951  
   internal condyle, 954  
   separation of lower epiphysis, 952  
   supracondylar, 951  
 separation of upper epiphysis, 948  
 shaft, 949  
 surgical neck, 947
- Hunchback**, 1123
- Hunger pain**, 606
- Hunterian chancre**, 66  
 ligature, 270
- Hutchinson's teeth**, 73
- Hydatid cyst**, 115  
 of bone, 1058  
 of kidney, 745  
 of liver, 692
- Hydatid of Morgagni**, 804
- Hydatidiform mole**, 113, 823
- Hydrocele**—  
 of hernial sac, 565  
 of neck, 361  
 of tunica vaginalis, 803  
   acquired, 804  
   congenital, 804  
   encysted, of cord, 804  
   idiopathic, 805  
   radical operation for, 806
- Hydrocele**—*continued*  
 tapping of, 806
- Hydrocephalus**, 852
- Hydrogen peroxide**, 181
- Hydronephrosis**, 723  
 bilateral, 724  
 closed, 724  
 intermittent, 724  
 pelvic, 724  
 renal, 724  
 unilateral, 723
- Hydrophobia**, 44
- Hygroma, cystic**, 361
- Hyperæmia**,  
 active, 1, 9, 10, 215  
 passive, 9, 26
- Hyperchlorhydria**, 597
- Hypercholesterinæmia**, 701
- Hyperidrosis**, 914
- Hypernephroma**, 109, 746
- Hyperplasia of thyroid at puberty**, 366
- Hypertension**, essential, 913
- Hyperterm**, 60
- Hyperthyroidism**, 369
- Hypertrophic pulmonary osteoarthropathy**, 1045
- Hypertrophy of breast**, 505
- Hypoglossal nerve**, 910
- Hypospadias**, 780
- Hyrtl's bloodless line**, 714
- Hysterical joints**, 1111
- I**
- Ichthyosis of tongue**, 338
- Ileo-cæcal tuberculosis**, 620
- Ileus**,  
 duodenal, 611  
 duplex, 682
- Ilium**,  
 fractures of anterior spine, 975  
 of crest, 975
- Immunity**, 17  
 acquired, 18  
 active, 18  
 natural, 17  
 passive, 18
- Impacted fracture**, 918
- Imperfectly descended testis**, 793  
 effect on function, 793  
 on vulnerability, 794  
 Pregnyl in, 794
- Imperforate anus**, 647
- Impetigo contagiosa**, 234
- Implantation cyst**, 115, 238  
 dermoid, 115, 238
- Impulse on coughing**, 566
- Incarceration of hernia**, 565
- Incised wounds**, 120
- Incisional hernia**, 576
- Incompatible transfusion**, 149
- Incontinence of urine**, 757
- Increased intracranial pressure**, 839  
 clinical picture, 839  
 methods of reducing, 843  
 stages of, 839
- Indelible pencil injuries**, 237
- Indigo calculus**, 739
- Industrial alcohol**, 181
- Infantile paralysis**, 1147  
 clinical stages of, 1148  
 deformities in, 1149  
   of ankle, 1150  
   of hip, 1149  
   of knee, 1150
- Infants**,  
 mixed tumour of, 747
- Infarction**, 279
- Infarction**, due to embolism, 279
- Infected wounds**,  
 treatment of, 126
- Infection**, 15  
 diffuse, 26  
 generalised, 28  
 latent, 16  
 spread of, 129<sup>1</sup>
- Infections due to worms**, 51
- Infective gangrene**, 176
- Infiltration**, 85
- Inflammation**, 1  
 blood cells in, 2  
 causes of, 1  
 colliquative necrosis, 2  
 diapedesis, 2  
 end results of, 3  
 exudate in, 2  
 plasma cells in, 4  
 tissue cells in, 3  
   changes in, 3  
 transudation of serum in, 2  
 types of, 5  
 vascular changes in, 1
- Inflammation, acute**, 6  
 general manifestation of, 7  
 local manifestation of, 6  
 treatment of, 8
- Inflammation, chronic**, 10  
 clinical picture of, 10  
 treatment of, 10
- Infra-red rays in treatment**, 216, 656
- Infusion of saline**. See Saline Infusion
- Ingrowing toe-nail**, 244  
 Watson-Cheyne's operation for, 244
- Inguinal hernia**,  
 anatomy, 569  
 direct, 572  
 en glissade, 572  
 indirect, 570  
 interstitial, 572
- Injection treatment for hæmorrhoids**, 658  
 of varicose veins, 286
- Injection treatment of hernia**, 568
- Injury to**—  
 abdominal contents, 529  
 wall, 529  
 arteries, 259  
   and veins, 262  
 atlas and axis, 1011  
 biliary system, 696



- Injury to—continued**  
 bladder, 760  
 bone, 915  
 brain, 840  
 chest, 462  
 diaphragm, 467  
 duodenum, 588  
 fingers and hand, 247  
 heart, 468  
 joints, 933  
 kidney, 721  
 knee joint, 989  
 liver, 531, 534, 686  
 muscles, 1161  
 neck, 358  
   cut-throat, 358  
 nerves, 890  
 pancreas, 532, 706  
 penis, 782  
 rectum, 648  
 salivary glands, 347  
 scalp, 825  
 skull, 828  
 spinal cord, 875  
 spine, 1007  
 spleen, 532  
 stomach, 587  
 tendons, 1161  
 testis, 795  
 thoracic duct, 289  
 tongue, 337  
 ureter, 749  
 urethra, 784  
 veins, 280  
 viscera, hollow, 529  
   solid, 531
- Innocent connective tissue tumours, 89**  
 epithelial tumours, 102
- Insect stings, 121**
- Instrumental pyelography, 227**
- Intensifying screens, 222**
- Intercostal nerves, 903**
- Intermittent claudication, 264**
- Internal condyle of humerus, fracture of, 954**
- Internal derangement of joint, 934**  
 of knee, 991
- Internal popliteal nerve, 905**
- Internal urethrotomy, 787**
- Interphalangeal joints, 971**
- Intersigmoid hernia, 583**
- Interstitial keratitis, 73**
- Intestinal obstruction, acute, 631**  
 due to intussusception, 663  
   gall-stones, 640  
   mesenteric vascular occlusion, 541, 644  
   paralytic ileus, 642  
   strangulated hernia, 635  
   strangulation by bands, 635  
   volvulus, 639  
 etiology, 631  
 general signs, 633  
 symptoms, 632  
 treatment, 634  
 with strangulation, 631  
 without strangulation, 632
- Intestinal obstruction, chronic, 641**
- Intestinal stasis, 616**
- Intestinal tract,**  
 absence and atresia, 614  
 anatomy, 613  
 development, 613  
 examination, 614  
 exomphalos, 614  
 inflammation of, 617  
 injuries to, 529-532  
 intestinal stasis, 616  
 lymph drainage, 613  
 Meckel's diverticulum, 614  
 neoplasms of, 626  
 tuberculosis of, 619  
 visceroptosis, 616
- Intestine, large,**  
 colitis, 617  
 congenital idiopathic dilatation of, 615  
 diverticulitis, acute, 622  
   chronic, 623  
   complications of, 624  
 dysentery, 618  
 faecal impaction, 615  
 growths of,  
   adenoma, 626  
   carcinoma, 627  
   polypi, 626  
   paratyphoid, 618  
   penetrating wounds, 532  
   rupture, 529  
   stricture, 625  
   tuberculosis, 619
- Intestine, small,**  
 contusion, 529  
 enteritis, 617  
 faecal fistula, 624  
 growths, 626  
 Meckel's diverticulum, 614  
 penetrating wounds, 532  
 regional ileitis, 621  
 rupture, 529  
 stricture, 625  
 tuberculosis, 619  
 typhoid, 618
- Intracanalicular fibro-adenoma of breast, 516**
- Intracapsular fracture of neck of femur, 979**
- Intracranial hæmorrhage, 847**  
 chronic subdural, 848  
   hæmorrhagic cyst in, 848  
 delayed, of Bollinger, 848  
 extradural, 847  
 from venous sinuses, 848  
 in new-born babies, 848  
 subdural, 848
- Intracranial operations,**  
 technique for, 863
- Intracranial tumours, 854**  
 arteriography in, 855  
 at base of brain, 857  
 electro-encephalography in, 855  
 epilepsy in, 854  
 localisation of, 854  
 of brain, 861
- Intracranial tumours—continued**  
 of meninges, 861  
 surgical technique in, 863  
 vascular, 863  
 ventriculography, 854
- Intramammary abscess, 509**
- Intraperitoneal abscess, 553**
- Intravaginal torsion of testes, 795**
- Intravenous urography, 227,**  
 717, 725, 735, 740, 748
- Intrinsic carcinoma of larynx, 457**
- Intussusception, 636**  
 acute, 638  
 apex of, 637  
 chronic, 639  
 colic, 638  
 ensheathing layer, 637  
 enteric, 637  
 entering layer, 637  
 enterocolic, 638  
 ileocaecal, 638  
 ileocolic, 638  
 intussusceptum, 637  
 intussusciens, 637  
 Jessett's operation, 639  
 neck of, 637  
 reducibility, 637  
 returning layer, 637  
 types of, 637
- Involucrum, formation of, 1017**
- Iodine, 180**  
 baths, 216, 258  
 deficiency in thyroid disease, 367  
 Lugol's solution, 373  
 medication, 373  
 salt, 367
- Ionisation, 218**
- Iritis,**  
 gonococcal, 60  
 syphilitic, 60
- Irritable ulcers, 166**
- Ischaemic paralysis, 957, 1166**
- Ischial bursa, 1174**
- Ischiorectal abscess, 653**
- Ischium, fracture of, 975**
- Ivory osteoma, 93, 1049**

## J

- Jackets in scoliosis, 1121**
- Jacksonian fits, 839, 845**
- Jaundice,**  
 acholic, 713  
 catarrhal, 688  
 with gall-stones, 703  
 with pancreatitis, 709
- Jaws, diseases of, 309**  
 infections, 311  
 injury, 309  
 growths, 314  
 necrosis, 312
- Jejunal ulcer, 610**
- Jessett's operation, 639**
- Joffroy's sign, 373**

**Joint mice, 1109****Joints,**

- acute infective arthritis, 1065
  - of special joints, 1067
- anatomy of, 915
- architecture of, 1059
- arthritis deformans, 1099
- diseases of, 1059
- dislocation, 934
  - recurrent, 935
- effusion into, 1061
- empyema of, 1061
- examination of, 1060
- hemarthrosis, 1061
- hemophilia of, 1109
- hysterical, 1111
- inflammation of, 1061
- injury, 933
- internal derangement of, 934, 991
- loose bodies in, 934, 1108
- neuropathic diseases of, 1110
- osteo-arthritis, 1102
- pathological dislocation, 934, 1065
- pyogenic infections of, 1064
- repair of joint injuries, 935
- rheumatoid arthritis, 1060
- specific types of arthritis, 1069
- sprains, 933
- Still's disease, 1101
- subluxation, 934
- synovitis, 1064
- syphilis, 1098
- traumatic synovitis, 1064
- treatment of joint injuries, 936
- tuberculosis of, 1071
- villous arthritis, 1059
- wounds of, 933, 936

**Jugular syndrome, 852****Junker's inhaler, 202****Jutte's tube, 209****K****Kahn test, 75****Kangaroo tendon, 185****Kangri cancer, 84, 537****K.C.C., 890****Keimboch's disease, 1045****Kelly's proctoscope, 647****Keloid scar, 13, 89****Keratoderma blenorrhagica, 61****Keratosis of tonsil, 432****Kernig's sign, 1148****Ketogenic diet, 728****Kidney,**

- actinomycosis, 732
- additional, 718
- anatomy, 714
- anomalies, 718
- bilharzia of, 732
- calculi, 736
- carbuncle, 731
- carcinoma, 747
- cystoscopy and ureteric catheterisation, 717

**Kidney—continued**

- cysts, 742
- examination of urinary tract, 715
  - of urine, 716
- fistula, 732
- fœtal lobulation, 719
- Grawitz' tumour, 746
- growths, 745
- horse-shoe, 719
- hydronephrosis, 723
- hypernephroma, 746
- infections of, 726
- injury to, 721
- ketogenic treatment, 728
- mandelic acid therapy, 728
- misplaced, 719
- movable, 720
- oxaluria, 742
- perinephric abscess, 730
- perinephritis, 730
- primary renal hypertension, 725
- pyelitis, acute, 726
  - chronic, 731
  - in pregnancy, 728
  - of children, 728
- pyonephrosis, 729
- renal efficiency tests, 717
- sarcoma, 747
- syphilis, 732
- teratoblastoma, 747
- tuberculosis, 733

**Kieselbach's area, 406****Kirschner's wire apparatus, 927**

- in fractures of femur, 985

**Klebs-Loeffler bacillus, 43****Klumpke's palsy, 897****Knee joint,**

- acute arthritis, 1067
- contusion, 989
- dislocation, 990
  - of patella, 991
- internal derangements, 992
- fracture of tibial spine, 995
- injury to external cartilage, 994
- injury to internal cartilage, 992
- loose bodies, 995
- nipping of a synovial fringe, 991
- rupture of cruciate ligaments, 994
- sprains, 989
- tuberculosis, 1081

**Knight's brace, 1134****Koch's bacillus, 38**

- tuberculin, 41

**Kocher's method of reducing**

- dislocated shoulder, 944

**Kohler's disease, 1044****Kohlmann's dilator, 787****Kondoleon's operation, 52, 292****Kraurosis vulvæ, 810****Kromayer lamp, 433****Kruckenberg tumour, 88, 595, 819****Kummel's disease, 1008****Kyphosis, 1122**

- adolescent, 1123
- adult, 1124
- in tuberculous spine, 1088

**L****Laceration of brain, 841****Lamina of vertebrae, fractures of, 1011****Laminectomy, indications for and against, 883**

- for spinal tumours, 885

- for tuberculous spine, 1098

**Langhan's cells, 113****Large round-celled sarcoma, 100****Laryngeal mirror, 424, 445****Laryngectomy,**

- complete, 461
- partial, 460

**Laryngismus stridulus, 448****Laryngitis,**

- acute, 448
- atrophic, 449
- chronic, 449

**Laryngofissure, 460****Laryngoscope, 424, 445****Laryngotomy, 457****Larynx,**

- anatomy, 444
- examination, 445
- foreign bodies in, 447
- growths, 455
- infection, acute, 448
  - chronic, 449
- operations on, 459
- paralysis, 452
- syphilis, 451
- tuberculosis, 450

**Latent infection, 16**

- test for, 983

**Lateral fistula of neck, 353****Lateral ligaments**

- of ankle, 1004
- of knee, 989

**Lateral sinus thrombosis, 392, 852**

- jugular syndrome in, 852

**Leaking gastric ulcer, 603****Leather-bottle stomach, 593, 595****Leiomyoma, 97, 1167****Leiomyosarcoma, 102****Leontiasis ossea, 314****Leprosy, 41****Leucocytes, increase in number of, 23**

- margination of, 2

**Leukoplakia, 337**

- vulvæ, 810

**Levator ani muscle, 646****Ligament of Cowper, 504****Ligature material, 184****Lightning stroke, 140****Limbs, elevation of, 8****Limitation of movement in tuberculous arthritis, 1073, 1076**

**Lindau's disease**, 863  
**Lingual cancerophobia**, 346  
   carcinoma, 342  
   thyroid tumour, 340  
   ulcers, 339  
**Linitis plastica**, 593  
**Linseed poultice**, 216  
**Lipoma**, 90  
   arborescens, 1103  
**Lips**, 305  
   growths of, 307  
   ulcers of, 306  
**Liquorrhoea**, 770  
**Lithalopary**, 766  
**Lithotomy**, 767  
**Lithotrite**, 766  
**Little's area**, 406  
   disease, 1152  
**Littre's gland**, 55, 779  
   infections of, 58  
**Littre's hernia**, 566  
**Liver**,  
   anatomy, 685  
   anomalies, 686  
   cirrhosis, 693  
   cysts, 692  
   growths, primary, 691  
     secondary, 692  
   infections,  
     actinomycosis, 689  
     amebic abscess, 689  
     cholangitis, acute, 688  
     catarrhal, 688  
     subacute, 688  
     gas gangrene, 689  
     pyelphlebitis, 687  
     syphilis, 689  
     tuberculosis, 689  
   penetrating wounds, 534, 686  
   rupture, 531, 686  
**Lobectomy, pulmonary**, 482, 488  
**Local anaesthesia**. See Analgesia  
**Localised peritoneal abscess**, 553  
   due to appendicitis, 677  
**Loose bodies in joints**, 934, 1108  
   in knee, 995  
   joint mice, 1109  
   melon-seed bodies, 1170  
   varieties of, 1108  
**Loosely's inhaler**, 203  
**Lordosis**, 1124  
   in tuberculous hip, 1076  
**Lorenz bifurcation osteotomy**, 1130  
**Low back pain**, 1154  
**Lower motor neurone paralysis**, 882  
**Lozenge-shaped vertebra**, 1119  
**Ludwig's angina**, 27, 328, 358, 427  
**Lugol's iodine**, 375  
**Lumbago**, 1159, 1169  
**Lumbar abscess**, 1092  
   hernia, 579  
**Lumbo-sacral plexus**, 903  
   strain, 1157

**Lung**,  
   abscess, 477  
   blast injury, 464  
   bronchiectasis, 483  
   growths, 490  
   injuries, 462, 465  
   tuberculosis, 497  
**Lupus erythematosus** 236,  
   vulgaris, 235, 433  
**Luxatio erecta**, 943  
**Lymphadenitis**,  
   acute, 292  
   chronic, 293  
**Lymphadenoid goitre**, 366  
**Lymphadenoma**, 296  
   of mediastinal glands, 495  
   of spleen, 713  
**Lymphangiectasia**, 111  
**Lymphangioma**, 290, 303  
**Lymphangioplasty**, 292  
**Lymphangitis**,  
   acute, 255, 289  
   chronic, 289  
   syphilitic, 290  
   tuberculous, 290  
**Lymphatic drainage of**  
   breast, 504  
   hand, 247  
   intestinal tract, 613  
   rectum, 646  
   scalp, 824  
   stomach and duodenum, 585  
   testis, 791  
   tongue, 328  
**Lymphatic glands**,  
   growths of, 299  
   infections of, 292  
   in syphilis, 67, 69  
**Lymphatic vessels**,  
   growths of, 290  
   infections of, 289  
   injury to, 289  
   obstruction to, 291  
**Lymphocytes**, 4  
**Lympho-epithelioma of pharynx**, 436  
**Lymphogranuloma inguinale**, 81  
**Lymphosarcoma**, 101, 299, 543  
**Lysol**, 181

## M

**MacCarthy's resectoscope**, 775  
**MacCrae Aitken's jacket**, 1121  
**Macrocephaly**, 835  
**Macrocheilia**, 305  
**Macroglossia**, 336  
**Macrostoma**, 302  
**Madelung's deformity**, 1114  
**Madura foot**, 51  
**"Main d'accoucheur,"** 593, 861  
**"Main-en-griffe,"** 902  
**Maisonneuve's urethrotome**, 787  
**Malarial inoculation**, 78  
   splenomegaly, 713  
**Male breast**, 526  
**Malignant connective tissue growths**, 96  
   epithelial growths, 105  
**Malignant pustule**, 42  
**Malignant syphilis**, 68  
**Mallein**, 43  
**Mallet finger**, 971, 1163  
**Malunion of fractures**, 920  
**Mandelic acid therapy**, 728  
**Mandible, fractures of**, 309  
   dental splints in, 310  
**Mandibular cleft**, 303  
**Manipulation of joints**, 1104  
   in flat foot, 1083  
   of ankle, 1107  
   of hip, 1106  
   of knee, 1107  
   of shoulder, 1107  
**Mantoux test**, 19  
**"March" fracture**, 1006  
**Marie-Strumpell's type of spondylitis deformans**, 887  
**Marjolin's ulcer**, 14, 170  
**Mass reflex**, 880  
**Massage**, 214  
   after fractures, 929  
**Massive collapse of lung**, 209  
**Mast cell**, 4  
**Mastitis, acute**, 508  
   stages, 509  
   treatment, 510  
   varieties, 508  
**Mastitis, chronic**, 510  
   blue-domed cysts, 511  
   in male breast, 526  
**Mastitis Neonatorum**, 508  
   of puberty, 509  
**Mastitis, tuberculous**, 513  
**Mastoid antrum**, 379  
   acute infection of, 389  
   operations on, 396  
**Matas' operation**, 270  
**Max Page's operation**, 1167  
**Maxilla, fractures of**, 309  
**Maxillary antrum**,  
   acute infection, 414  
   chronic infection, 418  
   treatment, 418  
**Measly pork**, 51  
**Meckel's diverticulum**, 614  
**Median nerve**, 900  
**Mediastinal abscess**, 1097  
   tumours, 495  
     of lymph glands, 497  
     of nerve tissue, 496  
     of thymus, 496  
     of thyroid, 495  
**Mediastinal emphysema**, 463  
**Mediastinitis**, 27, 358, 435  
**Medullated nerve**, 899  
**Medulloblastoma**, 98, 962  
**Melasma**, 142  
**Melanoma**, benign, 110, 241, 303  
**Melano-carcinoma**, 110, 241-304  
**Melano-sarcoma**, 110, 241-304  
**Meleney's gangrene**, 177  
   ulcer, 167

**Melon-seed bodies**, 1027, 1170  
**Ménière's disease**, 869, 911  
**Meningioma**, 858, 861  
     classical types of, 860  
     effect on bone over, 861  
**Meningitis**  
     cranial, 849  
     otitic, 393  
     serosa circumscripta, 884  
     spinal, 884  
     tuberculous, 1074  
**Meningocele**,  
     cranial, 835  
     spinal, 873  
**Meningocephalocele**, 835  
**Meningomyelocele (spinal)**, 873  
**Menorrhagia**, 813  
**Mercury biniodide**, 180  
**Mesenteric vascular occlusion**,  
     541, 644  
**Mesentery**,  
     affections of, 541  
     cysts, 543  
     tabes mesenterica, 542  
     tumours, 543  
**Metacarpals**, fractures of, 968  
**Metacarpo-phalangeal joint**, dis-  
     location of, 971  
**Metatarsal bar**, 1147  
     crescent, 1147  
**Metatarsalgia**, 1147  
**Metatarsals**, fractures of, 1006  
**Methods of removing cerebellar**  
     **tumours**, 867  
     cerebral tumours, 863  
**Mickulicz', von, disease**, 349  
**Microcephaly**, 835  
**Microstoma**, 302  
**Middle ear**. See Otitis Media  
**Middle meningeal artery**,  
     hæmorrhage from, 775,  
     780, 783  
**Middle palmar space**, 246  
     abscess of, 253  
**Milk engorgement**, 509  
**Millin's operation**, 758  
**Miner's elbow**, 1174  
**Mirault's operation for hare-lip**,  
     302  
**Mixed parotid tumour**. See  
     Salivary Gland Adenoma  
**Mixed tumour of children**, 113,  
     747  
**Moebius' sign**, 873  
**Moist gangrene**, 171  
**Mole**, pigmented, 241  
**Molluscum contagiosum**, 104,  
     236  
     fibrosum, 89  
**Monckeberg's disease**, 261  
**Mononucleosis**, infective, 45  
**Moon's teeth**, 73  
**Morbus coxæ senilis**, 1104  
**Morgagni**,  
     columns of, 646  
     crypts of, 646  
     hydatids of, 804  
**Morphia**, 191  
**Morton's toe**, 1147

**Mouth**,  
     development of, 327  
     floor of, 330  
     stomatitis, 329  
**Movable kidney**, 720  
**Movable spleen**, 711  
**Movements**,  
     active, 215  
     passive, 215  
**Mucocele of appendix**, 683  
     of gall-bladder, 698  
     of nasal sinuses, 419  
**Mucomembranous colitis**, 617  
**Mucous adenoma of umbilicus**,  
     540  
**Mucous cyst**, 306  
**Multilocular pseudomucinous**  
     **cyst of ovary**, 817  
**Multiple myelomatosis**, 97, 1056  
**Multiple neurofibromatosis**, 895  
**Mummery's excision of rectum**,  
     664  
**Murphy's sign**, 693  
**Muscle**,  
     drill, 187  
     hernia of, 1161  
     inflammation, 1165  
     injury to, 1161  
     rupture of, 1162  
     tumours, 1167  
     wounds, 1164  
**Muscular wasting**, 1073  
**Musculo-spiral nerve**, 899  
     dropped wrist in injury to, 899  
**Mycetoma**, 51  
**Myelitis**, transverse, 884  
**Myelocele**, spinal, 873  
**Myeloid epulis**, 96, 321  
**Myeloma of bone**. See Osteo-  
     clastoma  
**Myeloma of tendon sheath**, 1172  
**Myelomatosis**, multiple, 97, 1056  
**Myeloxanthoma**, 1172  
**Myoma**, 97  
**Myosarcoma**, 102  
**Myosarcoma of stomach**, 597  
**Myositis**,  
     acute suppurative, 1165  
     chronic,  
         actinomycotic, 1165  
         syphilitic, 1165  
         tuberculous, 1165  
     parasitic, 1165  
     simple, 1165  
     toxic, 1165  
**Myositis fibrosa**, 1166  
**Myositis ossificans**, 958, 1166  
     traumatica, 1166  
**Myxœdema**, 364  
**Myxoma**, 91  
**Myxosarcoma**, 101

**N**

**Nails**, affections of, 244  
     ingrowing toe-nail, 244  
     onychogryphosis, 244  
     paronychia, 249  
**Nasal bones**, fracture of, 406  
**Nasal obstruction**, 420  
     polypi, 412  
     septum, 404  
     abscess of, 405  
     deviation of, 404  
     hematoma of, 405  
     perforation of, 405  
     sinuses, 413  
     growths arising in, 420  
     inflammation, acute, 413  
         catarrhal, 413  
         chronic, 415  
**Naughton Dunn's reconstruction**  
     **of foot**, 1139, 1144, 1151  
**Neck**, 352  
     anomalies of development,  
         353  
     cysts, 352, 361  
     development, 352  
     growths, 362  
         branchiogenetic carcinoma,  
             362  
         carotid body tumours, 362  
     inflammation, 358  
         Ludwig's angina, 37, 329,  
             358  
     injury, 358  
         cut throat, 359  
**Necrosis**, 3  
**Nelaton's line**, 974, 1060  
**Nembutal**, 192  
**Neo-arsphenamine**, 77  
**Nephropexy**, 721  
**Nephroptosis**, 720  
**Nephrostomy**, lumbar, 752  
**Nephrotoxin**, 726  
**Nerves**, autonomic, 910  
**Nerves**, cranial, 905  
     abducent, 907  
     auditory, 909  
     facial, 907  
     glossopharyngeal, 909  
     hypoglossal, 910  
     involved in fractures of skull,  
         833  
     oculomotor, 906  
     olfactory, 906  
     operations on, 867  
     optic, 906  
     pathetic, 906  
     spinal accessory, 910  
     trigeminal, 906  
     vagus, 909  
**Nerves**, peripheral,  
     anatomy, 889  
     anterior crural, 903  
     brachial plexus, 895  
     cervical plexus, 895  
     circumflex, 899  
     deep sensibility in, 889  
     degeneration of divided  
         nerve, 890  
     epicritic sensation, 889  
     external popliteal, 905  
     great sciatic, 903  
     injury to, 890  
         complete division, 890  
         concussion, 890  
         contusion, 890

**Nerves, peripheral**—*continued*  
 intercostal, 903  
 internal popliteal, 905  
 lumbo-sacral plexus, 903  
 median, 900  
 multiple neurofibromatosis, 895  
 musculospiral, 899  
 neuralgia, 894  
 neuritis, 894  
 neuroma, 895  
 obturator, 903  
 of Bell, 898  
 partial division of, 894  
   causalgia in, 894  
 phrenic, 500, 895  
 physiology, 889  
 pressure on, 894  
 primary suture, 892  
 protopathic sensation, 889  
 reaction of degeneration, 890  
 regeneration, 890  
 secondary suture, 893  
 signs of recovery, 892  
 stereognosis, 889  
 suprascapular, 899  
 tibial, 905  
 Tinel's sign, 892  
 ulnar, 901

**Nervous lesions in Pott's disease,**  
 1092

**Neural arches, fracture of,** 1011

**Neuralgia, 894**

**Neurasthenia,**  
 traumatic, 829, 848  
 spinal, 883

**Neurilemma, 826**

**Neuritis, 894**

**Neuroblastoma, 98**

**Neurofibroma of mediastinum,**  
 496

**Neurofibroma of scalp, 828**

**Neurogenic bladder, 758**  
 shock, 155

**Neurofibromatosis, multiple, of**  
 von Recklinghausen, 895

**Neuroma, 98, 895**  
 plexiform, 895  
 traumatic, 891, 895

**Neuropathic arthropathy, 1110**

**Neurotrophic ulcers, 166**

**Newmarket method, 939**

**Nicola's method, 946**

**"Night cries," 1073**

**Nigrities, 339**

**Nipped synovial fringe, 991**

**Nipple,**  
 discharge from, 508  
 eczema, 506  
 Paget's disease, 507  
 retraction, 506

**Nitrous oxide anaesthesia, 204**

**Nitrous oxide and oxygen anaesthesia, 204**  
 complications after, 207  
 flow-meter for control of,  
 205

**Nocturnal enuresis, 757**

**Nodular goitre, 869**

**Noma vulvae, 177**

**Non-filarial elephantiasis, 292**

**Non-medullated nerve, 889**

**Non-union of fractures, 920**

**Nose,**  
 anatomy, 399  
 foreign bodies in, 406  
 growth of, 420  
 inflammation of,  
   rhinitis, acute, 409  
   atrophic, 411  
   chronic hypertrophic,  
   410  
 injuries to, 406  
 manifestations of allergy,  
   asthma, 408  
   hay-fever, 407  
   spasmodic rhinitis, 407  
 obstruction, 402  
 polypi, 412  
 septum, disease of, 404  
 sinuses, diseases of, 413

**Notch of Rivini, 378**

**Novocain, 211**  
 with adrenalin, 211

**Nucleus pulposus, prolapse of,**  
 888

**Nystagmus of pharynx, 434**

## O

**Oat-celled sarcoma, 101**

**Obturator hernia, 580**  
 nerve, 903

**Occipito-frontalis aponeurosis,**  
 824

**Occult blood in stools, 596**

**Oculomotor nerve, 906**

**Odontome, 94, 323**

**Oedema,**  
 of brain, 842  
 of glottis, 358, 360, 449

**Oesophagectasia, 439**

**Oesophagoscope, 424**

**Oesophagus, 439**  
 achalasia of, 439  
 anatomy of, 423  
 carcinoma of, 442  
 diverticulum of, 440  
 examination of, 225, 424  
 foreign bodies in, 425  
 growths of, 442  
 rupture of, 440  
 shortening of, 441  
 stricture, 440

**Olecranon, fracture of, 958**

**Olecranon bursa, 1174**

**Olfactory groove meningioma,**  
 859

**Olfactory nerve, 906**

**Oligodendroglioma, 98, 362**

**Oliguria, 716**

**Omentopexy, 693**

**Omentum, great,**  
 anatomy, 545  
 cysts of, 541  
 growths of, 540  
 torsion of, 540, 565

**Onychogryphosis, 244**

**Open pneumothorax, 466**

**Operating theatre, 182**

**Operation table palsy, 894**

**Operative treatment of fractures, 930**

**Ophthalmia, gonococcal, 60**

**Optic chiasma, glioma of, 859**

**Optic disc, 906**  
 papilloedema of, 906

**Optic nerve, 906**

**Orbital cellulitis, 27**

**Orchidopexy, 794**

**Orchitis,**  
 acute, 797  
 syphilitic, 800

**Os calcis, fractures of, 1004**

**Osgood-Schlatter's disease, 1044**

**Osteitis deformans, 1038**  
 fibrosa, 1040  
   generalised, 1041  
   solitary cyst in, 1040  
   spontaneous fracture, 1041  
 syphilitic, diffuse, 1028  
 tuberculous, 1026  
 typhoid of spine, 886, 1025

**Osteo-arthritis, 1102**  
 of ankle, 1107  
 of hip, 1104  
 of knee, 1106  
 of shoulder, 1107  
 of spine, 886

**Osteo-arthropathy, pulmonary,**  
 1045

**Osteochondritis, 1042**  
 Keinboch's, 1045  
 Kohler's, 1044  
 of os calcis, 1145  
 of spine, 887, 1045  
 pseudocoaxalgia of Perthes,  
 1042  
 Schlatter's, 1044

**Osteochondritis dessicans, 934,**  
 1108

**Osteochondritis, syphilitic, 1031**

**Osteoclastoma, 95, 1049**  
 of gums, 90, 321  
 of jaws, 96, 321  
 of long bone, 1049

**Osteogenesis imperfecta, 1041**

**Osteogenesis in laparotomy**  
 scars, 538

**Osteogenic sarcoma, 1052**

**Osteolytic sarcoma, 1052**

**Osteoma, 98, 1048**  
 cancellous, 93, 1048  
   single, 93, 1048  
   multiple, 93, 1048  
 ivory, 93, 1049  
 of external auditory meatus,  
 385  
 of jaws, 314  
 of skull, 837  
 of spine, 888

**Osteomalacia, 1085**

**Osteomyelitis, acute, 1018**  
 cloacæ in, 1017  
 involucrum in, 1017  
 necrosis in, 1016

**Osteomyelitis, acute—continued**

- of skull, 836
- Pott's puffy tumour in, 836
- of spine, 886
- pathology, 1016, 1018
- sequestrum, formation in, 1016
- symptoms, 1019
- traumatic, 1022
- treatment, 1020

**Osteomyelitis, chronic, 1022**

- Brodie's abscess, 1023

**Osteoperiostitis, chronic, of skull, 836****Osteophytes, 1103****Osteoporosis, senile, 1124****Osteosarcoma, 101, 1052****Osteoscopic pains, 1028****Osteotomy,**

- for coxa vara, 1133
- for genu valgum, 1135
- for genu varum, 1135
- for tuberculous hip, 1091
- in V, Lorenz' operation, 1130

**Otitis urethrotome, 787****Otitis externa, 385****Otitis media, acute, 387**

- complications of, 392
- pyæmia, 392

**Otitis media, chronic, 389**

- syphilitic, 73

**Otogenic encephalitis, 394**

- intracranial abscess, 394

**Otosclerosis, 386****Ovary,**

- carcinoma, 819
- cysts, 817
- dermoid cysts, 111, 818
- endometrioma, 818
- fibroma, 819
- pseudomucinous cyst, 817
- rupture of cyst, 819
- serous papillary cyst, 817
- suppuration in cyst, 819
- torsion of, 819

**Oxalate calculi, 738****Oxaluria, 742****Oxycephaly, 836****Oxygen inhalation, 135, 161****P****Pachydermatocœle, 823****Pachydermia of larynx, 450****Paquelin cautery, 217****Paget, recurrent fibroid of, 90, 357****Paget's bodies, 507****Paget's disease,**

- of bone, 1038
- of nipple, 507

**Pain, relief of, 186****Painful heel, 1144**

- due to disease, 1145
- due to static causes, 1145
- due to trauma, 1144

**Painful spurs, 1144****Palate,**

- abscess, 335

**Palate—continued**

- cleft, 330
- development, 327
- growths, 335
- perforation, 335
- ulceration, 335

**Palmar fascia, contracture of, 1168****Palmar ganglion, compound, 1171****Pancreas,**

- anatomy, 706
- calculi, 710
- carcinoma, 710
- cysts, 709
- examination, 706
- injuries, 706
- pancreatitis, acute, 707
- chronic, 709

**Pancreatic calculi, 710**

- cysts, 709

**Pancreatitis, acute, 707**

- catarrhal, 708
- fulminating, 707
- subacute, 708

**Pancreatitis, chronic, 709**

- jaundice in, 709

**Panus, 1066, 1072****Papilloma, 103**

- intracystic, 104
- of bladder, 767
- of breast, 515
- of face, 303
- of gums, 321
- of larynx, 456
- of lip, 307
- of mucous membrane, 103
- of penis, 60, 783
- of renal pelvis, 749
- of skin, 103, 238
- of tongue, 342
- of vulva, 63, 811

**Paracœcal hernia, 563****Paracæsis Willian, 386****Paraduodenal hernia, 563****Paraldehyde, 191****Paralysis of larynx. See Vocal Cords****Paralysis of pharynx, 434****Paralytic ileus, 642****Parametritis, 815****Paraphimosis, 781****Paraplegia,**

- in extension, 877
- in flexion, 877
- Thorburn's gravitation, 878

**Parasitic cysts, 115**

- of kidney, 745

**Parasitic myositis, 1165****Parasympathetic system, 911****Paratyphoid, 618****Parenchymatous goitre, 367****Paronychia, 249****Parotid tumour, 350****Parotitis,**

- acute suppurative, 348
- epidemic, 348
- simple, 348

**Paroxysmal hæmoglobinuria, 717**

- rhinorrhœa, 407

**Parrott's nodes, 73, 1030****Partial division of a nerve, 894****Passavant's cushion, 332****Pasteur's vaccine, 18****Patella,**

- dislocation of, 991
- habitual, 991, 1136
- fracture of, 987
- premature ossification of, 1082

**"Patellar tap," 1062****Patent urachus, 754****Pathetic nerve, 906****Pathological dislocation, 934, 1065****Pathological fractures, 916****Paul-Bunnell test, 45****Paul's operation, 630****"Peau d'orange," 521****Pedicle skin graft, 170****Pelvic abscess, 555****Pelvic appendicitis, 677****Pelvic girdle,**

- fractures of, 973
- injury to bladder and urethra in, 974

**Pelvic peritonitis, 553****Pelviorectal abscess, 652**

- cellulitis, 27, 652, 815

**Pelvis, fractures of, 973****Penetrating wounds, 120****Penis,**

- anatomy, 778
- anomalies, 780
- balanoposthitis, 782
- carcinoma, 783
- cavernositis, 783
- dislocation of, 782
- epispadias, 780
- hypospadias, 780
- injuries, 782
- paraphimosis, 781
- phimosis, 781
- preputial calculi, 782

**Pentothal, 193****Peptic ulceration,**

- etiology of, 597
- pathology of, 598

**Perforating ulcers, 71, 243****Perforating wounds, 122****Perforation of duodenal ulcer, 609**

- of gastric ulcer, 602
- of nasal septum, 405
- of palate, 335
- of stercoral ulcer, 642
- of typhoid ulcer, 619

**Perianal abscess, 653****Pericanalicular fibro-adenoma of breast, 516****Pericranium, 824****Perineal hernia, 580****Perinephric abscess, 730****Perinephritis, 730****Periosteal node, 1028****Periosteal sarcoma, 1052**

- Periosteum**, 1014  
**Periostitis**,  
 syphilitic, 1028  
 tuberculous, 1025  
**Perirenal capsules**, 714  
**Peritoneum**, anatomy, 544  
 foramen of Winslow, 544  
 great omentum, 545  
 greater and lesser sacs, 544  
 mesentery and mesocolon, 544  
 watersheds, 545  
**Peritonism**, 602  
**Peritonitis**, 546  
 gonococcal, 552  
 pelvic, 553  
 pneumococcal, 551  
 streptococcal, 553  
 tuberculous, 556  
**Peritonitis, acute**, 546  
 after treatment, 551  
 diffuse, 549  
 drainage, 551  
 due to appendicitis, 679  
 effect upon intestine, 547  
 localised, 548  
 peritoneal toilet, 551  
 toxic effects of, 547  
 treatment, 550  
**Peritonitis, chronic**,  
 generalised, 556  
 localised, 556  
 simple, 556  
 syphilitic, 558  
 tuberculous, 556  
**Peritonsillar abscess**, 429  
**Periurethral abscess**, 58, 788  
**Permeation**, 87  
**Persistent cervical sinus**, 353  
**Perthes' disease**, 1042  
**Pes cavus**, 1143  
 Naughton Dunn's operation, 1143  
 Steindler's operation, 1143  
**Pes Equinus in spastic paralysis**, 1153  
**Pes Planus**, 1139  
 treatment, 1141  
 arch supports in, 1141  
 manipulation in, 1142  
 operative, 1142  
 varieties, 1141  
**Petechiae**, 142  
**Petit's triangle**, 1092  
**Petrisage**, 215  
**pH of urine**, 729  
**Phagadema**, 177  
**Phalanges of fingers**,  
 dislocation, 971  
 fractures, 969  
**Phalanges of toes**,  
 fractures, 1006  
**Pharyngitis**,  
 acute, 426  
 chronic, 430  
**Pharyngotomy**, lateral, 461  
**Pharynx**,  
 anatomy, 423  
 examination of, 424  
**Pharynx—continued**  
 foreign bodies, 424  
 growths, 436  
 inflammation, acute, 426  
 chronic, 430  
 lupus, 433  
 nervous affections, 434  
 pouch or diverticulum, 435  
 syphilis, 433  
 tuberculosis, 433  
**Phimosis**, 781  
**Phlebitis**, 281  
**Phleboliths**, 277  
**Phlegmasia alba dolens**, 282  
**Phosphatic calculi**, 739  
**Phrenic nerve**, 895  
 avulsion of, 500  
 phrenicectomy, 500  
**Physiological hyperplasia of thyroid gland**, 366  
**Physiotherapy**, 213  
**Picric acid**, 181  
**"Pied Forcé,"** 1006  
**Pigeon chest**, 1033  
**Pigmented mole**, 241  
**Pile**, sentinel, 650  
**Piles**. See Hemorrhoids  
**Pilonidal sinus**, 875  
**Pineal tumours**, 859  
**Pituitary tumours**, 857  
**Plague**, 44  
**Plantaris tendon**, rupture of, 1163  
**Plasma-celled sarcoma**, 1056  
**Plasma cells**, 4, 10  
**Plasma transfusion**, 151  
 indications for, 151  
**Plaster of Paris**, 924  
**Plexiform angioma**, 287  
 neuroma, 90, 895  
**Plummer - Vinson syndrome**, 430, 913  
**Pneumatocele**, 362  
**Pneumaturia**, 717  
**Pneumococcal arthritis**, 1069  
**Pneumococcal peritonitis**, 551  
**Pneumococcus**, 48  
**Pneumectomy**, 493  
**Pneumothorax**, 463, 1012  
 artificial, 481, 499  
 extrapleural, 500  
**"Poker-back spine" in spondylitis deformans**, 887  
**Policeman's heel**, 1144  
**Politzer's bag**, 382  
 method, 382  
**Polycystic disease of kidneys**, 743  
 of liver, 693  
 of pancreas, 709  
**Polymasia**, 505  
**Polymorphic sarcoma**, 101  
**Polyorchism**, 793  
**Polypi of colon**, 626  
 of nose, 412  
 of rectum, 661  
 uterus, 821  
**Polythelia**, 506  
**Polyuria**, 716  
**Pond fractures**, 829  
**Popliteal nerve**,  
 external, 905  
 internal, 905  
**Popliteus bursa**, 1175  
**Position of election for ankylosis**, 213  
**Position of function**, 213  
 in treatment, 213  
**Posterior tibial nerve**, 905  
**Post-anal dimple**, 875  
**Post-operative treatment**, 186, 208  
**Postural drainage**, 481, 487  
**Potassium-permanganate**, 57, 62  
**Potato tumour**, 362  
**Potter-Bucky diaphragm**, 222  
**Pott's disease of spine**, 1087  
**Pott's fracture**, 1000, 1107  
 puffy tumour, 836  
**Pouch of Douglas**, 545  
**Pouch**, pharyngeal, 435  
**Pregnyl**, 794  
**Premammary abscess**, 509  
**Pre-operative ward**, 161  
**Preparation before operation**, 185  
**Prepatellar bursa**, 1175  
**Preputial calculi**, 782  
**Pre-sacral neurectomy**, 759  
**Preservation of function after fracture**, 929  
**Pressure gangrene**, 175  
**Pressure on nerves**, 894  
**Pressure ulcers**, 166  
**Prevention of deformity**, 213, 1113  
**Priapism**, 879  
**Primary hæmorrhage**, 153  
**Primary peritonitis of children**, 551  
**Primary renal hypertension**, 725  
**Primary suture of nerve**, 892  
**Proctitis**, 648  
 gonococcal, 60, 63  
**Proctocaine**, 651  
**Proctoscope**, Kelly's, 647  
**Proctotomy**, 652  
**Progressive bulbar paralysis**, 434  
**Prolan in urine**, 802  
**Prolapse of rectum**, 648  
**Prophylaxis of hand infections**, 247  
**Prostate**,  
 anatomy, 770  
 calculus, 771  
 carcinoma, 776  
 fibrosis, 776  
 inflammation, acute, 58, 771  
 chronic, 59, 771  
 tuberculous, 771  
 sarcoma, 777  
 senile enlargement of, 772  
 treatment of enlarged,  
 Frey's operation, 775  
 Harris's operation, 775  
 transurethral resection, 775  
 Young's median bar, 776



**Prostatitis**,  
acute, 58, 771  
chronic, 59, 771  
tuberculous, 771  
**Prostigmata**, 644  
**Protopathic sensation**, 889  
**Pruritus ani**, 660  
vulvæ, 810  
**Psoas abscess**, 1092  
bursa, 1175  
**Psoriasis linguae**, 338  
**Psychogenic shock**, 155  
"Pulled elbow," 956  
**Pulmonary abscess**, 477  
causation of, 478  
postural drainage, 481  
radiological appearances, 481  
**Pulmonary complications after anaesthesia**, 209  
**Pulmonary embolism**, 187, 209  
**Pulmonary gangrene**, 482  
**Pulmonary growths**, 490  
adenoma of bronchus, 490  
carcinoma, 491  
endothelioma, 494  
sarcoma, 494  
**Pulmonary osteo-arthritis**, 1045  
**Pulmonary tuberculosis**, 497  
acute pneumonic, 498  
artificial pneumothorax in, 499  
caseous, 498  
extrapleural pneumothorax n, 500  
fibrocavernous, 498  
fibroid, 498  
phrenic avulsion, 500  
thoracoplasty, 501  
**Pulsating exophthalmos**, 275  
haematoma, 260, 825  
**Pulsation in bone tumours**, 973  
**Pupils**, changes in head injuries, 845  
**Purpura hæmorrhagica**, 713  
**Pus**, 3  
**Pyæmia**, 17, 30  
**Pyæmic arthritis**, 1069  
**Pyelitis**, acute, 728  
accessory factors in, 727  
etiology, 726  
fulminating, 728  
ketogenic diet, 728  
mandelic acid therapy, 728  
of children, 728  
of pregnancy, 728  
subacute, 728  
**Pyelitis**, chronic, 731  
**Pyelography**, 237, 717  
in hydronephrosis, 724  
instrumental, 227, 717  
**Pyelography**, intravenous. See Urography

**Pyelography**, retrograde. See Pyelography, Instrumental  
**Pyelonephritis**, 729  
**Pyelotomy**, 741  
**Pylephlebitis**, acute suppurative, 30, 687  
**Pylorus**,  
infantile hypertrophic stenosis of, 590  
Rammstedt's operation, 591  
stenosis of, 606, 610  
**Pyogenic membrane**, 3  
**Pyonephrosis**, 729  
**Pyorrhoea**, 320  
**Pyosalpinx**, tuberculous, 815  
**Pyuria**, 717

## Q

**Quadriceps drill**, 986, 990  
**Quadriceps muscle**, rupture of, 1163  
**Queckenstedt's test**, 878  
**Quinidine**, 374  
**Quinsy**, 129

## R

**Rabies**, 44  
**Radial bursa**, 245, 250  
**Radiant heat bath**, 216, 257  
cradle, 158, 160  
**Radical mastoid operation**, 397  
**Radicular odontome**, 95, 325  
**Radium**, 230  
bomb, 230  
burns, 141  
**Radium therapy**, 230  
in carcinoma of breast, 524  
lung, 494  
oesophagus, 443  
pharynx, 438  
rectum, 664  
thyroid, 377  
tongue, 344  
vulva, 812  
in growths of carotid body, 362  
nasal sinuses, 420  
testis, 802  
**Radius**,  
congenital absence of, 1115  
fractures of, 959  
head, 959  
lower end, 963  
shaft, 960  
**Radius and ulna**, fractures of, 961  
**Railway spine**, 883  
**Ranula**, 349  
sublingual, 361  
**Rarefaction of bone**, 1018  
**Rat-tail appearance**, 492  
**Ray fungus**, 49  
**Raynaud's disease**, 175, 911  
"Razor back," 1119  
**Reaction of degeneration**, 890

**Reactionary hæmorrhage**, 153  
**Reception unit**, 159  
**Recto-urogenital fistula**, 647  
**Rectum**, 652  
absence, 647  
adenoma, 661  
anatomy, 646  
bilharzia, 661  
carcinoma, 662  
dysentery in, 650  
examination, 646  
fissure-in-ano, 649  
fistula-in-ano, 655  
fistula into other organs, 656  
growths, 660  
hæmorrhoids, 657  
inflammation, 649  
injuries, 648  
ischio-rectal abscess, 653  
pelvirectal abscess, 652  
perirectal abscess, 653  
prolapse, 648  
pruritis ani, 660  
sinus-in-ano, 655  
stricture, 651  
syphilis, 651  
tuberculosis, 650  
ulceration, 650  
**Rectus abdominis muscle**,  
rupture of, 1163  
**Recurrent laryngeal nerve**, 453, 909  
**Red glazed tongue**, 337  
**Red thrombus**, 277  
**Reduction "en masse,"** 568  
**Reduction of fractures**, 922  
anaesthesia in, 922  
by gradual traction, 923  
by manipulation, 922  
by open operation, 923  
**Referred pain**, renal, 716  
**Regeneration of nerve**, 890  
**Regional ileitis**, 621  
**Renal calculi**, 736  
complications, 742  
composition, 738  
irreversible colloids in, 736  
stone formation in urinary tract, 736  
symptoms, 738  
treatment,  
bilateral, 741  
unilateral, 741  
**Renal colic**, 716  
**Renal efficiency**, 717  
tests of excretion, 718  
of retention, 718  
urea clearance test, 718  
**Renal growths**. See Kidney  
**Renal pain**  
colic, 716  
in opposite kidney, 716  
local renal, 716  
referred, 716  
**Renal pelvis**,  
anatomy, 714  
growths, 749  
**Renal rickets**, 1035  
**Renal sympathectomy**, 814



**Renal tuberculosis, 733**  
 after-treatment following operation, 736  
 cystoscopy in, 735  
 frequency of micturition, 734  
 intravenous urography in, 735  
 involvement of ureter, 734  
 localisation of, 735  
 pathology, 733  
  
 pyramidal origin, 733  
 treatment, 736  
 tubercle bacilli in urine, 734  
 twenty-four hours' specimen of urine, 734  
 ulcero-cavernous type, 733  
**Repair, process of, 1, 3**  
**Respiration, artificial, 197**  
**"Restor" heat-cage, 160**  
**Restoration of function, 10, 214**  
**Resuscitation unit, 160**  
**Retention cysts, 115**  
**Retention of urine, acute,**  
 due to obstruction, 758  
 due to spasm, 758  
 use of "Doryl" in, 758  
**Retention of urine, chronic, 758**  
 effects on urinary system, 760  
**Retinitis, 73**  
**Retinitis pigmentosa, 913**  
**Retrocaecal appendicitis, 677**  
**Retromammary abscess, 509**  
**Retroperitoneal abscess, point-**  
 ing at umbilicus, 536  
**Retroperitoneal cyst, 543**  
 hernia, 583  
 neoplasm, 543  
**Retropharyngeal abscess, 429,**  
 1091  
**Reverdin's skin graft, 169**  
**Reversed Colles's fracture, 965**  
**Rhabdomyoma, 97, 1167**  
**Rhabdomyosarcoma, 102, 346**  
**Rhesus factor, 149**  
**Rheumatoid arthritis, 1060**  
**Rhinitis,**  
 acute, 409  
 atrophic, 411  
 chronic hypertrophic, 410  
 spasmodic, 407  
**Rhinophyma, 242**  
**Ribs, fracture of, 1012**  
**Richter's hernia, 576**  
**Rickets, 1031**  
 adolescent, 1034  
 general changes, 1032  
 osseous changes, 1032  
 deformity, 1033  
 greenstick fracture, 1034  
 Harrison's sulcus, 1033  
 "Pigeon chest," 1033  
 "Ricketty rosary," 1033  
 renal, 1035  
 "Rider's bone," 94, 1166  
**Riedel's disease, 365**  
**Riedel's lobe, 685**  
**Rigg's disease, 320**  
**Rigidity, abdominal, 548, 673**

**Rinne's test, 382**  
**Rivini, notch of, 378**  
**Robert Jones's splints,**  
 abduction frame, 984, 1080  
 brace, 1096  
 cock-up, long and short, 900  
 for fractures of humerus, 925  
 shoulder abduction, 948  
**Rodent ulcer, 106, 239**  
 of face, 304  
 of lips, 307  
 of skin, 239  
**Rose's operation for hare-lip,**  
 302  
**Rotanda syringe, 150**  
**Rupia, 68**  
**Rupture. See Hernia**  
**Rupture of**  
 arteries, 260  
 bladder,  
 extraperitoneal, 760  
 intraperitoneal, 760  
 colon, 530  
 duodenum, 588  
 gangrenous appendix, 670  
 jejunum, 530  
 ileum, 530  
 liver, 531, 686  
 muscle, 1162  
 oesophagus, 440  
 ovarian cyst, 819  
 spleen, 532  
 stomach, 587  
 tendon, 1162  
 ulcer, duodenal, 609  
 gastric, 602  
 stercoral, 642  
 urethra, 785

## S

**Sabouraud technique, 229**  
**"Sabre tibia," 1028**  
**Saccular aneurysm, 266**  
**Sach's-Georgi test, 75**  
**Sacralisation of lumbar spine,**  
 887, 1156  
**Sacrocccygeal tumour, 112,**  
 875  
**Sacro-iliac joint,**  
 osteo-arthritis of, 887  
 tuberculosis of, 1087  
**Sacro-iliac strain, 1156**  
**Sacrum, fractures of, 975**  
**Saint Anthony's fire, 47**  
**Saline infusions, 29, 145**  
 intramuscular, 145  
 intravenous, 29, 145  
 rectal, 29, 145  
 subcutaneous, 29, 145  
**Salivary calculi, 349**  
**Salivary fistula, 348**  
**Salivary gland adenoma, 350**  
**Salivary glands, 347**  
 growths of, 350  
 inflammation of, 348  
 injury to, 347  
 stones in, 349  
**Salpingitis, acute, 814**  
**Salpingo-oöphoritis, 815**  
**Salvarsan, 77**  
**Sampson Handley's theory of**  
 ileus duplex, 682  
 Paget's disease of nipple, 507  
**Santonin, 51**  
**Saphenous varix, 284**  
**Sapraemia, 29**  
**Sarcoma, 86**  
 classification of, 99  
 of bladder, 770  
 of breast, 526  
 of jaws, 315  
 of kidney, 747  
 of liver, 692  
 of lung, 494  
 of pancreas, 710  
 of pharynx, 436  
 of prostate, 777  
 of rectum, 665  
 of spine, 888  
 of stomach, 597  
 of testis, 801  
 of tongue, 346  
 varieties of, 98  
**Sarcoma of bone,**  
 chondrosarcoma, 1054  
 Ewing's, 1055  
 osteogenic, 1052  
 osteolytic, 1052  
 osteosarcoma, 1052  
 periosteal, 1052  
 plasma cell, 1056  
 pulsating, 1053  
 traumatic origin of, 1052  
 X-ray treatment of, 1055  
**Saturday-night palsy, 894**  
**Scalds, 129**  
**Scalene syndrome, 356**  
**Scalp,**  
 anatomy of, 824  
 blood supply of, 824  
 cellulitis of, 827  
 cephalhaematocoe of, 828  
 cephalhaematoma of, 825  
 circoid aneurysm of, 827  
 cysts of, 827  
 erysipelas, 827  
 growths of, 828  
 infections of, 826  
 injury to, 825  
 avulsion, 826  
 burns, 826  
 hematoma, 825  
 wounds, 825  
 lymphatics of, 824  
 occipito-frontalis aponeur-  
 osis, 824  
**Scaphoid,**  
 carpal, fractures of, 966  
 tarsal, Kohler's disease of,  
 1044  
**Scapula, fractures of, 941**  
 acromion, 942  
 body, 941  
 coracoid, 942  
 glenoid, 942  
 neck, 942  
**Scar, 4, 11**

- Scarpa, fascia of**, 789  
**Scheuermann's disease**, 887, 1045  
**Schick test**, 10  
**Schimmelbusch's mask**, 199  
**Schistosomum hæmatobium**, 52  
**Schlatter's disease**, 1044  
**Schliephacke's apparatus**, 219  
**Schwabach's test**, 382  
**Schwartze's operation**, 396  
**Sciatic hernia**, 579  
**Sciatic nerve**. See **Great Sciatic Nerve**  
**Sciatica**, 904  
**Scirrhous carcinoma**, 108, 519  
**Sclavo's serum**, 42  
**Sclerosis of bone**, 1018  
**Scolex of hydatid**, 115  
**Scoliosis**, 1118  
    functional, 1118  
    structural, 1119  
        changes in thorax, 1119  
        in vertebræ, 1119  
        "razor-back" in, 1119  
    treatment of, 1120  
        exercises, 1121  
        jackets, 1121  
**Scoliosis, operative treatment of**, 1122  
**Scott's dressing**, 11  
**Scrotal hæmatoma**, 807  
**Scrotum, diseases of**, 808  
**Scurvy**, 1036  
**Sebaceous cyst**, 242  
    of face, 305  
    of scalp, 827  
**Sebaceous glands**, 242  
**Secondary hæmorrhage**, 153  
**Secondary malignant disease of bone**, 1056  
**Secondary suture**  
    of nerves, 893  
    of wounds, 128  
**Secondary thyrotoxicosis**, 375  
**Sella turcica, alteration in**, 858  
**Semb's operation**, 502  
**Semilunar bone**,  
    dislocation of, 968  
    fracture of, 967  
**Semilunar cartilages, injuries of**, 992  
**Seminomalous bursa**, 1175  
**Seminal vesicles**, 777  
    inflammation of, 59  
    tuberculosis of, 798  
**Seminoma**, 801  
**Semon's law**, 453  
**Semile gangrene**, 174  
**"Sentinel pile"**, 850  
**Separation of dead tissue**, 172  
**Separation of epiphysis**, 918  
    of femur, lower end, 987  
        upper end, 978  
    of humerus, lower end, 952  
        upper end, 948  
    of radius, lower end, 965  
    of tibia, lower end, 1003  
        upper end, 996  
**Sepsis, latent**, 893  
**Septicæmia**, 17, 28  
**Sequestration dermoid**, 114  
**Sequestrum formation**, 1016  
**Serous cyst**, 115, 119  
**Serous effusion into joints**, 1061  
**Serum**, 19  
    antibacterial, 19  
    antitoxic, 19  
**Serum sickness**, 20  
**"Setting" of fractures**, 922  
**Shiga's bacillus**, 46, 818  
**Shipway apparatus**, 202  
**Shock, traumatic**, 155  
    primary, 155  
        clinical picture, 156  
        neurogenic, 155  
        psychogenic, 155  
        treatment, 156  
    secondary, 156  
        blood pressure in, 157  
        clinical picture, 157  
        pathology, 156  
        prevention, 158  
        treatment, 158  
        varieties of, 155  
**Short-wave therapy**, 219  
**Shortening of œsophagus**, 440  
**Shoulder joint**,  
    contusions of, 946  
    dislocations of, 942  
        complications of, 945  
        Kocher's reduction, 944  
        recurrent, 946  
        reduction by extension, 945  
        unreduced, 945  
        varieties of, 942  
    effusion into, 1062  
    position for ankylosis, 1063  
    tuberculosis of, 1085  
        caries sicca, 1085  
**Shrapnell's membrane**, 378  
**Sialo-adenitis**, 348  
**Siegle's speculum**, 380  
**Sigma test**, 75  
**Sigmoidoscope**, 647  
**Signs of anæsthesia**, 194  
**Silk thread**, 184  
**Silk-worm gut**, 184  
**Simple erythema**, 131  
**Singer's node**, 449  
**Single cyst of kidney**, 745  
**Sinus**, 24  
**Sinus formation in tuberculous arthritis**, 1072  
**Sinus-in-ano**, 655  
**Sinus pericranii**, 828  
**Sinusoidal current**, 218  
**Skeletal traction**, 924  
**Skin**,  
    coccal infection of, 231  
    cysts of, 237  
    growths of, 237  
    neuropathic affections of, 243  
    pre-operative preparation of, 185  
    tuberculosis of, 234  
    vascular affections of, 243  
**Skin grafting**, 128, 168  
    pedicle graft, 170  
    Reverdin's method, 169  
    Thiersch's method, 169  
    Wolfe's graft, 170  
**Skin lesions in syphilis**, 67, 69  
**Skull**,  
    acute osteomyelitis, 836  
    anomalies of, 835  
        aplasia cranii, 835  
        cephaloceles, 835  
        macrocephaly, 835  
        microcephaly, 835  
        oxycephaly, 836  
    chronic osteoperiostitis, 836  
    fractures of base, 831  
        anterior fossa, 832  
        Aran's theory of, 831  
        black-eye in, 832  
        by bursting, 832  
        by radiation, 831  
        compound, 832  
        deafness in, 832  
        escape of cerebrospinal fluid, 832  
        external hæmorrhage in, 832  
        facial palsy in, 832  
        involvement of cranial nerves, 833  
        middle fossa, 833  
        posterior fossa, 834  
        prognosis of, 834  
        treatment of, 834  
    fractures of vault, 828  
        compound, 830  
        depressed, 829  
        fissured, 828  
        " gutter," 829  
        intracranial complications in, 830  
        " pond," 829  
        punctured, 829  
        traumatic cephalhydrocele in, 831  
        treatment of, 830  
    growths of, 837  
        hæmangioma, 837  
        osteoma, 837  
        sarcoma, 837  
        secondary carcinoma, 837  
        "Pott's puffy tumour" in, 836  
    syphilis of, 837  
    tuberculosis, 837  
**Slough**, 3  
**Small round-celled infiltration**, 10  
**Small round-celled sarcoma**, 100  
**Smith-Peterson pin**, 961  
**Smith's fracture**, 965  
**Smoker's patch**, 339  
**Snake bites**, 121  
**Snapping hip**, 1134  
**Sodium tetraiodophenolphthalein**, 228, 694  
**Soft sore**, 80  
**Softening of brain, delayed**, 842, 848

**Souttar's operation**, 1149  
**Spa treatment**, 1101  
**Spastic paralysis**, 1152  
**"Spät-apoplexie" of Bollinger**, 842, 848  
**Speculum**, aural, 380  
**Spermatic cord**, 803  
**Spermatocele**, 804  
**Spermatocytoma**, 801  
**Spermatozoa**, count of, 813  
**Sphenoidal air sinus**,  
     acute infection of, 414  
     chronic infection of, 418  
     treatment of, 418  
**Spheroidal-celled carcinoma**, 106, 518  
**Spina bifida**, 872  
     varieties of, 872  
**Spina bifida occulta**, 872  
**Spinal accessory nerve**, 910  
**Spinal cord**,  
     anatomy, 871  
     complete transverse lesions of, 879  
     compression of, 876  
     concussion of, 875  
     gravitation hemiplegia, 878  
     hematomyelia, 878  
     hematorrhachis, 878  
     hemorrhage in, 878  
     hemisection of, 883  
     Horner's syndrome, 879  
     incomplete lesions of, 882  
     injuries to, 875  
     laminectomy in, 883  
     meningitis, 884  
     transverse myelitis, 884  
     traumatic neurasthenia, 883  
     tumours of, 885  
**Spinal nerves**,  
     lesions of, 895  
     pressure on, 894  
**Spindle-celled sarcoma**, 100  
**Spine**,  
     anatomy of, 871  
     anomalies of, 872  
     coccydynia, 888  
     deformities of, 1118  
     dislocations of, 1010  
     epiphysitis of, 887  
     fracture of, 1007  
     fracture-dislocation of, 1009  
     growths, 888  
     injuries to, 1007  
     meningocele, 873  
     meningomyelocele, 873  
     myelocele, 873  
     osteo-arthritis, 886  
     osteomyelitis, acute, 886  
     prolapse of nucleus pulposus, 888  
     rheumatoid arthritis of, 886  
     sacralisation of lumbar spine, 887  
     spina bifida, 872  
     occulta, 872  
     spondylitis deformans, 887  
     syphilis of, 886  
     syringomyelocele, 873

**Spine—continued**  
     tuberculosis of, 1087  
     tumours of sacro-coccygeal region, 875  
     typhoid osteitis, 886  
**Spine, tuberculosis of, 1087**  
     abscess formation in, 1089, 1091  
     deformity in, 1089  
     diagnosis of, 1094  
     nevous lesions in, 1092  
     pathology of, 1087  
     prognosis of, 1094  
     symptoms of, 1090  
     treatment of, 1095  
         of abscess, 1097  
         of paraplegia, 1097  
         operative, 1096  
     X-ray appearances in, 1093  
**Spinous processes of**, fractures of, 1011  
**Spirochaeta pallidum**, 64  
**Spleen**,  
     anatomy, 711  
     anomalies of, 711  
     growths of, 712  
     infections of, 712  
     injury to, 712  
     movable, 711  
     penetrating wounds of, 534  
     rupture of, 532  
     surgical splenomegalies, 712  
**Splenic anaemia**, 712  
**Splenic fever**, 42  
**Splenomegaly**, surgical, 712  
**Splints**, 923  
     Bohler-Braun, 984  
     Cramer's wire, 924  
     gutter, 923  
     Hodgen's, 981  
     plaster of Paris, 924  
     Robert Jones' humerus, 925  
     shoulder abduction, 925  
     hip abduction, 984  
     Thomas' arm, 925  
     knee, 925, 981, 983  
     traction, 925  
**Spondylitis ankylopoietica**, 1158  
**Spondylitis deformans**, 887, 1102, 1158  
     Marie-Strumpell type, 887  
     "Poker-back" spine in, 887  
**Spondylitis heredo-traumatica**, 887  
**Spondylitis rhizomélisque**, 887  
**Spondylolisthesis**, 1155  
**Sporioblastoma multiforme**, 98, 862  
**Spongy gums**, 320  
**Spontaneous fractures**, 916  
**Sporotrichosis**, 50  
**Sprains of joints**, 933  
     ankle, 1004  
     knee, 989  
     wrist, 967  
**Spreading gangrene**, 177, 682  
**Spreading ulcer**, 167  
**Sprengel's shoulder**, 1114  
**Spurious diarrhoea**, 629, 642, 652

**Spurs**, painful, of feet, 1144  
**Squamous-celled carcinoma**, 95  
     of face, 304  
     of larynx, 455  
     of lip, 307  
     of skin, 239  
     of tongue, 342  
**Stannard envelope**, 138  
**Staphylococcus**, 22  
**Stasis**, intestinal, 616  
**Steindler's operation**, 1143  
**Steinmann's pin apparatus**, 927  
**Stellwag's sign**, 376  
**Stercoliths**, 667  
**Sterilisation of**  
     dressings, 184  
     drums, 129  
     instruments, 184  
     ligature materials, 184  
     lotions, 129  
     rubber gloves, 184  
     ward utensils, 129  
**Sterility**, 813  
**Sternomastoid muscle**, rupture of, 1163  
**Sternum**, fractures, 1011  
**Stereognosis**, 889  
**"Stiffneck"**, 1165  
**Still's disease**, 1101  
**Stoerk's theory**, 109, 746  
**Stoffel's operation**, 1153  
**Stomach**,  
     acute ulcer, 598  
     anatomy, 584  
     carcinoma, 594  
     chronic progressive ulcer, 599, 600  
     complications of ulcer, 602  
     dilatation, acute, 591  
     chronic, 593  
     examination, 225, 586  
     fistula, 589  
     foreign bodies, 588  
     growths, 597  
     infantile pyloric stenosis, 590  
     inflammations, 593  
     injuries, 587  
     lymphatic drainage, 585  
     penetrating wounds, 534, 587  
     peptic ulceration, 597  
     rupture, 587  
     tetany in, 593  
     uncomplicated ulcer, 600  
**Stomatitis**, 329  
**Stored blood**, 151  
**"Stove-in" chest**, 464  
**Strangulated hernia**, 565, 632, 635  
**Strangulation of intestine**, 632  
**"Strawberry" gall-bladder**, 700  
**Streptococcal peritonitis**, 553  
**Streptococcus**, 22  
**Streptothrix actinomyces**, 48  
**Stricture of intestine**, 625  
     of œsophagus, 440  
     of rectum, 651  
     of ureter, 723  
     of urethra, 58, 786

- Stroma of new growths, 84**  
**Student's elbow, 1174**  
**Subclavicular dislocation of shoulder, 942**  
**Subcoracoid dislocation of shoulder, 942**  
**Subdeltoid bursa, 1174**  
**Subdural hæmorrhage, 848**  
     chronic type, 848  
**Subdural hæmorrhagic cyst, 848**  
**Subglenoid dislocation of shoulder, 942**  
**Sublingual ranula, 361**  
**Subluxation of head of radius, 956**  
**Submucous abscess of rectum, 647**  
**Subperiosteal hæmatoma, 916**  
**Subphrenic abscess, 553**  
     Barnard's classification of, 553  
**Subspinous dislocation of shoulder, 942**  
**Subungual exostosis, 94, 1048**  
**Sulphur yellow granules, 49**  
**Superficial burns, 131**  
**Superior laryngeal nerve, 909**  
**Superior longitudinal sinus thrombosis, 852**  
**Suppuration, 2, 5, 22**  
**Suppurative pleurisy, See Em-pyema**  
**Supracondylar fracture of humerus, 951**  
**Suprascapular nerve, 899**  
**Suprasellar cysts, congenital, 857**  
**Suprasellar meningioma, 857, 864**  
**Surface traction, 924**  
**Surgical emphysema, 463**  
**Surgical neck of humerus, fractures of, 947**  
**Surgical spirit, 181**  
**Surgical technique, 180**  
**Suture,**  
     primary, of nerve, 892  
     secondary, of nerve, 893  
**Sutures, varieties of, 125**  
**Swabs, 184**  
**Sylvester's method of artificial respiration, 197**  
**Syme's operation, 788**  
**Sympathetic nervous system, 910**  
**Symphysis pubis, diastasis of, 973**  
**Syncoytium, 113**  
**Synorchism, 793**  
**Synovial fringe, nipping of, 991**  
**Synovitis, 1064**  
     syphilitic, 1098  
     traumatic, 1064  
**Syphilis, 64**  
     aneurysm in, 71  
     bismuth metal in treatment of, 77  
     Charcot's disease, 71  
     clinical manifestations, 66  
**Syphilis—continued**  
     condylomata, 68, 72  
     congenital, 71  
     diagnosis, 74  
     diffuse infiltration, 70  
     extragenital infection, 65  
     immunity in, 66  
     iritis, 69  
     Kahn test, 75  
     lymph glands in, 69, 290  
     malignant, 68  
     marriage in, 80  
     mucous patches, 69  
     nodular cutaneous syphilide, 69  
     of larynx, 451  
     pathology, 64  
     perforating ulcer in, 71  
     primary lesions of, 66  
     prognosis of, 80  
     rupia, 68  
     sabre-tibia, 73  
     Sachs-Georgi test, 75  
     saddle-nose, 72  
     salvarsan, 77  
     secondary manifestations of, 67  
     Sigma test, 75  
     skin lesions in, 67  
     snail-track ulcers, 69  
     spirochæta pallidum, 64  
     tertiary manifestations of, 69  
     transmission of, 65  
     treatment of, 76  
     Wassermann reaction in, 75  
**Syphilis of bone, 1028**  
     congenital manifestations of, 1030  
     craniotabes, 1030  
     dactylitis, 1030  
     epiphysitis, 1031  
     gumma, 1029  
     osteoscopic pains in, 1028  
     periostitis, 1028  
     skull, 837  
     spine, 886  
     symmetrical overgrowth of tibia, 1030  
     varieties, 1028  
**Syphilis of**  
     breast, 513  
     bursa, 1174  
     ear, 391  
     joints, 1098  
         acquired, 1098  
         congenital, 1098  
         Clutton's knees, 1098  
     kidney, 732  
     larynx, 451  
     liver, 689  
     muscle, 1165  
     pharynx, 433  
     skull, 837  
     tendon sheaths, 1171  
     testis, 800  
     tongue, 339  
**Syphilitic arteritis, 264**  
     endarteritis, 264  
     lymphangitis, 294
- Syringe, aural, 384**  
**Syringomelia of joints, 1111**  
**Syringomyelocele, 873**
- T**
- Tabes dorsalis, juvenilis, 74**  
**Tabes mesenterica, 542**  
**Tabetic joints, 1110**  
**Tachycardia, 372**  
**Tænia echinococcus, 51, 115**  
     solum, 51  
**"Tailor's ankle," 1175**  
**Talipes equinovarus,**  
     congenital, 1137  
     clinical types, 1137  
     pathology, 1137  
     treatment, 1138  
**Talma-Morrison operation, 693**  
**Tannic acid in treatment of burns, 136**  
**Tapotement, 215**  
**Tapping of hydrocele, 806**  
**Tarsal joints, tuberculosis of, 1085**  
**Taxis, 568**  
**Taylor's spinal support, 1096**  
**T.C.P., 181**  
**Teeth,**  
     dental cyst, 322  
     development, 322  
     extraction, 326  
     infection, 322  
     in syphilis, 73  
     odontomes, 323  
     wisdom, 325  
**Teevan's urethrotome, 787**  
**Telangiectasis, 286**  
**Telescopic movement, 1127**  
**Teletherapy with radium bomb, 230**  
**Temporo-mandibular joint, 317**  
     dislocation, 317  
     infection, 319  
**Tenderness on rebound, 548**  
**Tendo achillis,**  
     bursa beneath, 1144, 1175  
     rupture of, 1164  
**Tendon sheaths,**  
     gouty affections of, 1171  
     infections of, 251  
     non-suppurative inflamma-tions of, 1169  
     of fingers, 245  
     of radial bursa, 245  
     of ulnar bursa, 245  
     suppurative inflammations of, 250, 1169  
     syphilis of, 1171  
**Tendon sheaths, tuberculosis of, 1169**  
     tumours of, 1171  
**Tendons,**  
     dislocations of, 1161  
     injury to, 1161  
     rupture, 1162  
     wounds, 1164

- Tenesmus**, 651, 663  
**Tennis elbow**, 956  
 leg, 1163  
**Tenosynovitis**,  
 acute, 250, 1169  
 chronic, 1169  
 suppurative, 250, 1169  
**Tenovaginitis stenosans**, 1117  
**Tension pneumothorax**, 463  
**Tension**, relief of, 8  
**Teratoblastoma**, 113  
 of breast, 526  
 of kidney, 747  
**Teratoma**, 111  
 of mediastinum, 495  
 of ovary, 111, 818  
 of testis, 112, 114, 801  
**Test meal**, gastric, 586  
**Testis**,  
 anatomy, 791  
 descent, 570, 792  
 diagnosis of swellings, 803  
 epididymo-orchitis, 797  
 gonococcal, 797  
 pyogenic, 797  
 errors in development, 793  
 growths, 800  
 carcinoma, 801  
 seminoma, 801  
 spermatocytoma, 801  
 teratoma, 112, 114, 801  
 hæmatocoele, 807  
 hydrocele, 803  
 acquired, 804  
 congenital, 804  
 encysted, of cord, 804  
 idiopathic, 805  
 radical operation for, 806  
 tapping, 806  
 imperfect descent, 793  
 infection, 796  
 injury, 795  
 orchitis, 797  
 spermatocele, 804  
 syphilis, 800  
 diffuse interstitial or-  
 chitis, 800  
 gumma, 800  
 torsion, 795  
 tuberculosis, 798  
 varicocele, 807  
**Tetanus**, 32  
 antitetanic serum, 34  
 emprosthotonus, 33  
 opisthotonus, 33  
 pleurothotonus, 33  
 risus sardonius in, 33  
 treatment of, 34  
 trismus in, 33  
 varieties of, 33  
**Tetany**, 374  
 gastric, 593  
**Theatre**, operating, 182  
**Thenar space**, abscess of, 253  
**Thermal injuries**, 129  
**Thermo-electro-chemical ulcers**,  
 167  
**Thiersch's skin graft**, 169  
**Third generation syphilis**, 74  
**Third ventricle**, operations on,  
 867  
**Thomas'**  
 arm splint, 925  
 caliper knee splint, 1083  
 knee splint, 925, 981, 983,  
 1083  
 spinal frame, 1094  
 wrench, 1138  
**Thoracic changes in scoliosis**,  
 1119  
**Thoracic duct**, injury to, 289  
**Thoracoplasty**, 477, 502  
**Thorax**,  
 foreign bodies in, 465  
 injury to, 462  
 penetrating wounds of, 465  
**Threatened gangrene**, 171  
**Three-way syringe**, Rotanda,  
 150  
**Thrombo-angiitis obliterans**,  
 265, 913  
**Thrombocytopænia**, essential,  
 713  
**Thrombosis**, 276  
 arterial, 278  
 lateral sinus, 392, 852  
 phleboliths, 277  
 red thrombus, 277  
 venous, 277  
 white thrombus, 277  
**Thrombosis of mesenteric ves-**  
**sels**. See Mesenteric Vas-  
 cular Occlusion  
**Thrush**, 329  
**Thymus**,  
 in Graves' disease, 370  
 malignant disease of, 496  
 simple enlargement, 496  
**Thyroglossal cyst**, 114, 353  
**Thyroglossal duct**, 353  
**Thyroid gland**, 363  
 anomalies, 364  
 colloid goitre, 368  
 development, 363  
 etiology of thyroid disease,  
 366  
 growths, 375  
 in mediastinum, 495  
 hyperplasia of puberty, 366  
 infections, 364  
 iodine deficiency in, 370  
 medication in, 373  
 nodular goitre, 369  
 parenchymatous, goitre, 367  
 simple goitre, 366  
 thyrotoxic goitre, 369  
**Thyroiditis**,  
 acute, 364  
 chronic, 365  
**Thyrotoxic goitre**, 369  
**Thyrotoxicosis**, 369  
**Tibia**,  
 fractures of, 994  
 shaft, 996  
 tubercle, 995  
 upper end, 994  
 with fibula, 997  
**Tibial spine**, fractures of, 995  
**Tic Douloureux**, 868, 907  
**Tidal drainage**, 759  
**Tinel's sign**, 892  
**Tongue**,  
 carcinoma of, 342  
 congenital anomalies of, 336  
 dermoid cyst of, 340  
 infections of, 337  
 lingual thyroid of, 340  
 ulcers of, 339  
 use of radium in, 345  
**Tongue-tie**, 336  
**Tonsillitis**,  
 acute, 428  
 chronic, 430  
**Tonsils**,  
 keratosis of, 432  
 removal of, 431  
**Tophi**, gouty, 234  
**Torsion**  
 of omentum, 565  
 of ovarian cysts, 819  
 of spleen, 712  
 of testis, 795  
**Torticollis**,  
 congenital, 356  
 spasmodic, 357  
**Tourniquet**, ill-effects of, 894  
**Toxæmia**, 10  
**Toxic gangrene**, 178  
**Toxic goitre**, 369  
**Toxins**, 16  
 endotoxin, 17  
 exotoxin, 16  
**Toxoid**, 18  
**Tracheal tug**, 278  
**Tracheotomy**, 459  
 tube, Durham's, 460  
**Traction**,  
 skeletal, 927  
 surface, 925  
**Traction splints**, 924  
**Transcælotomic implantation**, 88,  
 595, 819  
**Transillumination**, 806  
**Transitional-celled carcinoma**,  
 106  
**Transitional ulcer**, 167  
**Transurethral prostatectomy**,  
 775  
**Transverse myelitis**, 884  
**Transverse process**, fracture of,  
 1011  
**Traumatic cephalhydrocele**, 831  
**Traumatic gangrene**, 178  
**Traumatic neurasthenia**,  
 cranial, 829, 848  
 spinal, 883  
**Traumatic neuroma**, 991  
**Treatment**, post-operative, 186  
**Trendelenburg's sign**, 1061  
**Treponema pallidum**, 64  
**Treves**, bloodless fold of, 666  
**Trichina spiralis**, 51, 117, 1185  
**Trichinosis**, 51  
**Trichlorophenylmethyliodo-**  
**salicyl (T.C.P.)**, 181  
**Trichomonas**, 62  
**"Trident hand"**, 1067

- Trigeminal nerve, 906**  
 alcohol injection of, 868  
 removal of gasserian ganglion, 868  
 tic douloureux in, 868, 907  
**"Trigger finger," 1117**  
**Triple displacement of knee, 1082**  
**Triple dye, 137**  
**Trismus, 318**  
**Trochanters of femur, fractures of, 982**  
**Trophic ulcer, 243**  
**Truss, hernial, 567**  
**Tryparsamide, 78**  
**Tubercle of tibia,**  
 in Schlatter's disease, 1044  
 transplantation of, 1136  
**Tuberculosis, 38**  
 generalised, 1074  
 lymph glands in, 294  
 of bladder, 764  
 of bone, 1025  
 of breast, 513  
 of bursæ, 1173  
 of ear, 390  
 of individual joints. See under joint concerned  
 of intestinal tract, 619  
 of joints, 1071  
 of kidney, 733  
 of larynx, 450  
 of liver, 689  
 of mesenteric lymph glands, 542  
 of muscle, 1165  
 of peritoneum, 556  
 of pharynx, 433  
 of prostate, 798  
 of spine, 1087  
 of tendon sheaths, 1169  
 of testis, 789  
 of tongue, 339  
 of ureter, 734  
 of vesicles, 777, 798  
 pulmonary. See Pulmonary  
 Tuberculosis, 497  
 skin lesions, 234  
**Tuberculosis of bone, 1025**  
 caries necrotica in, 1026  
 dactylitis, 1027  
 of skull, 837  
 of spine, 1087  
 osteitis, 1026  
 periostitis, 1025  
**Tuberculosis of joints, 1071**  
 ankylosis in, 1072  
 bone lesions in, 1072  
 caries sicca, 1072  
 complications of, 1074  
 abscess formation, 1074  
 generalised tuberculosis, 1074  
 sinus formation, 1074  
 diagnosis of, 1073  
 "night cries" in, 1073  
 physical signs in, 1073  
 prognosis, 1073  
 synovial lesions in, 1072  
**Tuberculosis of joints—continued**  
 treatment, 1074  
 fixation, 1075  
 general, 1074  
 local, 1075  
 operative, 1075  
**Tuberculous lymphadenitis, 294**  
 in mesentery, 542  
 in neck, 296  
**Tuberculous lymphangitis, 290**  
**Tuberculous peritonitis, 556**  
 acute miliary, 556  
 adhesive, 558  
 ascitic, 557  
 chronic, 557  
 encysted, 557  
 purulent, 558  
**Tubulo-dermoid, 114**  
**Tumours, 83**  
 classification of, 88  
 dissemination of, 86  
 embolism in, 88  
 etiology, 83  
 infiltration in, 85  
 innocence and malignancy, 85  
 permeation in, 87  
 structure and growth of, 84  
 theories of formation, 84  
 transcœlomic implantation, 88  
 transplantation, 88  
**Tumours of**  
 abdominal wall, 537  
 anal canal, 665  
 appendix, 684  
 bladder, 767  
 blood vessels, 286  
 bone, 1046  
 brain, 854  
 branchial cleft, 362  
 breast, female, 515  
   male, 527  
 bronchus, 490  
 cartilage, 92, 1046  
 colon, 626  
 common bile duct, 705  
 duodenum, 612  
 face, 303  
 floor of mouth, 346  
 gall-bladder, 705  
 gums, 321  
 ileum and jejunum, 626  
 jaws, 314  
 kidney, 745  
 larynx, 455  
 lip, 307  
 liver, 691  
 lung, 490  
 lymph glands, 299  
   vessels, 290  
 meninges, 861  
 mesentery, 543  
 muscle, 1167  
 œsophagus, 442  
 ovary, 819  
 pancreas, 710  
 penis, 783  
**Tumours of—continued**  
 peripheral nerves, 895  
 pharynx, 436  
 prostate, 776  
 rectum, 660  
 renal pelvis, 749  
 retroperitoneum, 543  
 sacrococcygeal region, 875  
 salivary glands, 350  
 scalp, 828  
 scrotum, 808  
 sebaceous and sweat glands, 242  
 skin, 237  
 skull, 837  
 spinal cord, 885  
 spleen, 712  
 stomach, 597  
 tendon sheaths, 1171  
 testis, 800  
 thyroid, 375  
 tongue, 342  
 umbilicus, 540  
 urethra, 790  
 uterus, 820  
 vulva, 811  
**Tunica vaginalis, 803**  
**Turbinates, inferior, 399**  
 hypertrophy, 402  
 middle, 399  
 overgrowth, 402  
**Turn-buckle jacket, 1122**  
**Tympanic membrane, 378**  
 incision of, 389  
 injury to, 386  
 involved in fractures of skull, 834  
 perforation of, 388  
**Typhoid, 46, 618**  
 arthritis, 1069  
 osteitis, 886, 1025
- U**
- Ulcer,**  
 acute, 165  
 chronic, 165  
 eczematous, 166  
 gastrojejunal, 610  
 healing of, 5  
 herpetic, 306  
 irritable, 166  
 Meleney's, 167  
 neurotrophic, 166  
 of duodenum, 606  
 of jejunum, 610  
 of lip, 306  
 of palate, 335  
 of rectum, 650  
 of stomach, 600  
 of tongue, 339  
 perforating, 71, 243  
 pressure, 166  
 snail track, 69  
 thermo-electro-chemical, 167  
 treatment, 167  
 tuberculous, of skin, 235  
 varicose, 166

- Ulceration, 163**  
 classification, 163  
 clinical picture, 163  
 complications, 170  
 non-specific, 164  
 treatment, 167
- Ulcerative colitis, 617**
- Ulcer-cancer of stomach, 600**
- Ulna, fractures of, 958**  
 coronoid process, 959  
 olecranon, 958  
 shaft, 959
- Ulnar bursa, 245, 251**
- Ulnar nerve injury, 901**  
 at elbow, 903, 957  
 at wrist, 901  
 recurrent dislocation of, 903
- Ultra-violet light, 219**
- Umbilical hernia, 578**
- Umbilical polyp, 540**
- Umbilico-urachal sinus, 754**
- Umbilicus,**  
 acanthosis nigricans, 539  
 affections of, 539  
 "caput medusæ," 539  
 discoloration of skin, 539  
 endometrioma of, 540  
 exomphalos, 539  
 mucous adenoma, 540  
 retroperitoneal abscess point-  
 ing at, 539  
 secondary carcinoma, 540
- Uncinate process, 399, 402**
- Universal donor, 148**
- Unna's paste stocking, 168**
- Upper motor neurone paralysis, 882**
- Urachal cyst, 754**
- Urachus, patent, 754**
- Urate calculus, 736**
- Urea clearance test, 718**
- Urea concentration**  
 in blood, 718  
 in urine, 718
- Ureter,**  
 anatomy of, 715  
 anomalies of, 719  
 calculus in, 750  
 cyst of, 750  
 injury to, 749
- Ureteric calculus, 750**  
 anuria with, 752  
 operative treatment, 752  
 palliative treatment, 751  
 symptoms, 750
- Ureteric catheterisation, 717**
- Ureterocele, 750**
- Urethra, female, 812**  
 caruncle, 812  
 prolapse, 812
- Urethra, male,**  
 anatomy of, 778  
 calculus in, 790  
 fistula, 789  
 foreign bodies in, 790  
 growths of, 790  
 injury to, 784  
 complicating fracture of  
 pelvis, 973
- Urethra—continued**  
 occlusion of, 780  
 periurethral abscess, 788  
 rupture of, 785  
 stricture in, 786  
 urethritis, 54, 784
- Urethral chill, 729**
- Urethral stricture, 786**  
 acute retention with, 787  
 congenital, 786  
 external urethrotomy, 788  
 inflammatory, 786  
 internal urethrotomy, 787  
 results of, 786  
 spasmodic, 786  
 traumatic, 786  
 treatment, 787  
 by dilatation, 787  
 by operation, 787  
 varieties of, 786
- Urethritis,**  
 gonococcal, 54, 784  
 non-gonococcal, 61
- Uric acid calculus, 738**
- Urinary tract, examination of, 226, 715**
- Urine,**  
 examination of, 716  
 extravasation of, 760, 789  
 pH of, 729
- Urography, intravenous, 227, 717**  
 in hydronephrosis, 724  
 in renal calculus, 740  
 tuberculosis, 735  
 tumours, 748
- Uroselectan, 227**
- Uterus,**  
 adenomyoma, 821  
 carcinoma of body, 821  
 of cervix, 822  
 chorionic carcinoma, 822  
 fibroids, 820  
 hydatidiform mole, 823  
 polypi, 821  
 sarcoma, 822
- Uvula, elongation of, 335**
- V**
- Vaccines, 19**
- Vagus nerve, 909**
- Valsalva's experiment, 382**
- Valves of Houston, 646**
- Variocoele, 807**
- Variocose aneurysm, 271**
- Variocose eczema, 286**
- Variocose ulcer, 166**
- Variocose veins, 283**  
 complications of, 286  
 injection of, 286
- Varix, saphenous, 284**
- Vas deferens, 791**
- Vascular gangrene, 174**
- Vasomotor gangrene, 175**  
 rhinitis, 407
- Vegetative cystitis, 764**
- Veins,**  
 air embolus in, 281
- Veins—continued**  
 dilated in abdominal wall,  
 538  
 diseases of, 281  
 hæmorrhage from, 281  
 injection of, 285  
 injury to, 280  
 phlebitis, 281  
 thrombophlebitis, 281  
 varicose, 283
- Veneral diseases, 54**
- Venous hæmorrhage, 154**
- Venous sinus thrombosis, 852**
- Ventral hernia. See Incisional  
 Hernia**
- Ventriculography, 228, 853, 854**
- Verruca necrogenica, 235**
- Vertebra, fracture of,**  
 body, 1007  
 by compression, 1007  
 by flexion, 1009  
 with dislocation, 1009  
 without dislocation, 1010  
 neural arches, 1011  
 transverse processes, 1011
- Vertebral changes in scoliosis,  
 1119**
- Vesical calculus, 765**
- Vesical fistula, suprapubic, 764**
- Vesico-intestinal fistula, 624,  
 765**
- Vesico-umbilical fistula, 754**
- Vesico-urachal sinus, 754**
- Vesico-vaginal fistula, 765**
- Vesiculitis,**  
 acute, 59  
 chronic, 59  
 tuberculous, 798
- Villous arthritis, 1059**
- Vincent's angina, 329, 427**
- Virchow's chondro-arthritis,  
 1099**
- Virus infections, 44**
- Visceroptosis, 616**
- Vitamin deficiency, 1031, 1036**
- Vocal cords, 445**  
 abductor palsy, bilateral, 454  
 unilateral, 455  
 adductor palsy, 453
- Volkmann's ischæmic paralysis,  
 1166**
- Volvulus, 639**  
 of cæcum, 640  
 of sigmoid colon, 639  
 of small intestine, 640
- Vomit, faecal, 633**
- Vomiting after anaesthesia, 208**
- Von Bechterew's type of spon-  
 dylitis deformans, 887, 1124**
- Von Graefe's sign, 375**
- Von Mielkules' disease, 349**
- Von Pirquet's reaction, 40**
- Von Recklinghausen's disease,  
 of bone, 1040  
 of nerves, 89, 895**
- Vulva,**  
 cysts, 811  
 growths, 811  
 infections, 809

**Vulva**—*continued*  
 kraurosis, 810  
 leukoplakia, 810  
**Vulvitis**, 809

## W

**Wagstaffe's fracture**, 1002  
 "Waiter's arm," 896  
**War surgical unit**, 159  
**Wardill's operation**, 334  
**Wardrop's ligature for aneurysm**, 270  
**Warts**, 103, 238  
   venereal, 60, 63, 238  
**Wassermann reaction**, 19, 66, 75  
**Wasting of muscles**, 890  
   in tuberculous joints, 1073  
**Water balance**, preservation of, 9  
**Watson-Cheyne's operation**, 244  
**Wax in ear**, 384  
**Weaver's bottom**, 1175  
**Webbed penis**, 780  
**Weber's test**, 382  
**Wens**. See Sebaceous Cysts  
**Wheelhouse's operation**, 788  
 "White leg," 278  
**White thrombus**, 277  
**Whitman's frame**, 1122  
   operation, 1151  
   plaster, 981  
   valgus brace, 1142  
**Widal reaction**, 46, 618  
**Wilkie's technique**, 644  
**Wilm's tumour**, 113, 747  
**Wilson and Willis' theory**, 747  
**Wisdom teeth**, 325  
**Wolfe's skin graft**, 170  
 "Woody tongue," 49  
 "Woolsorters' disease," 42  
**Worms**, infections due to, 51

## Wounds, 118

  closed, 118  
   cross-infection of, 128  
   delayed healing of, 128  
   excision of, 124  
   gun-shot, 118  
   healing of, 4  
   incised, 120  
   lacerated, 122  
   open, 120  
   penetrating, 120  
   punctured, 120  
   secondary suture of, 128  
   treatment of, 124  
   varieties of, 118

## Wounds of

  abdominal wall, 529  
   arteries and veins, 261  
   biliary system, 696  
   bladder, 760  
   brain, 841  
   bursæ, 1172  
   duodenum, 588  
   intestinal tract, 532  
   joints, 933, 936  
   kidney, 721  
   liver, 534, 686  
   lung, 465  
   muscles, 1164  
   pancreas, 706  
   penis, 782  
   peripheral nerves, 890  
   rectum, 648  
   scalp, 825  
   skull, 828  
   stomach, 534, 587  
   tendons, 1164  
   ureter, 749

## Wrist joint,

  dislocation of, 968  
   effusion into, 982  
   position for ankylosis, 1063  
   tuberculosis, 1087

**Wry neck**. See Torticollis.

## X

**Xanthin calculi**, 739

**Xanthoma of tendon sheaths**, 1172

## X-rays, 222

  barium enema technique, 226  
   meal technique, 224  
   burns from, 141  
   dermatitis from, 141  
   in diagnosis, 222  
   in fractures, 222  
   in gall-bladder disease, 226  
   in gastro-intestinal disease, 224  
   intensifying screens, 222  
   in treatment, 229  
   of central nervous system, 228  
   of urinary tract, 226  
   Potter - Bucky diaphragm, 222

## X-rays in treatment, 229

  erythema dose, 229  
   in carcinoma of anal canal, 665  
   of prostate, 777  
   of vulva, 812  
   in chronic mastitis, 512  
   in growths of bone, 1055  
   of testis, 802  
   in leukoplakia vulvæ, 812

## Y

**Yaws**, 48

**Young's median prostatic bar**, 776

## Z

**Ziehl-Neelsen stain**, 38

**Zuckerkandl**, fascia of, 714







